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ORIGINAL ARTICLES.

A CLINICAL STUDY OF THE THERAPEUTIC VALUE OF THE CALCIUM SALTS IN GASTRIC TETANY, WITH AN ANATOMICAL REPORT ON THE PARATHYROID BODIES.[1]

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Recent researches tend to show that there is an intimate relation between the various forms of tetany and an insufficiency of the parathyroid glands; they also strongly suggest that there is an equally direct and intimate relation between disturbances of the calcium contents of the tissues of the body and tetany; and, finally, that the secretion of the parathyroid bodies exerts in an as yet imperfectly understood way a controlling influence upon calcium metabolism. Numerous researches have shown the moderating effect of the calcium salts upon the hyperexcitability of the central nervous system, and the recent noteworthy study by MacCallum and Voegtlin[2] has demonstrated the controlling influence of these salts upon the spasm of experimental tetany. The results observed by them have suggested the probable therapeutic value of the calcium salts in the various forms of human tetany.

Recently an opportunity has been afforded in the Presbyterian Hospital for a study of the effect of the calcium salts in a case of

[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 11 and 12, 1909.

[2] On the Relation of Tetany to the Parathyroid Glands and to Calcium Metabolism, Jour. Exp. Med., 1909, xi, 118.

gastric tetany, and of the parathyroid bodies after death. The case is of much interest in view of the rarity of that form of gastric tetany associated with dilatation of the stomach and stagnation of its contents, of which it is an example; in the severity of the tetanic symptoms; in the prompt inhibitory effect upon the spasms of the calcium salts artificially introduced directly into the blood stream; and, finally, in the findings at autopsy.

A male, aged thirty-three years, was received in my wards in the Presbyterian Hospital on October 25, with the diagnosis of gastric tetany by my interne Doctor Vaughan, a diagnosis fully confirmed on examination of the patient. The family and personal histories, briefly, are as follows: Both parents died of pulmonary tuberculosis. The patient at times has been a heavy drinker, and especially has been a large consumer of beer. There is a history of gastric symptoms from early in 1905, at which time he was admitted to Bellevue Hospital suffering apparently from an acute gastritis of alcoholic origin; he was discharged relieved of his symptoms. A year later he was again admitted on account of a severe gastric hemorrhage following a drinking bout. A diagnosis of duodenal ulcer was made. On discharge from the hospital, and up to the date of the illness for which he was admitted to the Presbyterian Hospital, he suffered from much epigastric distress, flatulence, and frequent vomiting. He states that occasionally as much as "a gallon of green fluid" was vomited, but at no time any blood. The night before admission to my wards the patient states that he drank copiously of aërated water, nothing alcoholic. He went to bed feeling well, slept, but awoke several hours later with severe cramps in the legs and arms. He was unable to move them, but felt no pain beyond a drawn and tense feeling in the cramped muscles. He called for assistance with difficulty, as his tongue and jaws were stiff. His abdomen was distended, and he vomited several times. He sweated profusely. He was very restless. Later his affected muscles became tender and were painful. The cramps and stiffness in no way relaxed up to the time of admission.

Examination on Admission. Patient is poorly nourished; looks ill. Is unable to speak intelligibly. Is extremely restless. Temperature (rectal), 101°. Pulse, 100 and of poor quality, regular in rhythm. Blood pressure, systolic, 112 mm. Respirations, 80. The attention is at once attracted to the abdomen and to the presence of tetanic symptoms. The contour of a dilated and distended stomach is easily appreciable. The lower outline reaches a point 10 cm. below the umbilicus in the median line, and to the right the distention reaches well beyond the parasternal line. Succussion sounds can be produced readily over the whole of the distended area. Both legs are held straight and stiff, the feet in extreme extension, the toes in plantar flexion. The muscles of the calf of legs are hard, tense, and drawn. The spasm is continuous, but pressure on the nerve

trunks and bloodvessels increases, if possible, the severity of the spasm (Trousseau's sign), and the same phenomenon obtains on tapping over the motor nerves supplying the affected muscles (Chvostek's sign). The forearms are flexed at a right angle on the arms, the wrists flexed to an extreme degree and cannot be extended, the fingers flexed at right angles at the metacarpophalangeal articulations and held rigidly straight in adduction, the thumb tightly against index finger—not drawn into palm. The spasms are continuous, but, as in the case of the lower extremities, they can be increased both by Trousseau's and Chvostek's methods. The tendon reflexes appear to be about normal in both upper and lower extremities. There are continuous twitchings of the muscles of the face, which are increased by tapping over the course of the motor trunks. A slight degree of trismus is present. The musculature of the trunk is free from spasm, and it cannot be excited by mechanical irritation of the motor nerves or the muscles. The tachypnœa is striking, the respirations, as previously stated, being 80.

The stomach was at once washed out and a large quantity of greenish ill-smelling fluid was recovered. A few hours subsequent to the lavage the spasms of the upper extremities were entirely relieved, but there was no intermission in those of the lower.

October 26. The spasms of the upper extremities are again present and are continuous, and there has been no intermission in those of the lower extremities. In spite of lavage twice used in the past twenty-four hours the patient has vomited several times, 80 ounces in all; the vomited matter a greenish fluid. The patient has not voided urine since admission, and catheterization recovered only 6 ounces of urine, of specific gravity 1016, containing a trace of albumin, no glucose, a trace of indican, and a few hyaline and granular casts. Subcutaneous clyses, 1000 c.c. of a normal salt solution, were ordered to relieve, if possible, the evident dehydration of the body tissues. The temperature fell gradually during the day to 97.5° at midnight; the respirations to 20 to 24. Blood examination: Red cells, 4,910,000; white cells, 13,500; hemoglobin, 75 per cent.; color index, 0.7; polymorphonuclears, 89 per cent.; large and small lymphocytes, 9.3 per cent; transitionals, 1.7 per cent.

October 27. There has been no appreciable diminution in the spasms of the extremities, which are still continuous. In addition to lavage and subcutaneous clyses, one-half grain of a parathyroid extract, furnished by Dr. Beebe, was directed to be given three times daily by the mouth, and caffeine as a heart stimulant. The temperature has been subnormal throughout the day. In view of the pulmonary signs present, von Pirquet's tuberculin test was tried, with negative results.

October 28 (A.M.). The patient was extremely restless and irrational in the early part of the night, but after lavage and a subcutaneous clysis of 2000 c.c. of salt solution, slept quietly, with a partial

relief of the spasms of the upper extremities, but with continued extensor spasm of the muscles of the legs. During the day the spasms in the upper extremities have again been present, and there has been no intermission in those of the lower extremities. Patient very restless and irrational. In addition to the parathyroid extract, 2 grams of calcium lactate in 1200 c.c. of normal salt solution was directed to be given intravenously. The immediate effect was to quiet the great restlessness of the patient, and a few hours later there was a marked diminution in the degree of spasm in the upper extremities, and for the first time since admission, in the lower extremities. The temperature rose slightly to 100° before the use of the calcium salt, and did not rise above this after its administration. Pulse, 80 to 96; respirations, 20 to 24.

October 29 (A.M.). The patient slept quietly the greater portion of the night. There is no spasm of the upper extremities and only slight spasm (extensor) of the legs. The electrical reactions were tested for the first time and were not found to be increased. (P.M.) Four grams of calcium lactate in 1300 c.c. of normal salt solution was given intravenously. The patient fell asleep during its administration, and for the first time since admission he became entirely relaxed. Temperature, 99.5° to 100.5°; respirations, 20.

October 30. The patient slept quietly during the night. The upper extremities are free from spasm. Late in the day slight spasm of the extensor muscles of the legs was noted. Tapping over the branches of the facial nerve still elicits slight spasm in the muscles of its distribution. This can be produced only after several moments of mechanical irritation, in marked contrast to the almost instantaneous response obtained before the first administration of the calcium salt. Four grams of calcium lactate in 1000 c.c. of normal salt solution was again given intravenously in the late afternoon. Temperature, normal. Pulse, 80 to 84; respirations, 20.

October 31. The patient slept quietly with an entire absence of spasm. Both upper and lower extremities are free from spasm, but there is slight rhythmic movement of the forearms, about 60 times a minute. The patient's mind is quite clear for the first time, and he takes an interest in all that is done for him and asks intelligent questions in regard to its purpose. The pulse has ranged from 82 to 96. The temperature (rectal) has been normal. Respirations, 20 to 24. White cells, 9900. Calcium lactate in 1000 c.c. normal salt solution was given.

November 1. The rhythmic movements of the forearms have disappeared. Spasm is entirely absent in the lower extremities. Upper extremities: From time to time the hand is momentarily flexed on forearm at intervals of about five seconds. Mechanical irritation of the nerve trunks does not increase this phenomenon. The calcium salt was omitted and a nucleoproteid preparation of the parathyroid, furnished by Dr. Beebe, was injected deep into the

gluteal region twice in the twenty-four hours. Pulse, 80 to 95 during day. Temperature, slightly subnormal. Respirations, 20 to 24.

November 2. The patient slept quietly during the night. The spasms of the muscles of the forearms have increased and there is very considerable tremor of the hands. There is also some tremor of the muscles of the chin. Pulse, 84 to 96. Temperature, subnormal during the day. Respirations, 20 to 24. In view of the increase in the spasms, in addition to the parathyroid preparation, 4 grams of calcium lactate in 1000 c.c. of normal salt solution was again given intravenously.

November 3. The patient is entirely relaxed and no spasm can be elicited by mechanical irritation of the nerve trunks in either upper or lower extremities or in the muscles of the face. Considerable tremor of the hands is present, however. The patient's general condition is not as good. Hiccough is present. The pulse is of poor force, 80 to 100 during the day. Temperature, subnormal. Respirations, 20. Four grams of calcium lactate in salt solution was given intravenously. From this date the calcium salts were discontinued and no other medication was used. There was no further spontaneous spasm, and it could not be excited by mechanical means. The patient's mind remained entirely clear, he was interested in measures taken for his relief, and was able partially to sit up in bed. There was little or no change in the temperature, pulse, and respirations from those last noted. Three days later, without the occurrence of other symptoms, the heart rather suddenly failed, and death followed speedily.

That portion of the clinical history which especially relates to the tetanic symptoms and to the effect secured by the administration of the calcium salts has been selected for detailed report. Other portions of the history will be given only in outline.

It was found impossible to nourish the patient by means of the stomach. Vomiting was frequent on each attempt, in spite of copious and frequent lavage. Nutriment introduced after lavage by means of the stomach tube, although sometimes retained, was recovered later by the tube in like amounts, showing no signs of gastric digestion—only decomposition processes. Nutrient enemas were only exceptionally retained. There was almost complete anuria for the first twenty-four hours and until abundant subcutaneous clyses of salt solution had been given. Later, the urine either was voided involuntarily or was recovered by catheterization. The scanty fluid stools were mostly involuntary.

Although the tetanic symptoms obviously could be controlled by the continued administration of the calcium salts, it was equally apparent that the patient would slowly die of inanition unless the evident gastric stenosis could be relieved by operation. In consultation, however, my late colleague, Dr. McCosh, decided against surgical intervention, on the ground that any operation would be immediately fatal.

Autopsy, sixteen hours postmortem, by Dr. Opie. The body is that of an emaciated man 160 cm. in length. Over the inner surface of the left elbow and on the right arm there are ecchymoses. The subcutaneous fat is present in very small amount, and has a deep yellow color. The peritoneal cavity contains no accumulation of fluid; fat has almost wholly disappeared from omentum. The greater curvature of the stomach is 16 cm. below the ensiform cartilage in median line. The stomach extends 9 cm. to the right of the midline, and is in contact with the ribs on the left side. The lesser curvature of the stomach in the midline is 6 cm. below the ensiform cartilage. The spleen is firmly adherent to the lateral abdominal wall. The stomach has fallen considerably, and above the lesser curvature the entire head and greater part of the pancreas is visible. The lungs are emphysematous and meet in the median line; a few adhesions bind each apex to the chest wall. The pericardial cavity contains about 10 c.c. of clear fluid. The appendix arises from the inner surface of the cecum, passes inward, and lies with the tip over the pelvic brim. It is possible to pass one finger with difficulty through the foramen of Winslow.

The stomach measures 25 cm. transversely and 8.5 cm. vertically at the cardiac end. Pyloric half is almost tubular and is 5 cm. in diameter. On opening the stomach it was found to contain several hundred c.c. of bright yellow fluid, from which a sediment settles. Adherent to the mucosa is mucus. The mucous membrane is thickened, the rugæ are prominent, and the surface has a homogeneous, deep red color. The wall of the stomach is thickened by hypertrophy of its muscular layer to about 4 mm. The duodenum immediately beyond the pylorus is bent backward and held in contact with the stomach by adhesions for a distance of about 1 cm. Gentle separation of the adhesions exposes a perforation, from which gastric contents escape. It is found impossible to pass a finger from the stomach through the pylorus into the duodenum, obstruction being in a large part due to the bend just described. Immediately below the pylorus in the mucous membrane of the duodenum there is a depressed scar, 0.25 x 1 cm., partially encircling the duodenum. At the upper angle of this area is a deeper ulceration, in diameter not more than 7 mm.; the surface here is black and eroded, and the partial perforation mentioned above allows the passage of a small probe. There is thick fibrous tissue immediately about the ulcer, but no marked induration. The duodenum is somewhat injected, but otherwise normal. The pancreas is normal. The heart weighs 255 grams. The organ is small; the muscle is firm and brownish in color. The aortic valve is normal; there is slight thickening of the free edge of the mitral valve; there are several minute nodular projections. The pulmonary and tricuspid valves are normal. The coronary arteries are smooth.

The left lung is voluminous, and abundant fluid escapes from the

cut surface. Throughout a large part of the upper lobe is scattered gray nodules partly caseous; there are several larger nodules 1.5 cm. across, and cavity formation has occurred in the centre of a caseous area. Right lung is much more voluminous than the left; tubercles are much more numerous in the upper lobe, and there occur several small cavities similar to the cavity in the left lung; it is in direct communication with a bronchus. The uppermost 2 cm. of the apex is almost entirely consolidated by tuberculous nodules. The lower lobe contains sparsely scattered tubercles; in large part it is firm and is partially consolidated by scattered areas of homogeneous, deep red color, though nowhere is it entirely airless. The liver weighs 1450 grams. The organ is firm and deep red; the mottling is indistinct. Gall-bladder is contracted and contains a small amount of viscid bile. The spleen is not enlarged; it measures 11 x 7.5 x 4.5 cm. It is very firm, and has a homogeneous, deep red color; the Malpighian bodies not well seen. The kidneys weigh 310 grams together. They resemble each other closely; the capsule is removed easily, leaving a red surface in which there are a few minute cysts. The cortex, which measures about 5 mm., is deep red. The glomeruli are conspicuous; the pyramids are injected. On the surface of the right kidney there is a yellow patch 0.5 cm. across (apparently aberrant adrenal tissue). Suprarenals together weigh 20 grams. They appear to be normal. The aorta is smooth save for a few slightly raised yellow patches. The urinary bladder is normal in size. Scattered over the inner surface, but chiefly on the posterior wall above the trigon, are submucous hemorrhages; in this situation are numerous minute vesicles. The rectum is deeply injected. The thyroid gland appears to be normal. The two parathyroids on either side were exposed, and as far as could be determined are normal. The small intestine in some places shows injection of the mucous membrane, most marked in the lower part of the ileum where the mucous membrane has a homogeneous, deep red color. The lumen of the appendix is patent. The large intestine throughout shows intense injection of the mucous membrane, but is otherwise negative.

Culture from the heart's blood shows a pure growth of Bacillus coli.

Microscopic Examinations. The liver tissue is much congested. The vessels of the kidney are all dilated and filled with blood. The epithelium of the tubules shows cloudy swelling; the cells are in places desquamated and the tubules are filled with debris. In the cortex there are many areas infiltrated with lymphoid cells. The bloodvessels of the lungs are everywhere congested, and in places there is serous exudate in the alveoli. There are many areas in which the alveoli are completely filled by polymorphonuclear leukocytes; there is a little fibrin. In several areas are chronic tuberculous lesions associated with newly formed fibrous tissue. The

heart muscle is normal. The bloodvessels of the spleen are much congested.

Parathyroid Glands. The right and left superior parathyroid bodies were examined microscopically. They are of normal size and exhibit no abnormality. Scattered cells with affinity for eosin occur in moderate number. There is no increase of connective tissue and the bloodvessels are normal. The right inferior body likewise appears to be normal in structure and size. In the connective tissue near this parathyroid is a small round mass of tissue which resembles the much larger parathyroid body from which it is wholly isolated. The cells which compose this nodule are smaller than those of the parathyroid, and their protoplasm stains more deeply; they are, like those of the parathyroid, arranged in columns between the capillaries. About this nodule connective tissue is concentrically arranged to form an ill-defined capsule; in this tissue lymphoid cells are numerous. At a short distance from the nodule just described, and from the normal parathyroid and situated just outside the wall of a small vein, is an isolated round group of cells identical with those which make up the aberrant nodule. This group consists of about 25 small cells closely packed together. Sections made through the middle of the left inferior glandule showed that parathyroid tissue is abundant and normal. The parathyroid glands therefore, exhibit no abnormality; no evidence of hypertrophy is found. (Near one parathyroid are small aberrant nodules of tissue resembling that of the parathyroid.) The thyroid gland is normal in structure. The alveoli contain colloid in normal amount.

Studies of the Urine. Guinea-pig I. Injected with 20 c.c. of urine into peritoneal cavity. Died after twenty-four hours; some fluid in peritoneum. No exudate. Cultures sterile. Sections, see below.

Guinea-pig II. Injected with 15 c.c. of urine into peritoneal cavity. Died in thirty hours. Findings as in No. I.

Guinea-pig III. Injected with 10 c.c. of urine. Died in thirty-six hours. Findings as above. Three controls using normal urine, all recovered.

Studies of the Stomach Contents. Guinea-pig IV. Given 10 c.c. of filtered stomach contents by stomach tube; after twelve hours looked sick but recovered.

Guinea-pig V. Injected into peritoneal cavity 5 c.c. of filtered stomach contents. Died in ten hours—peritonitis. No convulsions. Cultures showed many organisms.

Guinea-pig I A. Injected 0.1 c.c. into peritoneum. Died in twelve hours. No convulsion. Anaërobic cultures of peritoneum, sterile.

Guinea-pig II A. Injected 0.2 c.c. subcutaneously. After eighteen hours it developed a paralysis of the right fore leg. Became very spastic, especially the hind legs; there was incoördination; no hyper-

esthesia, but stimulation of the abdomen caused clonic contractions of the limbs, which manifested itself in the jumping of the animal in a forward direction, which seemed uncontrollable. On several occasions the animal jumped so high as to strike on the top of the cage. After the spastic stage, a period of great weakness with loss of reflexes ensued, and the animal died in this stage twenty-two hours after injection.

Cultures. Aërobic: heart blood, Bacillus coli(?) spinal canal, Bacillus coli(?) Anaërobic: heart blood, Bacillus coli; spinal canal, sterile.

The abdominal parietes were very œdematous and reddened; no abscess formation; peritoneum and viscera normal. Brain and cord showed no macroscopic lesion.

Guinea-pig III A. 0.5 c.c. injected subcutaneously. This animal showed exactly the same symptoms as No. II A, except that the motor excitement stage was shorter, came on more quickly; died in twenty-one hours.

Cultures: Aërobic, as above; anaërobic, as above. Pathological findings, as above.

The microscopical sections of Guinea-pigs I, II, and III revealed marked cloudy swelling of the viscera; otherwise nothing of note was found.

Filtrate of Stomach Contents (Berkefeld Filter). Injected into guinea-pig as in I A, II A and III A; no apparent effect

Cultures of the filtrate revealed a few colonies of Bacillus coli.

It is a matter of regret that an examination of the brain and cord was not allowed, as the few reports of autopsies in cases of tetany are conflicting both as regards the existence of any constant lesion of the nervous system and its form. The full anatomical report on the parathyroid bodies, by Dr. Opie, is of especial interest.

Certain facts stand out prominently in the clinical study of the case which has been related:

1. The rapid and controlling effect of the soluble calcium salts upon the tetanic symptoms.

2. The maintenance of this effect only by their continued use.

3. The comparatively slight effect of large infusions of salt solutions, used alone.

4. The slight influence, if any, of parathyroid preparations (nucleoproteid) given by the mouth in controlling the hyperexcitability of the nervous system. The effect of the nucleoproteid given subcutaneously cannot be estimated positively in the present instance, as it was given alone only during a period of twenty-four hours and when the tetanic symptoms had been controlled practically by the calcium salt. A distinct renewal of the spasm at the end of this interval led to the renewed use of the calcium salt in combination with the parathyroid material. The demonstration of the controlling influence of the soluble calcium salts upon the characteristic

symptoms of at least one important variety of human spontaneous tetany—the tetany of gastrectasis, with stagnating stomach contents—suggests a probable similar therapeutic value of these salts in other forms of the disease in human beings.

5. The occurrence of typical tetanic spasm in a case of gastrectasis with parathyroid bodies of normal anatomical structure and—presumably—of normal functioning power.

The facts elicited in the clinical and pathological study of the cases add little to our positive knowledge of the pathogenesis of gastric tetany. A profound disturbance of the calcium metabolism would seem probable in the present instance. The explanation of it is difficult. There is much experimental evidence to show that the parathyroid secretion in some way controls the calcium exchange in the body, yet in the present instance the functioning power of the parathyroids presumably was normal. The generation of a poison of unknown nature in such amount as to cause a relative functional inadequacy of the parathyroid bodies can only be a matter of conjecture.

A COMMON MODIFICATION OF THE FIRST SOUND OF THE NORMAL HEART SIMULATING THAT HEARD WITH MITRAL STENOSIS.[1]

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Something like an apology seems due from one who ventures to attribute to trite and familiar phenomena a meaning beyond that which is offered by the consensus of scientific opinion. Yet the adept in auscultation knows that the net value of his examination involves no less the neglect of adventitious sounds than the appreciation of those which are of diagnostic worth. Therefore physical signs which pass unnoticed because devoid of meaning at once attract attention when they are found to imply an important modification of vital activity.

It is the design of the present contribution to direct attention to a modification of the first sound of the heart which allies it qualitatively to the first sound as heard in mitral stenosis, but which predicates no organic lesion. I venture to suggest that the light recently thrown by physiologists on the nature and variations of blood flow within the normal heart may be directed to the illumi-

[1] Read by title at a meeting of the Association of American Physicians, Washington, D. C., May 11 and 12, 1909.

nation of certain clinical signs which are ordinarily disregarded, because not significant of organic disease.

The tone of the normal first heart sound is distinctly decrescendo in character; the accent is at the beginning of the sound. In the simplest typical case of mitral stenosis the tone of the first sound is crescendo-decrescendo; the most striking moiety of the sound being formed by its initial acute accent. By clinical inference the crescendo element of this sound is presumed to precede contraction of the ventricle and to be therefore presystolic in time. It is further designated as a "murmur," and is thereby at once genetically separated from the normal heart sounds. This topic will rest with simple reference to the fact that the pathological presystolic murmur may pass by insensible gradations from a sound of indefinite duration and extreme roughness of quality to one indistinguishable in quality from the normal first sound and only of sufficient duration to give a crescendo accent to the latter.

The clinical thesis here maintained is that, though the heart may have normal valves, its first sound may begin with the crescendo tone and thus assume the essential character of the sound as heard in mitral stenosis. It must be premised that such a modification of the normal first sound is found only in cases in which the general clinical condition signifies either essential cardiac weakness or perturbation due to acceleration of the circulation; this acoustic phase is therefore of distinctly diagnostic worth.

The crescendo tone of functional origin which may initiate the first heart sound varies considerably in quality, duration, and intensity. Most commonly it occurs as an extremely short, smooth tone, with an acute accent apparently sharply terminating with the beginning of ventricular contraction. Precisely this modification of the first sound of the heart is frequently the sole auscultatory sign of organic mitral stenosis when, presumably, the contractile energy of the left auricle is slight. The first sound as a whole often acquires, in the normal heart, a remote suggestion of the snapping first sound in mitral stenosis. It is not uncommon for the clinical observer to witness, in a given case from day to day, the presystolic murmur of organic mitral stenosis to vary insensibly from a most exaggerated and palpable vibration to a tone which has little intensity and scarcely perceptible duration, and which barely gives the impression of an acute accent initiating the first heart sound. On the other hand, postmortem examination has shown me the heart valves to be normal in cases in which, during life, the crescendo character of the first sound had been so pronounced as to lead to the suspicion of organic mitral obstruction. In other cases of what may be called the "functional presystolic murmur," the crescendo tone is rough and vibratory in character and of perceptible duration. No examples of this category have come to autopsy, but numerous cases have been noted in which the

auditory signs alone strongly suggested organic mitral stenosis, but in which the general signs of the disorder were lacking. One other presystolic auditory symbol, closely allied to the latter, is found in certain of the so-called "reduplications" of the first sound of the heart. When such a "reduplication" is the weaker of the two elements and precedes the contraction of the left ventricle without a perceptible intermission, the first sound may appear to begin with a crescendo tone. It may be said, in passing, that any explanation that holds for the production of the presystolic functional "murmur" would probably denote the origin of at least certain common reduplications of the first sound.

The above is a brief expression of clinical facts as they appear to me. The subject seems worthy of attention, because, first, there is often real difficulty in distinguishing clinically the presystolic sound due to organic valvular stenosis from that arising from functional causes, and second, a consideration of recent disclosures as to the mode of filling of the normal heart seems to suggest an explanation for the functional quasipresystolic murmur which makes it of diagnostic value. The following considerations are offered with a view of elucidating the subject.

The elements entering into the production of the first sound of the heart, especially when there exists obstruction at either auriculoventricular orifice, are exceedingly complex. The characteristic crescendo murmur of mitral stenosis is, according to accepted clinical teaching, produced by vibrations caused by the stream of blood ejected by the contracting left auricle. The murmur is therefore presystolic in time as regards the cycle of the ventricle. Yet, according to the admirable resume of the subject by Gibson,[2] several competent observers maintain that the murmur in question is produced by contraction of the ventricle only, ceasing at the moment of valve closure, and is therefore "systolic" or "protosystolic" in period. Without discussing this discrepancy of opinion, I will assume as correct that view which attributes the presystolic murmur of mitral stenosis to vibrations in the mitral valve and blood current produced by the systole of the left auricle. It only remains to recall the well-known fact that, without evidence of change in the degree of stenosis, the presystolic murmur in a given heart completely disappears either as a result of an exaggerated break in compensation or even, at times, when functional efficiency has been restored. In the first case the loss of the murmur is attributable to paralysis of the left auricle; in the second, to a recovery of the normal balance of pressures in the cardiac chambers.

In 1862[3] Austin Flint described a presystolic murmur as occasionally occurring in subjects suffering from uncomplicated insuffi-

[2] Diseases of the Heart and Aorta, 1898, p. 157.

[3] Amer. Jour. Med. Sci., 1862, xliv, 29.

ciency of the aortic valves while the mitral leaflets remained normal. Flint's description was so convincing that the murmur has commonly been designated by his name. As observed by Dock,[4] German writers commonly ignore Flint's contention, while, on the other hand, in a critical review of the subject, Thayer[5] concludes that "one may be justified in saying that in uncomplicated aortic insufficiency a rumbling, echoing presystolic or mid-diastolic murmur limited to the region of the apex of the heart is very common, occurring, when carefully looked for, in fully half of the cases. The characteristics of this murmur are in no way different from those commonly observed in true mitral stenosis, with the exception of the fact that it is usually of moderate intensity."

In explanation of the origin of his murmur, Flint, as quoted by Thayer, writes: "Experiments show that when the ventricles are filled with a liquid the valvular curtains are floated away from the ventricular sides, approximately to each other and tending to closure of the auricular orifice. In fact, as shown by Drs. Baumgarten and Hamernick, of Germany, a forcible injection of liquid into the left ventricle through the auricular opening will cause a complete closure of this opening by the coaptation of the mitral curtains, so that these authors contend that the natural closure of the auricular orifices is effected not by the contraction of the ventricles, but by the forcible current of blood propelled into the ventricles by the auricles. However this may be, that the mitral curtains are floated out and brought into apposition with each other by simply distending the ventricular cavity with liquid is a fact sufficiently established and easily verified. Now, in cases of considerable aortic insufficiency, the left ventricle is rapidly filled with blood flowing back from the aorta, as well as from the auricle before the auricular contraction takes place. The distention of the ventricle is such that the mitral curtains are brought into coaptation, and when the auricular contraction takes place the mitral direct current passing between the curtains throws them into vibration and gives rise to the characteristic blubbering murmur. The physical condition is, in effect, analogous to the contraction of the mitral orifice from an adhesion of the curtains at their sides, the latter condition, as clinical observation abundantly proves, giving rise to a mitral direct murmur of similar character."

It is not difficult to simulate on the excised heart the conditions premised by Flint, as has been demonstrated to me by Dr. A. D. Hirschfelder. When the left auricle is cut away and the left ventricle is distended with water through a tube tied in the aorta, the mitral valves float up and are closely coaptated. If, now, water be poured from a height upon the upper surface of the valve, simu-

[4] Nothnagel's System of Medicine, Translation, Diseases of the Heart, p. 373.

[5] Observations on the Frequency and Diagnosis of the Flint Murmur in Aortic Insufficiency, Trans. Assoc. Amer. Phys., 1901, xvi, 393.

lating the effect of auricular contraction, it will be seen that the mitral curtains are not forced apart throughout their whole line of contact, but are only separated for a fraction of it in a way to imitate the mechanical conditions of mitral stenosis. Most authors now admit the occasional occurrence of Flint's murmur, though most diverse opinions prevail as to its cause. An extreme example of dissent from Flint's explanation of the origin of the murmur is furnished by Gibson[6] who writes: "The opinion expressed by Flint is of course absolutely untenable. No one with any knowledge of the physiology of the intracardiac movements could for one moment uphold it." Nevertheless, it is just this opinion on which I venture to found the tenets of this paper.

The recent admirable investigations of Hugh A. Stewart[7] have thrown doubt upon all preconceptions as to the modification of intraventricular blood pressure as influenced by aortic insufficiency. Stewart experimentally ruptured the aortic valve in anesthetized dogs while recording, at the same time, the course of arterial blood pressure and volume changes in the ventricles. Stewart maintains that the volume of blood which regurgitates as a result of aortic insufficiency "is negligible." "When the insufficiency is extensive, the pressures in the aorta and ventricle become equalized during the period of relaxation, and there is no further transmission in the phase of ventricular diastasis," that is, during the period of fixed diastolic distention. When, however, the aortic lesion is slight, it is possible, says the author, that the aortic pressure may cause the pressure in the ventricle to rise during the whole period of diastole. Such variations of intraventricular pressure resulting from similar lesions may readily account for the divergence in clinical signs.

Our conceptions of the intracardiac circulation have been greatly illuminated by the now familiar researches of Yandell Henderson[8] whose conclusions have been essentially confirmed by Stewart and by Hirschfelder.[9] Henderson enclosed the ventricles of the living dog's heart within a rubber ball, the organ being inserted through an aperture whose rim could be made to fit accurately the auriculo-ventricular groove. The cavity of the ball being in connection with a tambour, the volume changes of the ventricles were registered with exactness.

The study of his tracings led Henderson to the deduction that the ventricular cycle is composed not, as usually taught, of two, but of three distinct phases, namely, *systole*, or period of contraction and emptying; *diastole*, or period of relaxation and filling; and *diastasis*, or period of rest. He insists that the act of relaxation or diastole is

[6] Op. cit., p. 501.

[7] Experimental and Clinical Investigation of the Pulse and Blood Pressure Changes in Aortic Insufficiency, Arch. Int. Med., 1908, i, 102.

[8] The Volume Curve of the Ventricles of the Mammalian Heart, etc., Amer. Jour. Phys., 1906, xv, 325.

[9] Johns Hopkins Hosp. Bull., November, 1908.

almost or quite as rapid as the systole; "the refilling of the ventricles occurs early in diastole; . . . it is as rapid a process as is the systolic emptying." Following the phase of what may be called active relaxation comes the period of diastasis. "During this period there may be no further relaxation of the ventricles if the number of beats executed in the few seconds preceding has been few, and the tonus is therefore slight. If, however, the beats have been occurring in rapid succession, there may be a gradual relaxation throughout this period as the tonus thus induced gradually wears off. This period (diastasis) is of extremely variable duration." When the rate of heart beat quickens, the increase is due to shortening of the phase of diastasis; when the beat is slowed, it is by prolonging diastasis. "Auricular systole plays a part which is mechanically of very minor importance in the normally beating heart. . . . The mammalian auricles are to be regarded as elastic reservoirs rather than as force pumps. When the dog's heart is beating at a slow natural rhythm the ventricles are refilled so rapidly after each discharge that a period of quiescence may intervene after the refilling is completed, and before the onset of auricular systole. Under such conditions the auricles inject only a small fraction of a cubic centimetre of blood into the already distended ventricles. The currents in the blood thus induced may cause the closure of the auriculoventricular valves. Except for this possible effect, auricular systole is a factor of almost negligible dynamic value." It would seem hard to overestimate the importance of this assumed effect of auricular contraction on the mechanical perfection of valve closure at the outset of ventricular systole. According to Henderson, at slow normal rhythms the ventricles are not completely emptied at systole, but still contain from 20 to 30 per cent. of their diastolic volume. As the heart beat quickens, the tonus of the muscle increases, the ventricle relaxes less in diastole, but an identical volume of blood is ejected at each systole because of more complete contraction of the organ. The optimum rate of beat, through which the maximum amount of blood is discharged within a given time, is a rate at which the ventricle contracts as soon as it is filled, and in which the period of diastasis is completely eliminated. A further increase of rate must diminish the total output, because each systole supervenes upon an incomplete diastole. Admirable researches by Thayer[10] and by Hirschfelder[11] have demonstrated the extreme probability of a frequent diastolic closure of the auriculoventricular valves, brought about by the reflux of the inflowing stream. The view is advanced that this closure is effected with sufficient energy to produce an occasional "third heart sound."

[10] On the Early Diastolic Heart Sound (the so-called Third Heart Sound), Boston Med. and Surg. Jour., 1908, clviii, 713.

[11] Loc. cit.

Students of the venous pulse are familiar with a wave, common in tracings from somewhat weak hearts, just preceding the wave made by auricular contraction. Such a wave could probably be produced by sudden uplift of the auriculoventricular valves.

Now, with all these facts in view, the imagination is compelled to conceive a series of valve movements which may well give rise to modification of the normal first sound of the heart. If, for example, the heart is beating near its optimum rate, the inrush of blood into the ventricles during diastole may be supposed more or less completely to approximate together by its reflux currents the auriculoventricular curtains, much as, according to accepted theory, they are coaptated by auricular systole at the end of the normal period of diastasis. But in the hypothetical case the auricles contract at the moment when the auriculoventricular valves are being actively urged upward by the reflux of blood entering the ventricle during its diastole. In such a mechanical situation the auricular stream would presumably find for itself a place of least resistance of relatively small caliber between the valve curtains, traversing which the fluid and the valve would be thrown into vibrations essentially similar in origin and period to those characteristic of mitral or tricuspid stenosis. It is obvious, also, that the modification of the first sound of the heart thus produced, while perceptible as a presystolic crescendo phase, would rarely be obtrusive, but require for its detection the conscious attention of the examining ear.

Summary. In the structurally normal heart, especially in conditions of circulation excitement, the first sound frequently begins with a crescendo tone, simulating closely the faint and brief presystolic murmur or acute accent initiating the first sound in certain stages of organic mitral stenosis. This modification of the first sound may be pronounced either in the tricuspid or the mitral area or both. An attempt has been made to show that when the ventricles are filled at a certain rate the reflux of the blood current may be expected to bring the auriculoventricular valves sufficiently into approximation, so that the auricles in contracting must force a channel between them and cause vibrations of perceptible intensity, thus giving rise to an audible presystolic tone or murmur.[12]

[12] After this article was in type, a reference was found to a dissertation by Perlis (Practical Medical Series, 1909, i, 174), in which it is maintained that a functional presystolic murmur due to inorganic mitral stenosis may be heard in certain diseases and conditions.

GASTRO-INTESTINAL AUTO-INTOXICATION.[1]

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It seems a well-established fact that products of intestinal putrefaction, when absorbed without being properly changed or destroyed by the antitoxic activity of the intestinal wall and particularly of the liver, may cause systemic disturbances. On the other hand, it must be said that there is a general tendency at present greatly to overestimate the role which gastro-intestinal toxemia plays in the development of certain diseases. The fashion of the day, following the teachings of the French school, attributes to gastro-intestinal intoxication various diseases of the nervous system, the circulatory system, the blood, the muscles, the skin, etc. When, however, we examine the arguments which are given to prove the intestinal autotoxic origin of most of these diseases, it is astonishing to find how very few reliable facts gained by experimental research, by exact chemical investigation, or even by accurate clinical observation, have so far been advanced to support this theory for each case. While the occurrence of gastro-intestinal auto-intoxication has undoubtedly been demonstrated for some conditions, the application of this theory to numerous diseases merely on the strength of analogy has led to a great confusion of the whole question, particularly in regard to the magnitude of the role which intestinal intoxication plays in different diseases.

I have no intention of discussing in this short paper the different aspects of this difficult topic; I must confine myself to a few general remarks.

Under the heading of toxemias of gastro-intestinal origin are commonly included all intoxications which can be ascribed to the absorption of toxic substances formed in the alimentary canal by the action of digestive ferments or of bacteria. These endogenous intestinal intoxications are distinguished from so-called exogenous intoxications, which are principally caused by the taking of decomposed food. Together with this decomposed food, products of protein putrefaction, especially of the ptomain type, are usually ingested and thus act as severe poisons. It seems, however, that in a number of cases of apparent ptomain poisoning, the severe symptoms are not caused by the action of poisons formed in the food before it was eaten, but by the effect of intestinal infection of this food by bacteria, mostly of the paratyphoid type, so that these cases of apparent exogenous auto-intoxication are actually cases of endo-

[1] Read at a meeting of the New York Academy of Medicine, Section on Medicine, December 15, 1908.

genous auto-intoxication. Whether these and other severe pathogenic intestinal infections lead to the formation within the intestinal canal of such bodies as ptomains has not yet been definitely proved. There can be no doubt, however, that intestinal infections of this kind produce toxic substances the absorption of which causes more or less severe systemic disturbances, although as yet no such substances have been isolated.

One meets with other conditions in which the resemblance of the clinical picture to that of ptomain poisoning suggests that they are caused by the action of intestinal toxins. I refer to the well-known attacks of sudden and uncontrollable vomiting with extreme ababdominal pains and serious vasomotor disturbances, for which exogenous intoxication can be excluded and no other cause is found. These attacks, by the way, sometimes end fatally. There is, however, the possibility that these are cases of metabolic, not of gastro-intestinal auto-intoxication.

Again, in acute intestinal obstruction some of the violent symptoms are very probably due to the activity of highly toxic substances formed in the alimentary canal. In chronic intestinal obstruction such symptoms as fever, albuminuria, and severe disturbances of the nervous system indicate the possible action of enterotoxins. Among the nervous syndromes observed in cases with obstruction in the alimentary canal, I have to mention the form of tetany which occurs with gastric ulcer and pyloric stenosis. Gastric tetany is often pointed out as one of the most striking illustrations of toxemia caused by the absorption of toxic substances from the dilated stomach. This positive statement is not well supported by facts.

Some years ago I offered another possible explanation for gastric tetany with pyloric obstruction.[2] I argued that it is caused by an impoverishment of chlorides in the system, which is brought on through the constant loss by vomiting and lavage of great quantities of chloride with the highly acid gastric secretion. This theory, that the attacks are caused by the loss of a constituent necessary to the intermediary metabolism, and not by the toxic effect of a substance absorbed from the stomach, is well corroborated by experiments. According to Taylor[3] tetanic seizures are observed in dogs in whom the duodenum is cut across and the ends brought into external fistulæ, so that the gastric contents leave the body and its constituents fail to return to the circulation by intestinal resorption. It is possible that different and perhaps more satisfactory explanations may be found for other diseases, in which some features of the clinical picture suggest the effect of intestinal intoxication. I mention for such examples, tetany of children suffering from diseases

[2] Gastrosuccorrhea and Tetanic Attacks Occurring with Chronic Ulcer of the Stomach, Amer. Jour. Med Sci., April, 1904.

[3] Osler's Modern Medicine, vol. i, p. 277.

of the digestive tract, also certain skin eruptions, notably when occurring with pronounced gastro-intestinal disturbances, such as vomiting, diarrhœa, etc.

In some conditions it is the resemblance of the symptoms to those brought on by the known effect of poisons which justifies the assumption of intestinal intoxication. In no disease has this been demonstrated with more clearness than in cases of pernicious anemia. The investigations of Dr. Herter[4] have greatly added to our knowledge of the intestinal processes connected with such cases. The fact itself, that in a group of cases of pernicious anemia the hemolytic substances originate and are absorbed from the digestive tract, was known for some time. It was proved long ago for patients who harbored Bothriocephalus latus, but it was also recognized in cases in which no such higher parasites were present. I may mention here, that about twenty years ago, while still an assistant at Kussmaul's clinic, I discussed this topic in an article reporting a case of pernicious anemia, caused by chronic gastro-intestinal disturbances and cured by appropriate treatment.[5]

In other conditions which are usually attributed to intestinal intoxication the characteristic symptoms are not such as to suggest intestinal intoxication as the most probable cause for their development. In asthma dyspepticum, for example, the direct mechanical effect of the gas-distended abdominal viscera, usually found in these cases, and the effect of nervous reflex action, offer a much more plausible explanation. The fact that the attacks occur in connection with digestive disturbances and disappear with their improvement does not throw any light upon their assumed toxic character. The same may be said of numerous other conditions looked upon as intestinal toxemias, particularly diseases of the nervous and circulatory system, the skin, etc.

It is true that digestive disturbances provoke epileptic attacks, and that the treatment of such disturbances, especially the proper care for regular evacuation of the bowels, reduces, perhaps more than any other form of treatment, the number of attacks. Yet the good effect of such treatment itself is not sufficient to show that the digestive disturbances are the primary cause of the epileptic condition, and still less that this is effected by the action of toxins of intestinal origin.

I am far from underestimating the importance of digestive disturbances in the development of diseases of other organs. For some diseases we have a rational explanation other than autotoxic which makes us understand the causal connection between digestive and other systemic diseases. In other conditions this causal connection is still obscure. The fact that we do not know the cause does

[4] Jour. Biol. Chem., August, 1906; Bacterial Infections of the Digestive Tract, 1907.

[5] Zwei Fälle geheilter perniciöser Anämie, Berl. klin. Woch., 1890, No. 10.

not justify the present tendency to consider these diseases as principally caused by intestinal intoxication. If we are to accept this theory, we should be convinced by the demonstration of sufficient direct evidence in each instance. It should further be shown whether intestinal intoxication, when present, is a primary or only a secondary cause, and how great a role it plays in the pathogenesis of each condition.

Before discussing the direct evidence generally offered, it should be said that it is the same for conditions of distinct digestive disturbances as for those in which no gastro-intestinal symptoms at all, or only constipation, is present. The evidence consists principally of the appearance in the urine of products of intestinal bacterial disintegration. However, products of intestinal putrefaction are not the only substances accused of acting as toxins. It is further claimed that products of digestion, and even the digestive fluids themselves, may eventually have a toxic effect upon the system, when absorbed without being changed by the antitoxic activity of the intestinal wall and the liver. To dispose of the digestive fluids and the products of digestion first, I quote Taylor,[6] who, in his excellent article on this topic, states that there is not a single reported experimental or clinical fact which is explained by the assumption of the resorption and non-distoxication of the digestive juices; nor has it been shown that the products of normal digestion ever act as toxins.

We may now turn to the substances formed by the action of bacteria within the alimentary tract. The products of fermentation of carbohydrates, the acids, are, on the whole, harmless; increased in quantity (or with irritable organs in moderate amounts), they form a local irritant, causing vomiting or diarrhœa. When absorbed they are readily oxidized in the body; it is possible, however, that when formed in excessive quantities and quickly absorbed, they may lead to acid intoxication by robbing the system of great quantities of alkali. Acidosis observed in infantile enteritis is considered by some as of intestinal origin; others maintain that acidosis is always of metabolic origin, in this as well as other instances. Lactic acid, with bacterial gastric fermentation, seems to be well tolerated even when produced in large quantities. I had occasion to observe for a long period the unusual case of a very pronounced non-carcinomatous bacterial fermentation of the stomach, with constant production of great quantities of lactic acid, in which no symptoms appeared which pointed to toxemia. Of products of fat decomposition, cholin derived from lecithin is declared a highly poisonous compound, and it is said that some of the ill effects observed after eating excessively of such foods as eggs or calves' brains may depend upon the toxic

[6] Osler's Modern Medicine, i, 270.

action of cholin.[7] More weight is attributed to the products of intestinal putrefaction of proteins, which are chiefly held responsible for intestinal toxemia. A certain amount of bacterial disintegration of proteins in the intestines is normal. Normal is also the finding in the urine of a certain amount of those substances by which the degree of intestinal putrefaction is measured, that is, the ethereal sulphates and indican.

Estimation of the ethereal sulphates is a more reliable method of determining the degree of intestinal putrefaction. But since the indican reaction is the only test available to the general practitioner, it is very extensively used as an index of intestinal protein putrefaction, and the finding interpreted as a sign of auto-intoxication. In discussing the value of the indican test, it should first be stated that with adults living on a mixed diet a positive indican test is the rule. For a number of years I have examined systematically every urine of adults for indican and the cases in which the test was negative were very rare. The amount varies; it does not depend exclusively on the degree of intestinal putrefaction, but also on the degree of its absorption and other factors, so that there is no constant relation between the output of indican and the degree of intestinal putrefaction. As a rule, however, an outspoken excess of indican points to an increased intestinal putrefaction of proteins. The same considerations apply to an increase of ethereal sulphates.

I shall abstain from discussing the different conditions which may account for an excessive output of indican and of ethereal sulphates. In taking an increased intestinal putrefaction as its principal cause, I shall proceed to the questions: (1) What information do we obtain from the finding of increased intestinal putrefaction in regard to intestinal intoxication? (2) How far are we justified in interpreting excess of indican in the urine as a sign of intestinal intoxication? At present it is very difficult to answer these questions exactly. We have only to consider different possibilities.

Increased intestinal putrefaction may cause an increase of the known normal products of putrefaction, and it may lead to the formation of other new products of toxic character. It is often assumed that with increased intestinal putrefaction new substances are formed, which may possibly act as toxins. As long as such hypothetical substances have not been demonstrated it is of questionable value to estimate their presence and quantity by measuring the degree of intestinal putrefaction in general. The mere presence of such substances in the feces would not mean anything in regard to their toxic effect upon the system, because they may be detoxicated after absorption. All efforts, however, to find such enterotoxins in the urine have so far failed.

Especially the determination of the so-called urotoxic co-efficient

[7] Quoted from Wells, Chemical Pathology, p. 480.

cannot be considered a reliable method of diagnosticating the presence of enterotoxins in the urine, and still less of measuring their quantity. In regard to the known products of gastro-intestinal putrefaction, it must be said that according to our present knowledge, only few are toxic to any considerable degree, and these are produced in too small quantities to cause any appreciable effect.

Only for one product, for hydrogen sulphide, has it been demonstrated that it may be formed and absorbed in such quantities as to cause symptoms of intoxication. Such cases, first described by Senator[8] as hydrothionemia, are very rare; so are the cases described by Stokvis[9] as enterogenic cyanosis, apparently caused by the action of hydrogen sulphide of intestinal origin on the blood. These are characteristic clinical pictures of intoxication, which may be excluded from our general discussion on the diagnostic value of the excessive output of indican and the ethereal sulphates.

Of the other products of intestinal putrefaction, neither indol nor skatol, nor the sulphurous bodies and the amino-acids, are toxic to any considerable degree. Even an excess may be tolerated, as is shown by the observations that some individuals, who are otherwise healthy and show no signs of intoxication, eliminate very large quantities of indican and ethereal sulphates. It has been claimed that the same products of intestinal putrefaction, which are comparatively innocuous under ordinary circumstances, may become toxic under other conditions, especially when acting over a longer period. Herter,[10] for instance, maintains that in certain diseases the liver and other cells most concerned in oxidation show less ability to oxidize indol and to pair it with sulphuric acid, thus forming indican. He further claims that free indol has a toxic influence upon the neuromuscular system, and that this effect accounts for the development of such diseases as myasthenia gravis. Without discussing the merits of this theory, which has not yet been verified by the demonstration of free indol in the blood or in the urine, it must be stated that the excessive output of indican observed in such cases does not speak in favor of the assumed action of free indol. On the contrary, the finding of great quantities of indican shows that the pairing of the indol has been very satisfactorily perfected. This and similar considerations make it clear that excess of indican of itself is not sufficient to diagnosticate intestinal toxemia. Neither the indican test nor the estimation of the ethereal sulphates furnishes direct evidence that substances absorbed from the alimentary canal act as toxins. An excessive output of these bodies merely indicates that more protein than usual is undergoing putrefaction. This increased putrefaction may be innocuous. However, it must be admitted as possible that,

[8] Ueber Schwefelwasserstoff im Harn, Berl. klin. Woch., 1887.

[9] Zur Casuistik der autotoxischen enterogenen Cyanosen, Festschr. f. v. Leyden, 1902, p. 597.

[10] Bacterial Infections of the Digestive Tract, chapter on Indol and Indicanuria, 1907.

under certain conditions, products of increased intestinal putrefaction may cause systemic disturbances.

Finding so far no direct evidence, we have to turn to characteristic clinical symptoms for an indirect support of this theory.

In some disease we again find the only available support in the analogy to the effects of other known toxins, for instance, in neurasthenia. In neurasthenia we are familiar with an increased susceptibility to exogenous toxins like alcohol, tobacco, etc. It is assumed that a like increased susceptibility exists for the toxic effect of products of intestinal putrefaction, which under ordinary circumstances are well tolerated by the system. Taking this for a correct view, it would only show that the toxic effect of intestinal products may aggravate the neurasthenic condition. But it cannot be taken as an argument for the claim of some writers, that intestinal intoxication is an important primary cause of neurasthenia. I agree with those who consider heredity, worry, overwork, etc., as primary causes of neurasthenia. The increased susceptibility for toxins forms already part of the neurasthenic condition. Since digestive disturbances are often associated with neurasthenia, the effect of increased intestinal putrefaction may prove deleterious in creating a vicious circle. Similar considerations apply to the relation of digestive disturbances to other diseases of the nervous system.

Among the symptoms often quoted as characteristic of intestinal intoxication I have to mention albuminuria. The kidneys are very sensitive to poisons, and albuminuria occurs regularly when poisons are passed by the kidneys. Since albuminuria is a frequent symptom with diseases of the alimentary tract, it seems justified to attribute this to the effect of passing intestinal toxins. This may be, and certainly is, correct for some conditions, but not for all. From my own observations I have come to the conclusion that in one distinct group of digestive disturbances albuminuria is caused by increased acidity of the urine, with and without uric acid or oxalic acid sediment. When, in these cases, alkalies, especially preparations of magnesia, are administered in such quantities that the urine becomes slightly alkaline, the albumin disappears, to recur again with renewed increased acidity. In one of my patients I have been able to verify this numerous times during the course of years. In these cases, therefore, albuminuria is not caused by the direct action of enterotoxins on the kidneys, but by the effect of products of disturbed metabolism.

This leads us to a different aspect of toxemias connected with diseases of the digestive tract. Gastro-intestinal disorders often interfere with the activity of the liver, thereby disturbing the normal processes of metabolism. The manifold results of disturbed metabolism, symptoms of uric acid diathesis, the development of diseases of the kidneys, the arteries, the heart, etc., are often described as caused by the action of toxins of intestinal origin. If by the term

gastro-intestinal toxemia we mean the intoxication caused by the action of substances absorbed from the alimentary canal, then these are not cases of gastro-intestinal but of metabolic toxemia. Metabolic derangement may be instituted by gastro-intestinal disorders, but it may have other etiological factors, as mental strain and other disturbances of the nervous system.

It is necessary to distinguish strictly between gastro-intestinal toxemias and metabolic toxemias, especially at present, when Metchnikoff's ideas have taken such a hold on laity and profession, and when it is almost taken for granted that the so-called antifermentative treatment of digestive disorders is the most important and the most effective way of dealing with chronic systemic diseases.

Effective treatment of gastro-intestinal disturbances is certainly of great importance in systemic diseases. But I doubt very much whether this difficult task can be accomplished when the plan of treatment is based on the theory that the action of enterotoxins is at the bottom of it all. The fascinating theory of gastro-intestinal toxemia is in most instances still very much in need of support by objective facts. If we wish to gain more evidence and to clear up the many questions connected with this theory, we must show a more critical attitude than is generally displayed at present.

ACQUIRED CHRONIC ACHOLURIC JAUNDICE, WITH A BLOOD PICTURE AT ONE TIME RESEMBLING THAT OF PERNICIOUS ANEMIA.

By F. Parkes Weber, M.A., M.D., F.R.C.P.,

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The patient, Mrs. L. P., now aged thirty-eight years, was admitted under my care at the German Hospital in January, 1908, with blood changes characteristic of pernicious anemia, and on April 13, 1908, she was shown at the Medical Society of London as a "Case of Enlargement of the Spleen and Liver with Pernicious Anemia." She was again shown at the same society on February 8, 1909.

Up to the time of admission the history was that the patient had previously been healthy. About three years back she began to get paler, and about a year later she had an attack of deep jaundice (with pains in the right side), which was followed by dropsy. After that she gradually became weaker and paler and short of breath on exertion. Menstruation became scanty, and for a time there was complete amenorrhœa. She suffered from recurrent epistaxis.

She was troubled with coldness and numbness of the hands and feet; sometimes she had a sensation of pins and needles in the fingers, which would often "go blue."

In regard to the family history it was to be noted that her mother had died of liver disease, aged fifty-two years. The patient herself was a widow, who had been married twice. By her first husband she had two children, the first one born dead, the second one still living and healthy. By her second husband (who was addicted to alcohol) she had no children.

After admission to the hospital the condition noted was the following: The skin and conjunctivæ were yellowish. There was no pruritus or xanthoma. The legs were œdematous. The heart seemed slightly dilated, and there was a faint systolic murmur, apparently not due to valvular disease. The liver and spleen were both evenly enlarged. The liver extended two or three finger breadths below the ribs, and the spleen, which was hard, reached almost to the anterior superior iliac spine. The condition of the patient's mouth was not bad, though she had lost many of her teeth. Occasionally there was slight bleeding from the gums. Attacks of epistaxis were frequent. Ophthalmoscopic examination (Dr. C. Markus) showed numerous bright red, round, retinal hemorrhages in both eyes, chiefly in the upper halves of the fundi. The urine, of specific gravity 1013, contained albumin (1 per mille by Esbach's tube on admission), but no tube-casts. A later note of the urine stated it to be of specific gravity about 1015, of deep orange color, free from albumin and sugar, and giving no Gmelin's reaction for bile pigment, but containing excess of urobilin. The feces were never acholic.

Examination of the blood on January 31, 1908, gave the following result: Hemoglobin (by Haldane's method), 18 per cent.; red corpuscles, 900,000 in the cubic millimetre; white corpuscles, 6000; color index, 1. From a preparation of the same date Dr. A. E. Boycott made the following differential count of 500 white cells: Lymphocytes, 45.6 per cent; intermediates, 4.8; large hyalines, 2.4; neutrophile polymorphonuclears, 46; eosinophiles, 0.8; mast-cells, 0.4. During the count of 500 white cells he saw no ordinary normoblasts, but eight typical megaloblasts (that is to say, megaloblasts with cytoplasm staining as in the typical megaloblasts of pernicious anemia) and 16 smaller nucleated red cells resembling the typical megabololoblats except in regard to their relatively small size. There were a few polychromatophilic red cells and many punctate basophilic red cells. Poikilocytosis (as to shape and size of the red cells) was characteristic for pernicious anemia. No myelocytes were seen. An examination (May, 1908) of the contents of the stomach one-hour after a test breakfast consisting of dry bread and tea showed the absence of free hydrochloric acid.

The treatment in the hospital consisted chiefly of rest (at first rest

in bed), subcutaneous injections of the arsenic preparation, known as atoxyl, chalybeate medicines by the mouth, and acid glycerin of pepsin after meals. The amount of atoxyl injected was usually 0.1 gram (that is to say, 1 c.c. of a 10 per cent. solution) twice weekly. Calcium lactate was occasionally given on account of urticaria and subcutaneous hemorrhages. For about a week in September inunction of the splenic region with an ointment of the biniodide of mercury was tried, but with no definite result. The atoxyl treatment was commenced very soon after the patient's admission in January, 1908. It was continued with intervals till July 21, 1908, and then discontinued.

The patient has been over a year in the hospital. Progress was for a long time very slow and rather unsatisfactory, but decided improvement was observed in November, 1908, and has continued since then. The patient now feels and looks almost quite well. She has long been allowed to get up. She is free from jaundice, fever, and œdema and has gained considerably in body weight (133½ pounds in February, 1909, as compared with 118¼ pounds in February, 1908). The blood shows only slight anemia. The heart is not dilated, and shows nothing abnormal except a very slight systolic murmur best heard in the pulmonary area. The pulse frequency is 82 to 96 in the minute. The brachial systolic blood pressure is 100 mm. Hg. The spleen can only just be felt. The edge of the liver cannot be felt, though the organ is still probably somewhat enlarged. The retinæ are free from hemorrhages, but the patient continues to suffer from attacks of epistaxis at short intervals. Menstruation has recently recommenced, after a prolonged period of amenorrhœa. The stomach contents after a test meal show the presence of free hydrochloric acid. There is no excess of urobilin in the urine and the blood serum (kindly examined by Dr. Leonard Dudgeon on November 25, 1908) is free from bile pigment.

Several features of the patients' illness require special mention and consideration.

Abdominal Pain. At various times the patient has complained of pain or tenderness in the upper part of the abdomen, sometimes in the region of the liver, sometimes associated with fever. An attack about the middle of August, 1908, was associated with vomiting and temporary increase of the icteric tinge; a short attack in December was accompanied by a sudden rise of temperature to 103° F.

Jaundice. While under observation the patient was never deeply jaundiced, but the conjunctivæ for a long while were distinctly yellow. With the improvement in the general condition in November the jaundice entirely disappeared, and has not reappeared since. With a single doubtful exception (during a temporary exacerbation of the jaundice), bile pigment was never present in the urine; the feces were never acholic.

Temperature. At first there were recurrent periods of moderate fever, but since September there has been hardly any. The last occasion on which any fever occurred was on December 13, when a temperature of 103° F. was noted in association with abdominal pain.

Retinal and Other Hemorrhages. The retinal hemorrhages noted by Dr. C. Markus were peripheral in distribution, and chiefly in the right eye. They temporarily disappeared in the early part of April, 1908, but were noted again at the end of the month and in August and September. There are none to be seen at present. Recurrent attacks of epistaxis have been a feature throughout the illness. They still occur at short intervals, but for several months she has had no bleeding from the gums. In addition to these hemorrhages several bluish bruise-like subcutaneous hemorrhagic patches were observed during August and September, 1908. They were nearly all on the limbs. It is a question whether these various hemorrhages, as well as an urticarial tendency noted in February and March, 1908, might not be connected with diminished coagulability of the blood, or increased tendency to hemolysis (see later on).

Condition of the Blood. On May 19, 1908, the number of red cells in the cubic millimetre of blood had risen from 900,000 (on admission) to 2,000,000, and the hemoglobin from 18 per cent. to 35 per cent., but this improvement was hardly maintained. On July 4 the red cells were only 1,550,000, and the examination of blood films still showed the presence of typical megaloblasts. On August 15 another blood count was made. The red cells were then 1,300,000 and the white cells 8200 in the cubic millimetre. Dr. Boycott kindly forwarded the following differential count of 500 white cells: Lymphocytes, 51.2 per cent.; intermediates, 9.6; large hyalines, 1.2; neutrophile polymorphonuclears, 37.6; eosinophiles, 0.4; mast-cells, 0. He likewise reported that one neutrophile myelocyte and two nucleated red cells (small type with polychromatophilic cytoplasm) were found. The red cells showed moderate poikilocytosis and variation in size, but there were extremely few that were too big. The blood picture was certainly no longer that of pernicious anemia. By December 8 the number of red cells had risen to 3,277,000 in the cubic millimetre, and the hemoglobin was estimated by Haldane's method at 70 per cent.; nothing much abnormal could be made out by microscopic examination. On December 21 the red cells numbered 4,123,000, and the white cells 6250; hemoglobin, 74 per cent. Dr. Boycott, who again kindly examined blood films, found nothing abnormal in the red and white cells. His differential count of white cells then gave lymphocytes, 26.6 per cent.; intermediates, 2.2; large hyalines, 2; neutrophile polymorphonuclears, 68.4; eosinophiles, 0.8; mast-cells, 0.

It is interesting to note the increase in blood-viscosity accompanying the increase in the number of corpuscles. On February 23,

doubtless by reason of the corpuscular deficiency, the viscosity was very low; by Determann's clinical viscosimeter at 20° C. it was found to be only 2.6 times that of water at the same temperature. Estimated in the same way, on January 20, 1909, it was found to be 4 times that of water.

Owing to the hemorrhagic tendency manifested in the patient, her blood coagulability was repeatedly estimated by Sir A. E. Wright's coagulometer. The coagulation time varied on different occasions between ten minutes and fifteen minutes twelve seconds.

As it was supposed that excessive hemolysis might be the cause of the patient's acholuric jaundice ("acquired hemolytic jaundice" of A. Chauffard, F. Widal, Abrami, Brulé, etc.), the resistance of the red cells to hemolysis was repeatedly tested by Ribierre's method.[1] It was found that hemolysis usually occurred when a few drops of the patient's blood diluted with normal salt solution were added to a solution of between 0.46 and 0.44 parts per cent. of sodium chloride in distilled water. On one occasion it occurred still more readily (between 0.48 and 0.46), but lately the resistance of the red cells has apparently somewhat increased, for it now takes a solution of between 0.44 and 0.42 parts per cent. of sodium chloride in distilled water to produce hemolysis. The fragility, therefore, of the red cells, though rather high, appears not to be very greatly in excess of the normal. The hemolysis was not, however, examined by the method of adding the patient's red cells deprived of their plasma to the various solutions.

Dr. Leonard Dudgeon very kindly made some further investigations with the patient's blood on November 25, 1908, when her general condition was rapidly improving. From her blood he separated samples of her blood serum and of her red cells. He found that her serum had no hemolytic action on the red cells obtained from a normal (healthy) person or on her own red cells (that is, her serum possessed no autohemolytic action); neither did the blood serum from a normal person exert any hemolytic action on the patient's red cells. Furthermore, no hemagglutinative action was observed (by the methods employed by Dudgeon) on adding the patient's blood serum to a normal person's red cells, the patient's red cells to a normal person's serum, or the patient's serum to her own red cells.[2] No hemophagocytosis was observed.

On the whole, the present case seems to fit in best with the cases described by various authors on the Continent as examples of

[1] I am greatly indebted to Dr. Chapuis, one of the house physicians at the hospital, for much assistance in the clinical examination of the case, especially in regard to the hemolysis examinations, and to Dr. Trendelenburg, who replaced Dr. Chapuis for a short time.

[2] For Dr. Dudgeon's methods see his preliminary report "On the Presence of Hemagglutinins, Hemopsonins, and Hemolysins in the Blood Obtained from Infectious and Non-infectious Diseases in Man," Proceedings of the Royal Society, London, 1908, Series B, vol. lxxx, p. 531.

"Acquired Chronic Acholuric Jaundice with Splenomegaly and Anemia." The present case confirms Chauffard's opinion (as cited by Rolleston) that acquired cases of splenomegalic jaundice show more anemia and less jaundice than the congenital cases.[3] The remarkable feature of the case was the severity of the anemia which was at first accompanied by a myeloid (hemopoietic) reaction of the megaloblastic type, as in typical cases of pernicious anemia. It is not certain in the present case that the anemia was really hemolytic in origin, although the resistance of the patient's red blood cells to hemolysis was decidedly less than that noted in various healthy persons (controls) with whose blood hers was repeatedly compared.

Postmortem examinations have shown that some cases of acquired chronic acholuric jaundice are complicated by cholelithiasis. In the present case, indeed, the abdominal pains gave rise to a suspicion of gallstone trouble, while the recurrent attacks of epistaxis suggested the association of hepatic cirrhosis. The possibility of cholelithiasis secondary to chronic infection of the bile passages with typhoid bacilli was likewise considered, but the patient says she has never had typhoid fever, and her feces have been bacteriologically examined for typhoid bacilli with a negative result; moreover, Widal's reaction was twice tried, and on both occasions the result was negative.

The great improvement that has taken place in the patient's condition is very satisfactory, but this result cannot with certainty be attributed to the atoxyl treatment, as the period of the most decided improvement commenced a considerable time after the atoxyl treatment had been concluded. The main improvement appeared almost sudden enough to be termed a "crisis," but it was not accompanied by the blood changes (especially the large proportion of erythroblasts, some of them in stages of mitotic division) characteristic of typical "blood crises" in various forms of chronic anemia; that is to say, unless a true blood crisis of the kind commenced and ended during a specially long interval between two blood examinations.

[3] I have, however, satisfied myself that patients with congenital splenomegalic acholuric jaundice may sometimes (for a time at least) completely lose their icteric tinge. In other words, the jaundice may disappear and leave a condition of splenomegalic anemia only. Moreover, in the same family one child may suffer from congenital splenomegalic acholuric jaundice, whilst another child may suffer from chronic splenomegalic anemia without any manifest jaundice, though the blood picture be the same in both children. Evidently, in such instances the splenomegalic anemia is the same disease, whether it be accompanied by visible jaundice or not.

THE MEANS BY WHICH INFECTIOUS DISEASES ARE TRANSMITTED.

By Alvah H. Doty, M.D.,
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The belief that infectious diseases are commonly transmitted through the medium of clothing, baggage, cargoes of vessels, rags, money, etc., constitutes what is known as the fomites theory. This has been so universally accepted that even the most reliable and satisfactory proof which may be presented in opposition to it, is reluctantly admitted. The fomites theory in some form has existed from the earliest times, and therefore it could not have been suggested as the result of careful scientific investigation. Since the publication of the bacteriological researches of Pasteur and Koch about 1880, practical sanitarians who have given this subject close and careful observation have been slowly but surely securing indisputable evidence that the material or articles above referred to transmit disease only in rare instances. The fomites theory is plausible, and also very popular, for when it is accepted as an explanation of an outbreak of infectious disease in a community, a thorough and extended inspection to detect the presence of other cases, which necessarily involves considerable time and trouble, is not urgently called for. Less than ten years ago a statement that yellow fever was not transmitted by fomites would have been promptly rejected, and would have found but few if any supporters, because the evidence presented as proof that clothing, etc., transmitted this disease was believed to be conclusive. We now know that yellow fever is transmitted only by the mosquito, and that clothing, bedding, and even the discharges of the patient do not act as a medium of infection and are perfectly harmless. It has also been proved that the mosquito is the only medium of infection in malaria; however, until recently the theory prevailed that this disease was transmitted by miasma, or poisonous emanations from swamps. Furthermore, it was believed that plague was commonly transmitted by fomites; now satisfactory proof has been presented to show that this disease is communicated by the rat, through the medium of the fleas which infest them, and that fomites have but little if anything to do with the extension of the disease. Thus are rejected theories regarding the transmission of infectious diseases, which have not been subjected to careful study, or supported by scientific and practical evidence.

While many are willing to change their views regarding the means by which yellow fever and some other diseases are transmitted, they still claim that clothing, rags, etc., constitute an active agent in the transmission of smallpox, typhus fever, measles, and

scarlet fever, although there is an abundance of reliable evidence to show that this is not true. I may cite an instance that occurred in my own experience, which presented an unusual opportunity to study this subject. During the fall of 1892, an outbreak of two of the most infectious diseases with which we have to deal, that is, smallpox and typhus fever, occurred in New York City and continued during the following spring (1893), involving 842 cases of smallpox and 714 cases of typhus fever, almost all of which were removed from tenement and lodging-house districts. The Bureau of Contagious Diseases of the Department of Health then consisted of about 80 persons, including diagnosticians, medical inspectors, disinfectors, ambulance drivers, etc., who were more or less constantly in close and prolonged contact with the cases in overcrowded and ill-ventilated apartments. These men wore no gowns and were protected in no other way. They went frequently and freely to and from their homes; but during the entire period not a case of smallpox or typhus fever was transmitted to members of their families or their friends. Such evidence as this deserves the most serious consideration and points to but one thing, that the clothing and effects, particularly of well persons, is a very uncommon means of transmitting disease. We find statements that smallpox infection is carried through the air to a distance of half a mile or more; that clothing which has been laid away for years may act as a medium of infection when exposed, and that persons have become infected in rooms which, months previously, had been carefully disinfected. Such statements as these, in view of the knowledge we now possess, are accepted by practical sanitarians with a great deal of skepticism. Coincidences are chiefly responsible for the apparent proof of these statements, and I have many times, by continued investigation, proved this to be the case. It is true that the specific organisms of typhus fever, measles, scarlet fever, and some infectious diseases have not yet been identified. Still we are justified from our knowledge of known specific organisms to decide approximately the power of resistance, etc., of the unknown ones; besides, we have no good reason to assume that in the diseases just referred to the specific organisms are any longer lived or resisting than those which have already been identified.

During the outbreak of smallpox and typhus fever in New York City in 1892–93, to which I have already referred, the most careful and painstaking investigation was made in connection with every case brought to the notice of the Board of Health. As a result satisfactory evidence was constantly being presented to show that these diseases were transmitted by personal contact, and not by the clothing and effects of well persons. I remember distinctly one instance in which it appeared that the fomites theory only could satisfactorily explain the appearance of a case of typhus fever, and the sequel showed what a careful investigation will usually prove.

A case of typhus fever occurred in the lower and east side of the city, which at first was not correctly diagnosticated or reported to the Department of Health. Among those who visited the patient was a married woman living in the upper part of the city on the west side. At the time of her visit she took with her a friend, a woman from Philadelphia. Her husband did not accompany her, nor did he at any time visit the case. About ten days after the visit the married woman was taken ill with typhus fever. A day or so subsequent to this the husband was also taken ill with typhus fever. This seemed to be reasonable evidence that the latter was infected through the medium of the clothing of his wife or her friend, who remained well. If the fomites theory had prevailed, this history would have constituted sufficient evidence as to the manner in which the disease was transmitted, further investigation would have been discontinued, and the case would have been referred to as satisfactory proof that clothing transmits disease. However, this view was not taken, and the names and addresses of the man's relatives and friends, and the places visited by him, were carefully obtained and a thorough investigation made. This showed that the husband was in the habit of spending a large part of his time in one of the low saloons on the extreme west side of the city, and had been a constant companion of a group of men in this place, two of whom had been removed some time previously to the hospital with typhus fever. Although the period of invasion of typhus fever is short and persons affected with this disease as a rule promptly succumb, there were cases during this outbreak removed from lodging houses, cheap saloons, etc., who had been sick for a day or so without the disease having been detected, or the patient seeking relief, and there is no reasonable doubt that the man referred to contracted the disease through this source, particularly as it is well known that typhus fever is more easily transmitted in close and badly ventilated apartments. This may illustrate one of the coincidences that not infrequently occurs; in the absence of a careful investigation it lends valuable support to the fomites theory, and, if accepted, discourages thorough investigation to discover the real cause of outbreaks of infectious disease, and in this way aids in their extension.

It is also a common belief that rags and money are usual agents in the transmission of disease, and it is also believed that certain infectious diseases, such as measles, scarlet fever, and diphtheria, are greatly increased at the opening of the school term, because infection is transmitted to others by the clothing of well children in whose families some form of infectious disease exists. Considerable literature is presented in support of these theories.

During the past few years municipal health officials, particularly those in the larger cities, have had ample opportunity to investigate the means by which infectious diseases are transmitted in schools.

School corps have been appointed, consisting of medical inspectors whose duties require them carefully and frequently to inspect each child in the public schools. The work has presented some rather astonishing and particularly satisfactory results; children are constantly being found in the schools apparently well, but who are in the active stage of infectious disease in a mild or unrecognized form, or are in the convalescent stage. In schools where the secretions from the throat and nose are subjected to a bacteriological examination, many children are found to have diphtheria bacilli, but without visible evidence of the disease; and what is believed to be an ordinary sore throat is frequently proved to be scarlet fever, with the accompanying desquamation. Besides children apparently affected with simple coryza are often found to have measles. These results are constantly being presented wherever this careful investigation is in operation, and offers a sane and practical explanation of the spread of infectious disease in these institutions.

The belief that rags used for commercial purposes, particularly those which are soiled, transmit disease is a favorite theory, and is apparently reasonable, as rags come from all parts of the world—frequently from areas where infectious diseases are known to exist. It is often reported, and alleged satisfactory evidence produced to show, that infectious diseases occur in paper warehouses as the result of this means of infection. I have given this part of the subject long and careful investigation, and have yet to find conclusive or satisfactory evidence that either domestic or foreign rags act as a medium of infection. In addition to my researches in this country, I personally investigated this subject in Egypt at the rag depots at Alexandria. Egyptian rags consist principally of the worn-out garments of the natives, which consist of one piece similar to an ordinary night-gown, and is usually worn directly against the skin. Therefore, as smallpox, or some other infectious disease, is almost always present in Egypt, it is fair to assume that the rags there would be a good test as to the possibility of this material acting as a means of infection. The carefully prepared statistics furnished me by the English sanitary authorities, who are in charge of this work in Egypt, showed not the slightest evidence that the men, women and children who were constantly in contact with these rags in the sorting room were more prone to infectious disease than those following other pursuits. It has been suggested by those who believe in the fomites theory, that probably foreign rags do not transmit disease because a long period occurs between their collection and the time that they are used in the manufacture of paper here. My investigation in Egypt related to domestic rags which had recently been collected, although when they reach this country they become foreign rags.

Paper money is also regarded as an ordinary medium of infection. Fortunately, in this instance, it is not very difficult to reach a scien-

tific and practical conclusion in regard to the danger from this source, as it is only necessary carefully to investigate the subject in connection with the employes of banks and other places where money is constantly being handled. Such an examination will show that these persons do not have infectious diseases any oftener than anyone else. The most effective test may be found in the statistics of the Treasury Department at Washington, in relation to which I have already conferred with the United States Treasurer. It will be shown that although many clerks are constantly handling and rehandling enormous quantities of old and filthy money, there is no proof that this article is a medium of infection. If an employee of a rag house or a bank becomes ill with an infectious disease, it is usually heralded as proof that these articles transmit infection: as a matter of fact, this is not scientific proof. In the consideration of this subject, it seems to be almost entirely forgotten that persons who work among rags or in banks are subject to the same outside exposure and danger of infection that others are, and the fact that they are ill with one of these diseases may be superficial, but not scientific, evidence that money or rags transmit disease. This would be indicated if proper proof were presented to show that those who handle rags or money contract infectious diseases oftener than others. Exhaustive investigation proves that this is not the case. If rags and money transmitted disease, the evidence of it would be forced upon us—we would not have to look for it.

In connection with this, I may add that a careful bacteriological investigation made by Mr. Warren W. Hilditch, at the Sheffield Laboratory of Bacteriology and Hygiene at Yale University, regarding paper money as a medium of infection,[1] goes a long way to confirm the statement which I have many times publicly made regarding this subject. Mr. Hilditch's article concludes as follows: "From the observation that I have made it would seem that the bacteria present on paper money are non-virulent, and that the form most present are the air forms. . . . One conclusion that may be drawn after a careful study of this subject is that money constitutes an unimportant factor in the transmission of disease. . . ."

Practically, it is only the plausibility of the fomites theory that supports the belief that cargoes of vessels transmit disease, because there is no satisfactory or reasonable proof of it. Fears are sometimes expressed that old rugs, etc., from the East and other sections where infectious diseases are more or less constantly present may act as a medium of infection. The danger of this exists almost entirely in the minds of those who present these theories. The diseases in the Orient which are to be feared are principally cholera and plague. Old rugs and curios are constantly being brought from that part of the world to New York—still these diseases do not appear as a result of the exposure of these articles.

[1] Popular Science Monthly, August, 1903.

At the New York Quarantine Station cargoes of vessels are not disturbed even when infectious diseases are found on board, and not the slightest evidence has been presented to show that this policy has in any way contributed to their extension. On the other hand, it relieves commerce of an enormous expense and great delay, which the treatment or disinfection of a vessel's cargo would involve. What has been said relative to cargoes of vessels may also be applied to baggage, etc.

Curiously enough, the concern with which some regard clothing, rags, etc., as a means of infection does not extend to a consideration of the real danger of discharges from the respiratory and alimentary tract of persons ill with certain infectious diseases. No one doubts that the stools of typhoid fever patients transmit this disease; still, they are carelessly handled in the sick room. The means which are usually taken to render them harmless consist in the addition of a liquid disinfectant which may or may not be left for a short time in contact with the mass, the mixture afterward being thrown into the water-closet or privy vault. Discharges from the respiratory tract are often treated in practically the same way. Experiments made at the New York Quarantine Laboratory show that disinfecting solutions, such as chloride of lime, carbolic acid, etc., do not disinfect when used in the manner above described. The experiments consisted in treating fecal matter in receptacles with the agents above referred to. It was found that at the end of twenty-four hours only the most superficial part of the mass was affected. Similar experiments conducted by Dr. William H. Park, of the Department of Health of the City of New York, produced practically the same result. This would indicate clearly enough that dis infecting solutions added to discharges are of no value unless they are thoroughly incorporated with every portion of the mass to an extent which would be hardly practical to carry out, and I am quite sure that no nurse or other attendant upon the sick would take the time to do it. Therefore, infected stools under the ordinary treatment are not disinfected, and remain a medium of infection. Discharges from the respiratory tract are also frequently dropped into receptacles containing disinfecting solutions or powders, for the purpose of destroying the specific organism which they contain. As mucus is particularly tenacious and resisting, it is very doubtful if the disinfectant, with which this comes in contact, ever properly fulfils the requirement.

It is generally believed that privy vaults may be disinfected by the use of various solutions and powders: such a thing is practically impossible. If a single discharge cannot be disinfected by lime, carbolic acid, etc., certainly a receptacle containing a large amount of fecal matter in all stages of consistence and decomposition cannot be. In small communities which have no sewerage system, it is required that each house shall have a receptacle for the deposit of

fecal matter. In the presence of typhoid fever, for instance, the proximity of these receptacles or privy vaults to wells (which are commonly near by) is very properly regarded as not an uncommon means by which infection is transmitted. So far as the ordinary privy vault is concerned, there is not the slightest reason or justification for its existence. If a receptacle of this kind is necessary it should be placed above the ground, and not below it. This method of construction answers the requirement, and insures an air space underneath, which practically prevents ground contamination and the transmission of infection even when only ordinary care is observed, and renders any attempt at disinfection practically unnecessary.

It is evident from a careful review of this subject that there is not a uniformity of opinion regarding the usual means by which infectious diseases are transmitted. The bar to this has unquestionably been the popularity of the fomites theory, not because it has been supported by scientific evidence, but because it presents a prompt and easy explanation for outbreaks of infectious disease; besides, there are many who believe we should discard no theory which may possibly aid in the prevention of infectious disease, whether or not it is reasonable or scientific.

No one who has had long practical experience with infectious disease doubts that in some unusual and rare instances clothing, rags, or almost any article may transmit disease. However, this is not the most important factor for practical sanitarians to consider. In order to deal successfully with outbreaks of infectious disease, we must ascertain what are the usual or ordinary means by which they are transmitted, as these must decide the character of regulations to be enforced and the precautions which must be taken to prevent their extension. General regulations cannot be made to deal with rare exceptions. If an attempt is made to do this, both the public and commerce are unfairly and unjustly treated, and unnecessary annoyance, delay, and expense is involved. Modern practical sanitation does not insure complete or absolute safety in its operation, and efforts made to secure this almost always defeat the end in view.

The acceptance of the fomites theory as an ordinary medium of infection aids in the extension of infectious disease, chiefly because public health officials who believe in this cannot fully appreciate the imperative necessity for an extended and thorough inspection to detect the presence of other cases, which are usually responsible for the extension of infectious disease. What I have said in regard to this subject is not intended to imply that we should dismiss entirely from our minds the possibility or probability of fomites transmitting disease. The clothing, bedding, etc., in contact with persons actively sick, which sometimes contain discharges, etc., if exposed to other persons within a comparatively short period may

transmit disease, and it is for this reason that the disinfection of the contents of the apartment of the patient is justified. As I have already stated, in rare or unusual instances, the clothing or effects of well persons may be the medium of infection; however, this happens so seldom that it is only entitled to secondary consideration.

Indisputable evidence has been presented to show that infectious diseases are usually transmitted directly from the sick to the well. Besides, in recent years we have learned of another common means by which infectious diseases are transmitted, of which conclusive proof has been presented, and that is that well persons act as carriers of pathogenic organisms without presenting constitutional or local evidence of it. This refers to the diphtheria and typhoid organisms, and particularly to the cholera bacilli. There is no doubt whatever that cholera is frequently spread by well persons in whose alimentary tract the germ of cholera is present, and who are responsible for outbreaks of this disease by infecting in various ways food and drink without exhibiting any symptoms of the disease themselves. Then we have the transmission of disease by insects. With such known means of infection as these we are hardly justified in considering clothing, etc., as usual agents of infection, particularly as these articles can hardly be regarded as a proper media for organisms even for a brief period; besides, organisms which usually cause infectious diseases in human beings contain no spores.

There can be no question that the secret of success in dealing with outbreaks of infectious disease consists in the most thorough and extended inspection and investigation to discover the presence of the sick, and, when found, to secure their prompt and careful isolation. This is applicable to tuberculosis also, and there is no doubt that the proper and logical means of exterminating or controlling this disease is by isolation. In addition, we must always bear in mind the probability of infection being transmitted by well persons or carriers, and also insects. These constitute the modern and scientific methods of treating these emergencies. If they are employed, there will be no outbreak which cannot be brought under control, and the ravages which infectious diseases have caused in the past, and which are even now occurring in certain sections of the East, need never occur again. The fomites theory is not in harmony with these principles, neither can those who support this theory fully understand the frequency with which outbreaks of infectious diseases are caused by mild, ambulant, or unrecognized cases, which I believe constitute one of the most common and dangerous factors with which we have to deal. In my experience, both in municipal work and at Quarantine, abundant evidence of this is constantly being presented, and, furthermore, that this type of disease can be detected only by a most careful inspection; for instance, two of the cases of bubonic plague which were discovered on incoming vessels at the New York

Quarantine during the past few years were so mild that the men affected were not confined to their quarters, but were able to perform at least part of their work, and would have passed an ordinary visual examination. It was not until their temperatures were taken, and a careful glandular examination made, that the disease was identified. Very mild cases of smallpox, yellow fever, etc., are not uncommonly found on incoming vessels, chiefly because they are looked for. If these cases were not detected on shipboard and were allowed to proceed to their destination and there cause secondary cases of these diseases, the fomites theory would unquestionably be called upon to explain the outbreak. The value of a careful inspection and investigation in the presence of infectious disease is not only of great importance at the point where the outbreak occurs, but frequently aids in the detection of similar cases among the patient's family or friends who may recently have been with him, and who are residents of other towns. This extended investigation is frequently most fruitful in its results.

The knowledge we now possess regarding the means by which infectious diseases are transmitted, justifies a great diminution in the disinfection hitherto performed. The disinfection of the clothing, baggage, etc., of well persons who have been exposed to the sick, or have been in infected areas, the cargoes of vessels, rags, money, etc., should be eliminated from this treatment unless there is some specific reason for employing it. The cargo of a vessel cannot be disinfected unless it is removed from the ship, notwithstanding statements to the contrary. This is an exceedingly expensive process and involves great delay and annoyance to all concerned, and I have yet to meet with an instance where it was justified. Gaseous disinfectants cannot be depended upon to penetrate, and are of value only when the material to be treated is fully exposed or spread out. Therefore, the introduction of sulphur dioxide, or formaldehyde, into the hold of a vessel for the purpose of disinfection would practically be valueless. As rags for commercial use usually consist of small pieces, which could not be properly spread out, it would be necessary to disinfect them in bulk by steam. This process would be exceedingly expensive, particularly if applied to imported rags which arrive in tightly rolled bales. Although steam has great penetrating power, it could not be depended upon to enter sufficiently far into these packages to insure a germicidal action, and the packages would have to be opened for treatment at the port of destination, as it would probably never be done at the point of collection, or at the port of embarkation. The question of the disinfection of money can be dismissed in a word—any attempt to carry this out would be farcial and of no practical value.

The requirement of the fomites theory frequently involves the detention at home of those who have been exposed to infection, or in whose home infectious disease may exist, for the reason that the

clothing of these people is believed to transmit disease. In many instances this is a great hardship, and frequently seriously interferes with the family revenue. The modern view of this subject does not justify this procedure, particularly when the patient is properly isolated and cared for. In these instances it is only required that a daily examination shall be made of suspects, which should include the use of the thermometer to early detect the invasion of disease.

Unnecessary disinfection has led to a misunderstanding of its function and value. It is frequently left for members of the family or other laymen to carry out, whereas, when it is required, its application should be under the immediate direction of the physician or health official in charge, and should be performed in a proper and scientific way. We still find saucers of carbolic acid, etc., placed under the bed and about the sick room, and it is not uncommon to find that small pieces of sulphur are occasionally burned in the room for the purpose of disinfecting it. It is hardly necessary to say that these attempts to disinfect an apartment are perfectly worthless, and only annoy the patient and belittle disinfection.

I have already referred to the inefficient methods which are used at present, both in hospitals and dwelling houses, in the treatment of the discharges of those who have infectious disease, which I am sure all agree constitutes a very important means of infection. From a practical standpoint there is but one agent that can be depended upon to destroy the organism in this matter, and that is heat, either by boiling or incineration. The principle of modern sanitation, which governs this detail, requires that discharges, refuse, etc., shall be destroyed as near as possible to the point of production, and heat is the proper agent for the purpose.

Boiling water is one of the most valuable and practical disinfectant we possess, but unfortunately it is not fully appreciated. Its action is sure, it is safe, simple, and without expense. The destruction of the specific organism in the discharges of the sick by boiling water is under ordinary circumstances an easier and a more practical method than incineration. At present, a simple boiler is successfully used at the Swinburne Island Hospital, New York Quarantine, for this purpose. Its action is quick and made practically odorless by having a close fitting cover, from the top of which an outlet connected with a flexible tube conducts the steam outside of the window. An apparatus suitable for this purpose can be made by any tinsmith for a small price, or if necessary can be improvised by the use of ordinary household appliances. In the country this method would practically prevent danger from infection through the medium of a privy vault.

The practical abandonment of the fomites theory not only does not, as many believe, involve danger, but substitutes for it a scientific, practical, and successful means of dealing with outbreaks of infectious diseases.

UNCINARIASIS IN PANAMA.

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It is not the object of this paper to discuss the general pathological anatomy of fatal cases of uncinariasis, but rather to note the presence of hook-worms in the routine autopsy cases, and the fact that the old world hook-worm (Ankylostomum duodenale) and the new world hook-worm (Necator americanus or Uncinaria americana) are often present in the same case. A few points relating to the pathology of the jejunum will be discussed—especially hemorrhage and inflammation in the submucosa. No attempt will be made to review the literature which has been very carefully compiled by A. Looss.[1]

During the year from September, 1907, to September, 1908, 420 autopsies were performed at the pathological laboratory of the Ancon Hospital, C. Z., Panama, and in the last 232 cases the intestinal parasites were studied with some care. In this series of 232 consecutive cases the presence of hook-worms was recorded in 72 cases (31 per cent.). In 39 cases no mention of intestinal parasites was made, but we feel sure that in these cases the hook-worm was absent. If, however, this number is deducted from 232, the percentage of cases infected with hook-worms rises to 37 per cent. The whites in this series numbered 37, with but 2 cases of uncinariasis, a frequency of only 6 per cent. Both cases were in Spaniards. The whites included Americans, Spaniards, Greeks, Italians, etc. The blacks came principally from the Southern West Indies and Central America. If we deduct the white cases, we find the frequency of hook-worm infection to be 36 per cent. Heavy infections with hook-worms were not common, and cases showing 100 or more worms occurred only five times in the 232 cases. Both old and new world varieties were present in every one of the severe infections, Necator americanus predominating. Of the entire 420 cases, only 9 showed a heavy infection—that is, over 100 worms. These cases came from Jamaica (3), Martinique (2), Costa Rica (1), Colombia (1), Antigua (1), and Guadeloupe (1). As the sanitary conditions on the canal zone are excellent and most of the laborers wear shoes, we may say that the great majority of these cases were infected in their native lands. The length of their stay on the Isthmus varies from one month to two years. Cases among native Panama negroes show light infections, but both types of the hook-worm are present.

The old world hook-worm (Ankylostomum duodenale or Uncinaria duodenalis) was present in 39 cases (17 per cent.), and in 25 cases was found associated with the new world variety (Necator ameri-

[1] Handbuch der Tropenkrankheiten, 1905, i, 116; Centralbl. f. Bact., 1907, xxxv, 752.

canus). The new world hook-worm was present in 49 cases (21 per cent.), and predominated in all heavy infections.

It is not a difficult matter to separate the two types of hook-worm. The parasites were washed in running water until clean, then placed in Petri dishes on a glass plate painted black. After some practice, one can separate the two types by using the lens of a dissecting microscope. The old world worm is thicker, and has a milky look. It is more active, and has a blunt head end. The mouth and œsophagus are over twice the size of those of the new world variety. The new world worm has a more delicate body, with a smaller head end and œsophagus. It has a more translucent appearance, except when containing blood. The two types of worms separated with the aid of a low-power lens are placed in saline solution in rows on slides and examined under a No. 3 objective (Leitz). The four prominent hooks found in the mouth of the old world worm are easily seen, and establish the diagnosis. In many cases the dorsal rays of the caudal bursa in the males were studied, as well as the location of the vagina in the females. Ankylostomum duodenale in appearance corresponds very closely with Ankylostomum caninum, which is present in great numbers in all the native dogs.

232 Cases: 37 Whites; 195 Blacks.

	White.	Black.	All Cases.
Hook-worms present	2 cases (6%)	70 cases (36%)	72 cases (31%)
Old world hook-worm, Ankylostomum duodenale	2 cases (6%)	37 cases (19%)	39 cases (17%)
New world hook-worm, Necator americanus	2 cases (6%)	47 cases (24%)	49 cases (21%)
New and old world hook-worm together in same case	2 cases (6%)	23 cases (12%)	25 cases (11%)
Whip-worm (Trichuris triciura)	3 cases (8%)	37 cases (19%)	40 cases (17%)
Ascaris lumbricoides	(0%)	19 cases (10%)	19 cases (8%)

The following table comprises 62 cases, in which the worms were studied and classified. In some cases the males and females were not separated and numbered, but this was carefully done in the majority of cases. The intestinal tract opened, but not washed, was looked over by a labaratory assistant, who picked out most of the worms; then the person performing the autopsy examined it carefully and collected any remaining parasites. We feel sure that very few worms escaped this routine examination. Whip-worms and round-worms were recorded in the 232 cases. Whip-worms were present in 40 cases (17 per cent.)—three of the cases were in white patients. Ascaris infection was found in but 19 cases (8 per cent.), all in black patients; 134 cases were free from intestinal parasites (58 per cent.).

Home.	Uncinaria.				Whip-worm.	Ascaris.	Remarks.
	Americana.		Duodenalis.				
	F.	M.	F.	M.			
Jamaica	91	43	17	4			Worms contain fresh blood.
Jamaica	60	26	3	1			Worms contain fresh blood; many submucous ecchymoses.
Jamaica	2	2	16	22			Worms contain fresh blood.
Jamaica	(20)						
Jamaica	2	1			1		
Jamaica	2	4					
Jamaica	4	2					
Jamaica	1				2	1	
Jamaica			(3)				
Jamaica	(8)						
Jamaica		1				1	
Jamaica	+		+		+		
Barbadoes	14	5	3	2	3		Many submucous hematomas.
Barbadoes	8	2	1		1		
Barbadoes	2	2	6				
Barbadoes	2	1			1		
Barbadoes	(5)						
Barbadoes			2	4	1		
Barbadoes			2	2			
Barbadoes	3						
Barbadoes			1				Many submucous hematomas.
Barbadoes			1			2	
Barbadoes	(1)						
Barbadoes			1				
Barbadoes	Few hook-worms.						Many submucous hematomas; blood cyst contains hook-worm.
Barbadoes			+				
Martinique	(200)		3	4			Worms contain fresh blood.
Martinique	82	77	9	8	1		Many submucous ecchymoses, worms contain fresh blood; small blood clots adherent to mucosa.
Martinique	9	6	4		1		Worms contain fresh blood.
Martinique	10	4			6		
Martinique	3		15	4	1		
Martinique	3	1			9		
Martinique			2				
Martinique	(1)		(1)				
Martinique	+		+				Many submucous hematomas; blood cyst contains hook-worm.
Antigua	(100)		(60)				
Antigua	2		(30)				
Antigua			1	1			
Grenada	(38)		(1)				
Grenada	(35)		(2)				
Grenada			1				
Gaudeloupe	(11)		1	1	2		
Gaudeloupe	(6)				1		Many submucous hematomas; blood cyst contains hook-worm.
Trinidad	19	9	25	30			Many submucous ecchymoses;
St. Vincent	6	5			1	2	many submucous hematomas;
St. Lucia	(1)				1		small blood clots adherent to mucosa.
. . .	6	3					Many submucous hematomas;
Spain	(20)		(3)				blood cyst contains hook-worm.
Spain	2	2	3		3		
Cuba	2						
Panama	(75)						
Panama	(10)		(2)		12	1	
Panama	11	3	1	1	1		Worms contain fresh blood.
Panama	8	2				12	
Panama			2	1	4		
Panama				1			
Colombia	23	19			3		
Colombia	8	7	1				
Colombia			(2)			3	
Colombia	1						
Costa Rica	1		1				Worms contain fresh blood; many submucous hematomas.
Costa Rica	(100)		(10)		3		Many submucous ecchymoses; worms contain fresh blood.

A glance at the table under "Remarks" will show that our findings do not correspond with those of Ashford and King,[2] in Porto Rico, concerning intestinal ecchymoses and the presence of blood in the digestive tract of the hook-worms. They state that ecchymoses are very rare, only a few being found in a single case in their series, and that the worms very rarely contain blood.

Ecchymoses in the mucosa or submucosa were a common finding in the jejunum infected with hook-worms, both at the point of attachment of the worm's head and in other places where presumably the worm had been feeding. When the area of hemorrhage exceeded 3 to 4 mm. in diameter, it was referred to as a "submucous hematoma." A note of "ecchymoses" or "submucous hematomas" was made in 18 cases, and in several cases they were very numerous (No. 1441), giving a speckled appearance to the mucosa.

The blood cysts described by Bilharz and Grassi were not infrequent, and were often found with an included hook-worm—7 cases (Figs. 1 and 2). Such cysts measured from 5 to 10 mm. in diameter; more rarely the worm was completely buried in the submucosa with very little extravasation of blood (Fig. 2). Sections show great numbers of large intestinal bacilli associated with the worms buried in the submucosa, and these bacteria cause an extensive inflammation of the adjacent mucosa and submucosa which are infiltrated with large numbers of polymorphonuclear leukocytes and eosinophiles.

This series of cases shows that the worms often contain fresh blood, both the new and old world forms, more frequently the latter. A note that "many worms contain fresh blood" was made in 16 cases, and it was unusual not to find a few worms containing blood in an infected case when the autopsy was performed within two or three hours after death. It was noted that in cases of advanced anemia the worms rarely contained blood, while in cases showing no anemia the worms very often contained fresh blood and ecchymoses were more evident. This may explain the findings of Ashford and King, who maintain that the hook-worms very rarely contain blood. These writers examined fatal cases of uncinariasis which showed extreme anemia. When worms which were full of bright blood were removed from the intestinal mucosa and placed in water or 0.8 per cent. saline solution, they soon discharged the blood. Under the microscope one could watch the red blood cells and blood-stained fluid being poured out from the mouth and anus. This explains the fact that worms found in the feces or at an autopsy performed several hours after death may contain very little or no fresh blood.

Looss and others insist that the worms live on the intestinal epithelium, and cannot digest the blood. It seems rather hasty to

[2] Jour. Amer. Med. Assoc., 1907, xlix, 471; Boston Med. and Surg. Jour., 1907, clvi, 415; Report of the Commission for the Study and Treatment of Anemia in Porto Rico.

decide that a worm can digest epithelial cells from the human host, yet cannot digest red blood cells.

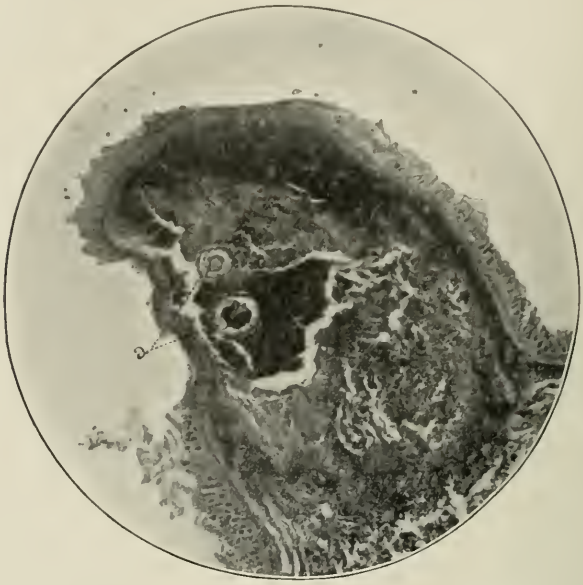

FIG. 1.—Blood cyst below the mucosa of the jejunum, with cross-section (*a*) of included hook-worm.

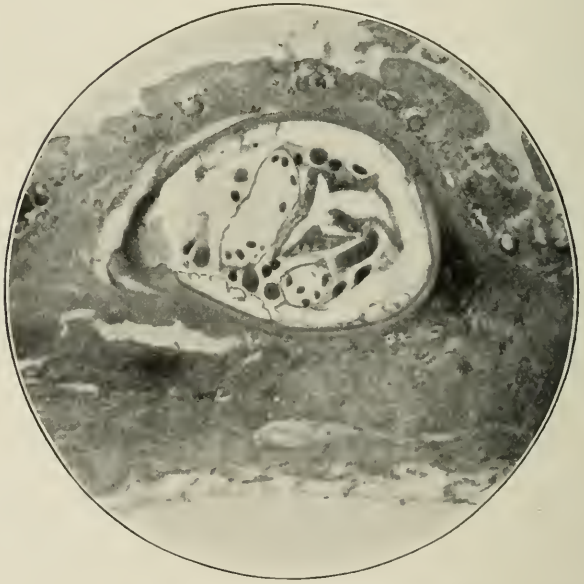

FIG. 2.—Hook-worm in the submucosa, with very little extravasation of blood; cross-section of the body of a female worm.

The following fact proves that the hook-worm can live and thrive for a considerable length of time in blood. Hook-worms removed from the "blood cysts" in the intestinal mucosa were invariably very active and vigorous and contained fresh blood. Sections through some of these "cysts" showed an invasion of the blood and adjacent submucosa by leukocytes and various wandering cells in considerable numbers. Such a reaction required from two to four days, during which time the worm lived in the blood and ingested more or less of it, as can be seen in sections which show the

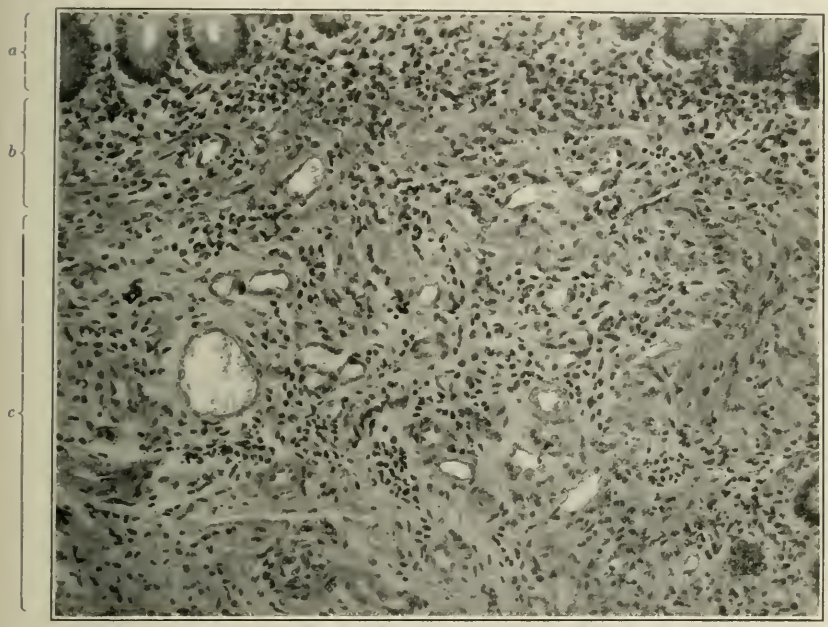

FIG. 3.—Jejunum in a fatal case of uncinariasis, showing extensive scarring and inflammation of the intestine. (*a*) Bases of crypts of Lieberkühn; many polymorphonuclear leukocytes in the interglandular tissue; (*b*) muscularis mucosæ distorted and scarred, invaded by many wandering cells, many of them eosinophiles; (*c*) submucosa, which is much thickened and full of every type of cell—eosinophiles, polymorphonuclears, and "polyblasts." There is much scar tissue; new-formed bloodvessels are seen.

worm's intestine to contain red cells and granular detritus. It is admitted by all writers that blood is found in the worm's intestine, and it seems reasonable to conclude that the parasites live on both the blood and the epithelium, but we feel sure that the blood is the main factor when the worms are feeding on a fairly normal mucosa. In a heavily infected case, when the mucosa is very anemic, somewhat thickened, and infiltrated with leukocytes (Fig. 3), it is fair to suppose that the worms may live chiefly on the epithelium and mucus. Sections of worms show the intestinal canal containing red blood cells, coagulated albuminous detritus, and epithelial cells.

Many writers (Ashford, Baker,[3] Yates[4]) believe that uncinaria produces some obscure hemolytic toxin analogous to that of Bothriocephalus latus, but this has never been demonstrated. Clinically, too, the blood pictures are very different (Boycott,[5] Adami[6]), as the anemia of uncinariasis is distinctly of the secondary type. I hope to show in another article[7] that the hook-worm of man does contain a hemolytic factor which is constant but very weak. It is present in all parts of the worm, probably in the intestinal canal—not in the cephalic glands alone.

That the hook-worm does not contain a powerful hemolysin is shown by a study of the "blood cysts" described above (Fig. 1). The worms often remain in these sumbucous blood clots for a few days at least, as shown by the number and type of wandering cells in the edge of the clot. The worms are not injured by their stay in the blood, yet there are well-preserved red blood cells in all parts of the "blood cysts." It seems that this is almost a perfect experiment performed by the hook-worm: the parasite is included in a small sac of the host's blood, and all conditions are as they should be to demonstrate the presence of a hemolytic agent—yet the blood cells are not dissolved in any great number. Here we have a small amount of blood isolated in the human tissues and containing an active hook-worm. In many cases there is less than 0.5 c.c. of blood in the cyst, and the parasite lives in this medium one, two, or three days, yet can cause no marked hemolysis. The parasite by its activity has deprived its host of a fraction of a cubic centimeter of blood, but cannot hemolyze this small amount during a considerable interval of time. Thus, the worm can cause the loss of more blood through damage to the mucosa than it can hemolyze under favorable conditions. It does not seem likely, therefore, that this same worm could secrete a hemolytic substance which, taken up by the human intestine, could act on the circulating blood, where surely conditions would be less favorable for hemolysis than in the "blood cysts." I believe that this observation alone is sufficient to exclude the presence of a powerful hemolysin in the hook-worm—a hemolysin which could be absorbed by the host and cause an advanced secondary anemia.

The cause of the anemia is not clear: various explanations have been advanced. Some writers disregard the blood abstracted by the worm, and the numerous "bites" which often bleed after the worm has changed its feeding ground (No. 1441). Loeb[8] has demonstrated a substance, which definitely inhibits the blood coagulation, to be present in the cephalic glands of Ankylostomum caninum, and this may be a factor in promoting the oozing of blood from the

[3] Brit. Med. Jour., 1903, i, 718. [4] Johns Hopkins Hosp. Bull., 1901, xii, 364.
[5] Brit. Med. Jour., 1907, ii, 1318; Jour. Hyg., January, 1904, iv, No. 1.
[6] Montreal Med. Jour., 1903, xxxii, 186. [7] Jour. Exp. Med., 1909, xi, No. 2.
[8] Proc. Path. Soc., Philadelphia, June, 1904; Centralbl. f. Bact., 1906, xl, 740.

mucosa. It has been proved that this worm is a bloodsucker, and the daily abstraction of a small amount of blood cannot be disregarded. Many writers attribute the anemia to an obscure toxin produced by the cephalic glands of the worm and absorbed by the host. No proof of the existence of any such toxin has been furnished, and until this proof is forthcoming such an explanation is merely evading the question.

Others (Ferguson)[9] have advanced the theory that the "bites," which are always greater in number than the parasites, cause little foci of infection in the mucosa, a chronic inflammation, and anemia. The anemia may be due to the poisonous products absorbed from these foci of inflammation in the intestine. This, I believe, is the most important factor in the production of the anemia, and explains the fact that some cases with relatively few uncinariæ may show a profound or even fatal anemia. Case 1137 may be cited as an example of this: A male, Jamaican, negro, aged sixty years, shortly before his death, had 15 per cent. of hemoglobin. There was nothing unusual about the gross pathology of the viscera. The tissues were all very pale. The heart was greatly dilated, with watery, pale clots—hydremia. The lungs were pale and cushiony. The liver and kidneys showed a definite yellowish brown pigmentation and anemia. The bone marrow was very firm, pink, and cellular. The jejunum contained between 100 and 200 hook-worms, but the types were not identified. The mucosa was very pale and smooth. There were no notes concerning the presence of fresh blood in the worms, and no notes regarding ecchymoses. We cannot assume that the worms were discouraged by the anemia, abandoned the mucosa, and were carried out in the feces, for we find cases with even greater anemia and many hundreds of worm in the intestines. Microscopic sections of this jejunum are most interesting (Fig. 3). The mucosa shows rather short villi and crypts, with a little distortion of the architecture of the glands, due to an increase in connective tissue. The interglandular tissue in the villi and at the base of the gland tubules shows a marked infiltration with polymorphonuclear leukocytes and eosinophiles, which in some fields are more numerous than the mononuclears. The muscularis mucosæ is thickened, scarred, and infiltrated with wandering cells of all types. The submucosa is thickened and scarred. It is not sharply separated from the muscularis mucosæ, and many polymorphonuclear leukocytes and eosinophiles are present. Accumulations of large mononuclear wandering cells are found about the vessels in the submucosa. The outer muscle coats are normal. This is a very definite pathological change, a picture of diffuse inflammation, evidently of some duration, due to the hook-worm, as evidenced by the eosinophiles.

When one considers the amount of tissue which was involved in this

[9] Brit. Med. Jour., 1907, ii, 1320.

change—several feet of the jejunal mucosa—it is not difficult to account for an advanced anemia. Perhaps the same explanation may hold for the liver necroses which were present in this case, and similar to those described by Yates in a fatal case of infection with the new world hook-worm. Many of the liver lobules show a necrosis of the central half or third, where the liver cells are quite hyaline, and wandering cells are invading such areas. The other liver cells show some fatty infiltration, and a deposit of a finely granular, yellow pigment.

Reasoning from this series of cases, it seems probable that the anemia of uncinariasis is due to two factors at least: (1) Loss of blood through feeding of the worms and oozing from damaged areas where the worms have broken the mucosa; and (2) infection of the intestinal mucosa and submucosa, due to bites of the worms which give points of entrance for intestinal bacteria.

Summary. 1. Hook-worms are found in the routine autopsy examinations in 31 per cent. of the cases.

2. The old world hook-worm (Ankylostomum duodenale) is present in 17 per cent. of the cases, together with Necator americanus in 11 per cent. of the cases.

3. The new world hook-worm (Necator americanus) is present in 21 per cent. of all the cases, and predominates in all heavy infections.

4. The whip-worm (Trichuris triciura) is present in 17 per cent., and Ascaris lumbricoides in 8 per cent. of the cases.

5. Hook-worms removed from the intestinal mucosa a few hours after the death of a patient very often contain fresh blood.

6. Ecchymoses and small submucous hematomas are not rare findings in an infected jejunum.

7. Hook-worms ingest both blood and epithelial cells and presumably digest both.

8. The anemia is due not only to direct loss of blood through activity of the hook-worms, but to a diffuse inflammation of the mucosa and submucosa of the jejunum. This inflammation is caused by the bites of the parasites which damage the mucosa and give entrance to the intestinal bacteria.

9. The severity of the anemia depends upon the number of hook-worms, and upon the intensity and extent of this diffuse inflammation. This second factor may explain the cases of fatal anemia in which only relatively few hook-worms are present.

10. Study of the "blood cysts" containing hook-worms shows that the parasites can live in blood for days and cause no active hemolysis of this blood—strong evidence against the presence of a powerful hemolysin in the hook-worm.

In conclusion, it is a pleasure to express my sincere thanks to Colonel W. C. Gorgas and Dr. S. T. Darling, for permission to use the material at the Ancon Laboratory, and to Dr. A. H. Dodge, who assisted in much of the routine work.

MALIGNANT NEWGROWTH IN CHILDHOOD.

I. MALIGNANT DISEASE OF THE UTERUS, OVARY, AND VAGINA IN CHILDREN.[1]

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Reviewing the literature of malignant newgrowths in children one is struck with the increasing frequency with which diseases of the genital organs in children are being recorded. About twenty years ago, when I commenced to call attention to malignant diseases of the pelvic organs in female children, the references to be found in the literature were few indeed. I now make a special plea for the early recognition of malignant diseases in children. These diseases are no longer medical curiosities, but will be seen by all of us if they are looked for with care and intelligence. It is the early recognition that they need, because with early recognition early radical treatment will reduce the present rather apalling mortality. I make this appeal to the general practitioner; it is to him that we must look for any advances in our knowledge and treatment of these diseases. He has the first chance, and upon his diagnostic skill and acumen depends our ability to classify, study, and successfully treat the little sufferers.

Anyone who is specially engaged in the study of pediatrics will agree with the statement that female children, even young infants, are subject to the same affections of the genital organs that are found in adult females; in fact, they are probably more prone to at least one of them, notably gonorrhœa. We must also remember that many diseased states brought to our attention by adult women have their etiology in conditions which existed long before puberty was established. We rarely discover malformations of the genital tract in children, but they are studied with great ease in the adult. Salpingitis and localized peritonitis are rather frequent in little girls. Marx[2] reports the postmortem examination of fifteen children who had symptoms of salpingo-oöphoritis similar to those found in the adult. In five of them the Fallopian tubes contained pus and their uterine attachments were sealed.

The examination of a child, no matter how young it may be, for the detection of malignant disease, is in all respects similar to that of the adult, except the vaginal and bimanual examination. We rarely examine young children by the vagina; in fact, it is hardly

[1] Read by invitation at a meeting of the California Academy of Medicine, San Francisco, October 22, 1908.

[2] Gaz. de gynec., November 15, 1895.

justifiable, unless the disease is manifestly vaginal or a vaginal discharge is evident. When it is necessary to examine the vagina of a young child, the cystoscope of Kelly, No. 8, 9, or 10, as suggested by himself, will be found the most available instrument. It is introduced with great ease and little pain if cocaine has been used; the child soon gets used to it, and if necessary applications of remedial drugs may be made to the different areas. The knee-chest position is of great value in making these examinations; of course, the usual dorsal position is of great value, but in children it is less valuable than in the adult. Of greatest value of all in children is the rectal examination; a combined rectal and bimanual examination is ideal, no matter how young the child may be. If this be made a matter of routine, we shall be surprised how often grave disease is detected in these little bodies. Some years ago Carpenter, of London, was the anesthetizer for an examination of a case in obstinate constipation in a little girl which had resisted all usual methods of treatment. The examination was carried out by means of the rectal and abdominal method and a myeloid sarcoma growing from the anterior surface of the sacrum was found to be the cause of the obstinate constipation.

In young children, from birth to the fifth year, the relatively greater length of the examining finger in comparison with the small pelvis and small abdominal cavity permits wide excursions over practically all of the pelvis and almost the entire abdomen. Of course as the child grows older this examination is of less value, because the size of the body now nearly approaches that of the adult. The rectal examination is best made under an anesthetic; this may not always be necessary, but a gynecological examination in a resisting child is devoid of value. The child is placed in the dorsal position, the legs are flexed on the thighs and the thighs on the abdomen. The pelvis is elevated as for a cystoscopic examination. The examiner now places the left hand on the abdomen and the right index finger in the rectum. A routine examination is now made first of the left side of the pelvis and abdominal cavity and then to the right up to or above the level of the umbilicus, which can usually be reached with great ease. Any abnormal growth not only in the sexual organs and abdominal viscera, but in the intestines and retroperitoneal structures, may be readily recognized.

In little children the finger in the rectum and the hand on the abdominal wall are separated only by the thickness of the comparatively thin walls of the child's abdomen, and a growth can be outlined with startling exactness. The iliac, hypogastric, and umbilical regions are within one's grasp; the important regions of the appendix and the contiguous structures are almost within one's hand, and we can at once recognize the so-called intestinal matting; even a slight degree of peritonitis, just sufficient to agglutinate the omentum, may be recognized by the combined examination in expe-

rienced hands, particularly those hands that have learned to know that when peritonitis is present the intestines move en masse, as it were, and do not quickly glide under the fingers as the healthy gut will.

A note of warning must be sounded here in regard to the danger of mistaking a partially filled bladder for a tumor or a peritoneal exudation. The means of guarding against this error is self-evident; I only mention it because those of us who are teaching sometimes see the younger practitioner fall into error. We must also guard against mistaking a pus collection or serous accumulation for a filled bladder.

Let us for a moment consider the anatomical peculiarities of the female pelvic organs in children. The sacrum and in consequence the rectum are almost straight. One will miss the well-known hollow of the sacrum, and the large lax ampullar distention does not exist. In young children, those who have not walked much, the bladder is about the size and shape of an egg, the base downward, and its site is almost purely abdominal. As the child walks more the bladder sinks lower in the pelvis, but until about puberty it can readily be displaced into the abdominal cavity by an examining finger, so loose is its attachment. The infantile uterus has but little body; it is practically a thin cord made up mostly of the cervix which at first appears unduly bulky. It lies comparatively high in the pelvis, but can be rolled between the fingers and the symphysis pubis. In young children the ovaries are at the side of the pelvis close to the external iliac artery: the pulsation of this vessel is one of the guides for their location, the other being the sickle-shaped uterosacral ligaments. It is to be understood that these landmarks are subject to as many variations in the child as in the adult. The usual variation is a higher location, but we must remember those cases of congenital prolapse of the uterus to which I have called attention,[3] and we must distinguish this prolapse from congenital hypertrophic elongation of the cervix. The prolapse may be of any degree, and is usually without marked symptoms; indeed, some of the children have performed the functions of the bladder and rectum in a normal manner. These cases of congenital prolapse are likely to be associated with lumbosacral spina bifida and rectal ectropion, often with clubbed feet, sometimes with hydrocephalus and hypertrichosis.

Other causative factors have been thought to be an abnormally large size of the pelvis, an enlargement of the uterine body or cervix or of the entire organ, and increased abdominal pressure; such a case for example as that of Stepkowski,[4] who saw a woman, aged twenty-five years, with complete prolapse of the uterus and vagina

[3] Cyclopedia of the Diseases of Children, Keating and Edwards.
[4] Gaz. lek in Przegl. Chir., la Gynic., 1897, No. 1.

which was caused by excessive vomiting when she was but thirteen years of age, and that of Karczewski,[5] who saw a girl, aged thirteen years, with complete procedentia developing gradually from carrying heavy burdens. Uterine displacement other than prolapse also occurs in very young children. Spencer[6] reports a case of retroflexion of the uterus in a newborn child, and Berli,[7] one of congenital displacement in the newborn. Carpenter,[8] who was one of the pioneers in the rectal examination of children, says that a uterus in a child, aged about two years, is an inch long and an inch broad at the fundus, the tubes about one and three-fourths inches, the right ovary five-eighths inch in length, and the left one-half inch in length, and each about one-sixth inch in diameter. The ovaries vary from five-sixteenths inch long by one-fourth inch broad in a child a few weeks old. The organs measure one and one-half inches by one-half inch in a child approaching puberty. Ovaries show some variations in size in children of similar ages. One ovary is not infrequently decidedly larger than its fellow. The Fallopian tubes for clinical purposes are about equal to the vas at a similar age, at their narrowest part, but they gradually enlarge as they pass along to the fimbriated extremity; in length they vary from a little over one inch to a little over three inches, according to age.

As I have already stated, the two anatomical aids to the recognition of these structures when examining by the rectum are the iliac arteries and the uterosacral ligaments. These ligaments, as Keating and I showed many years ago, form a curve surrounding the rectum in children, and when the finger is introduced and passed gently upward their sharp edge is readily recognized. Following up this edge we surely come to both the tube and the ovary which can be outlined by pressing toward the bony wall of the pelvis. The ureters are also palpable under the same guidance; in this way an impacted calculus has been detected in a tuberculous ureter in a child.

In examining children we must ever bear in mind that displacements of the ovaries and tubes into the inguinal or crural openings are not extremely unusual, and if we find difficulty in locating them in the pelvis, these latter structures, the navel, the ischiadic, and obturator foramina should be examined; it is well also to remember that these prolapsed ovaries may be cystic and cause considerable difficulty in diagnosis.

Diseases of the uterus, tubes, ovaries, and vagina in childhood are far more frequent than is generally supposed by those who have not given special attention to the study of those diseases in the very young. Even as late as 1888 no less an authority than Lusk[9]

[5] Gaz. lek in Przegl. Chir., la Gynic., 1897, No. 1.
[6] Trans. Obstet. Soc., London, 1892–93, xxxiv, 25 to 28.
[7] Univ. Med. Mag., 1893–94, vi, 185.
[8] Pediatrics, 1896, i, 481
[9] American System of Gynecology,

wrote that no case of uterine sarcoma is known to have occurred previous to puberty, a statement as incorrect then as it is now. The literature on this subject, for the most part, is scattered throughout periodical publications, but of recent years Howard A. Kelly and I have made an attempt to consolidate these scattered publications into standard text-books.[10]

To show the frequency of pelvic disease in children it is only necessary to say that Bland Sutton has recently collected one hundred cases of ovariotomies performed on children under sixteen years of age. Weil[11] has added sixty cases under ten years of age, and I have over 275 references to pelvic and abdominal diseases in little children. But to those of us who have engaged in this study for a number of years, the manner in which writers report their cases is extremely disappointing, when we are endeavoring to elucidate new facts. Because pediatrics as a special department is new, it is most important to record every detail in the clinical history and in the operative procedures, but most important of all is the later history of the patient, as by this knowledge alone shall we be able to formulate the factors upon which to base reliable conclusions both as to the immediate prognosis and the expectancy.

An interesting point is that all forms of diseases of the adult female pelvis have been recorded in childhood. Again, in the child certain growths seem to have certain selective age periods: sarcoma and dermoids usually occur under the third year; and ovarian cysts become more frequent about puberty, although they are seen at all ages, even from the earliest infancy. Chiene[12] removed one at the early age of three months; Boullard[13] saw a newborn infant in whom both ovaries contained a large number of cysts; Schultz[14] saw an ovarian cyst in a still-born child; Doran[15] reported proliferating cysts in the ovary in a seven-months fœtus; and Lederer[16] operated upon a fifteen-weeks-old infant for cystic degeneration of the left ovary and in addition found the right ovary in the inguinal canal. Some very early successful cases of removal of sarcoma of the ovary are recorded: Harris,[17] for example, removed such a tumor in a child, aged twenty-two months; Hoffman's[18] patient was aged thirty-three months (fatal result); Cameron's[19] was forty months old (also fatal result). Byford[20] had a successful case at the age of four years and eight months. Altogether a large number of sarcomatous ovaries have been observed and recorded in children from an early age

[10] Cyclopedia of Diseases of Children, Keating and Edwards, first, second, and third editions, 1889 to 1901, J. B. Lippincott Co., Philadelphia, Pa.; Gynecology and Abdominal Surgery, Kelly and Noble, vol. i, p. 809, W. B. Saunders & Co., 1907, Philadelphia, Pa.

[11] Johns Hopkins Hospital Bulletin, March, 1905, xvi, No. 168.

[12] Edin. Med. Jour., June, 1884.

[13] Bull. soc. anat. de Paris, 1854, xxix, 15.

[14] Verhandl. d. Gesellsch. f. Geburtsch. in Berlin (1857–58), 1859, xi, 55 to 57.

[15] Proc. Path. Soc. London, 1880–81, xxxii, 147 to 150.

[16] Allg. Wein. Med. Zgt., 1895, x, 253.

[17] Amer. Jour. Obst., October, 1904, 530.

[18] Ibid., xxxvi.

[19] Glasgow Med. Jour., 1889.

[20] Chicago Med. Record, 1891–92, ii.

up to about the eighteenth year (see Tables I and II)—a later age excluding their study for this paper. Nor is carcinoma of these structures unknown during the earlier years of life: Brown[21] reports one encephaloid disease of the right ovary in a child (age not stated); Marjolin's[22] remarkable case was aged but one year, but had a very large cancerous tumor. Weil[23] reports an adenocarcinoma of the left ovary in a girl, aged five years. The ovary was the size of a large cocoanut, freely movable, and was removed with the left tube without any hemorrhage. The child made a remarkable recovery. Stolypinsky[24] reports a carcinoma of the ovary in a girl, aged eight years; Leopold[25] one in a girl aged nine years; Corse[26] one involving the uterus and ovaries in a girl, aged twelve years, and Michel[27] removed by operation a carcinoma of the ovary that was the size of a man's head, in a girl aged sixteen years. In two years she died of recurrence. At autopsy a large tumor in the left side of the abdomen was found, with metastasis to the liver and kidneys, but most interesting of all is the fact that the microscope showed the tumor to have a chorion-epithelioma-like structure.

Many more instances of the early involvement of the ovary by malignant disease could be cited from the literature, but sufficient has been given to make good my contention that the disease can no longer be considered extremely rare, even at the very early periods of life.

Let me briefly consider the clinical modification in the disease due to the tender age of the subject. The age, by the way, is absolutely no contra-indication to operation. It is true that the very young do not stand this sort of surgery as well as the very old; nevertheless their resistance is surprisingly good and sufficiently reliable to warrant extensive surgery in the face of malignant involvement of removable structures. Malignant ovarian tumors are more common in children than in very old women; of these, the ovarian dermoids and sarcomas are perhaps the most frequent. We must not forget the presence of carcinoma at this period of life. Many of the earlier cases are no doubt rather carelessly worked out in regard to the microscopic diagnosis. Cullen says that adenocarcinoma of the ovary is extremely deceptive unless many sections from the various parts of the growth are examined and then it will be found that the advancing edges are most characteristic of the carcinomatous arrangement.

[21] Lancet, London, 1858, i, 456.
[22] Bull. soc. de chir. de Paris (1860), 1861, 2 s. 1, 667–72.
[23] Ibid.
[24] Rev. de gynecol. et de chir abd., viii, 708.
[25] Centralbl. f. Gyn., 1874, xviii.
[26] Trans. Coll. Phys., Philadelphia (1856–62), 1863, n. s., iii, 336
[27] Zentralbl. f. Gynäk., 1905, No. 14.

The symptoms of a malignant pelvic growth in a child are very likely, in my experience, to be extremely indefinite. Certainly this is so in the earlier stages and in the two cases of which I have notes, the sarcoma of the ovary had reached such a size as to be noticeable externally and the parents discussed the change of contour in the abdominal segment; but the children denied all abnormal abdominal sensations. Neither of these children had emaciated at all, and were brought to me because they had a large growing abdomen.

If we are unable to satisfy ourselves that the diagnosis is complete and that malignant disease or other serious pelvic abnormalities do not exist, there can be no possible objection to an exploratory incision in the child when life seems threatened by an abdominal growth. If possible, one should remove the growth at this time; if not, the simple incision carefully made and carefully attended to afterward does not add to the danger of the already existing disease. I always prefer the median incision in children, made one inch above the symphysis pubis and extending upward one and one-half to two inches.

This incision renders all the organs accessible, and its closure is perhaps less subject to hernial complications. The peritoneum in the child is a very delicate membrane; it should be picked up in a fold and very slightly nicked, when air will enter the general cavity and the incision may be cautiously enlarged. It may be torn the full length of the incision, but I prefer the use of scissors. The entire pelvis, contiguous and related organs, should be carefully and systematically explored. My rule has been the uterus first, because it is more readily recognized, then the right and the left palpation of ovaries, tubes, broad ligaments, ureters, kidneys, and appendix. This can be very gently and quickly done in the child, and with but little manipulation, so accessible are the organs. Of course, very simple means should be employed first, but I never hesitate to advise exploratory incision if the diagnosis is not perfectly clear or if there is any question at all of malignancy.

In the child, unlike the adult, the abnormal pelvic conditions are always of a serious nature. An abdominal tumor in a child is likely to be either a dermoid cyst, sarcoma, or adenocarcinoma of the ovaries, malignant or cystic renal diseases, or certain somewhat rare and atypical retroperitoneal renal disorders. So we must of necessity look upon the presence of an abdominal tumor in a child as of serious import, and exhaust all methods of arriving at an accurate diagnosis.

Henning's[28] treatise, written nearly thirty years ago, is still the best on the diseases of the female sexual organs in children. He tells us that dermoid cysts are apt to be carried into the years of puberty, on account of their slow growth and lack of early symptoms;

[28] Gerhardt's Handbuch der Kinderkrankheiten.

TABLE I.—Malignant Disease of the Ovary in Children.

Reporter	Age	Tumor	Remarks	Reference
Doran	7 mos. fœtus	Cancer	.....	Trans. Path. Soc., London, 1882
von Franqué	fœtus	Sarcoma	.....	Weil; Johns Hopkins Hosp. Bull., March, 1905, No. 168
Hollander	9 mos.	Spindle- and round-cell sarcoma	Operation: recovery. This case appears also in Table II	Deut. med. Woch. Vereins, Beilage, 1896, xxii, 16; Ped. 1896, ii
Marjolin	1 yr.	Cancer of ovary	Very large	Bull. soc. de chir. de Paris (1860) 1861, 2 S., i, 667-672
Harris	1 yr. and 10 mos.	Sarcoma and endothelioma	Recovery; twisted pedicle	Amer. Jour. Obstet., October, 1904
Pick	2 yrs.	Spindle-cell sarcoma	Vagina and all pelvic organs infiltrated	Arch. f. Gynäk.,1894, xlvi, p. 592
Gibb	2½ yrs.	Cystic sarcoma	.....	Glasgow Med. Jour., lx, No. 1, p. 33
Evers	2½ yrs.	Sarcoma	.....	St. Louis Courier of Med., August, 1884
Hoffman	2 yrs. 9 mos.	Sarcoma	Death	Amer. Jour. Obstet., xxxvi
Cameron	3 yrs. 6 mos.	Sarcoma	Death	Glasgow Med. Jour., 1889
Smith, F. C.	3 yrs.	Myxosarcoma	Recovery; vaginal and uterine polyps, uterine subperitoneal growth. This case also appears in Table II	Ibid
Schwartz	4 yrs.	Adenocystoma	Recovery	Arch. f. Gyn., xiii, 4
Foerster	4 yrs. 6 mos.	Sarcoma	Death	Amer. Jour. Obstet., xxxi
Byford	4 yrs. 8 mos.	Sarcoma	Recovery	Chicago Med. Rec., 1891-2, ii
Weil	5 yrs.	Adenocarcinoma	Operation; recovery. Tumor size of a cocoanut; removed with tube; no hemorrhage; alive and well at time of report	Johns Hopkins Hospital Bull., March, 1905, xvi, No. 168
Demme	5 yrs.	Congenital fibrosarcoma	.....	Jahrsb. des Jernnerschen Kinder spetales ze Berne, 1881
Turner	6 yrs.	Medullary sarcoma of both ovaries and of peritoneum	Death	Trans. Path. Soc., London, 1892-3, xliv, 110
Page	6 yrs.	Sarcoma	Recovery	Lancet, December, 1895
Parry	7 yrs.	Carcinoma	Death	Lancet, 1907, ii, 1607
Croom	7 yrs.	Sarcoma	Recovery; last report two years later; still well. Croom considers premature sexual development in relation to ovarian tumors	Edin. Med. and Surg. Jour., 1893, p. 689
Olshausen	8 yrs.	Carcinoma	Autopsy; no operation	Weil, Ibid
Stolypinsky	8 yrs.	Carcinoma	Result not stated	Rev. de gyne. de chir. abdom., viii, 708
Gussenbauer	8 yrs.	Carcinoma	Recovery	Wien. med. Woch., 1894, No. 41
Sharp, W. H.	8 yrs.	Cystic encephaloid	Not stated	N. Y. Med. Jour., 1874, xix, 52-54
Chenoweth	8 yrs.	Sarcoma	Recovery	Amer. Jour. Obstet., xv
Malius	9 yrs.	Cystosarcoma	Death	Lancet, 1890, i, 1174
Forbes	9 yrs.	Myxosarcoma	Recovery	Australian Med. Jour. Melbourne, 1894, xvi
Leopold	9 yrs.	Carcinoma	Death	Centralblatt f. Gynäkologie, 1894, xviii
Palmer	9 yrs.	Cancer	.....	Trans. Med. Soc., Dist. Columbia
McBurney	10 yrs.	Sarcoma	Recovery	Annals Surg., 1895, xxi, 706
Wagner	10 yrs.	Sarcoma	Recovery	Arch. f. klin. Chir., xxx, 704
Croom	11 yrs.	Sarcoma	Recovery	Obstet. Trans. Edin., xiv, 93

Reporter	Age	Tumor	Remarks	Reference
Edwards, Wm. A.	11 yrs.	Sarcoma	Recovery; child made excellent operative recovery; died in six weeks from general metastasis	Present communication
Kelly. H. A.	12 yrs.	Sarcoma	Perfect recovery; tumor large enough to fill a hat; died 2 years later from dysentery	Cyclop. Dis. of Children, Keating and Edwards, iii, 739
Eckhardt	13 yrs.	Sarcoma	Recovery; double oöphorectomy as opposite ovary was cystic; 1½ years later showed no recurrence	Deut. med. Woch., 1895, S. 96
Bode	13 yrs.	Sarcoma	Recovery; tumor filled entire abdomen; grew under sheath of abdominal muscles	Central. f. Gyn., Bd. xviii, 1171
Anderson	13 yrs.	Sarcoma	Recovery; died in few months from recurrence	Bland Suttou., Diseases of Tubes and Ovaries
Marien	13 yrs.	Sarco-epithéliome	.....	Univ. Méd. du Canada, Montreal, 1908, xxxvii, 317-320
Joüon	13 yrs.	Tumeur maligne de l'ovaire	Recovery	Gaz. med. de Nantes, 1907, 2, S., xxv, 17
Smith	13 yrs.	Carcinoma	Death; both ovaries diseased	Lancet, 1874, ii, 501
Gage	15½ yrs.	Sarcoma	Recovery; tumor filled abdomen and very adherent	Jour. Amer. Med. Assoc., December, 1894
Thornton	15 yrs.	Carcinoma	Death	Med. Times and Gaz., 1883, i, 211.
Von Szabo	15 yrs.	Carcinoma	Death; hemorrhage into abdominal walls and intestinal adhesions	Arch. f. Gyn. Bd. xxxii, S. 193
Michel	16 yrs.	Carcinoma	Recovery; died in 2 years from recurrence; growth size of man's head removed at operation; autopsy, large tumor on left side of abdomen; metastasis to liver and kidneys; microscope showed that the tumor had a chorionepithelioma-like structure	Zentralbl. f. Gynäk., 1905, No. 14; Amer. Med., August 26, 1905, p. 377
Homans	17 yrs.	Sarcoma of pelvic organs, omentum and mesentery	Died of shock in three hours; about 13 pounds of encephaloid cancer removed	384 Laparotomies for Ovarian Disease; Sawyer & Sons, Boston, 1887
Binaud	17 yrs.	Primary (large) cancer of ovary	Perfect recovery; cancer was very voluminous(31b. 7,oz.) with ascites; a perfect cure by ovariotomy	Jour. med. de Bordeaux, 1894, xiv., 345
Donhauser	Children	Malignant ovarian tumor	.....	Albany med. Ann., 1906, xxvii, 20–30
Mo. G.	Child, age not stated	Sarcoma	Death	Osservatore, Torino, 1876, xii, 84
Fenemenow	Child, age not stated	Sarcoma	Death	Rev. de gyn. et de chir. abdominale, 1904, viii, 708
Piwowarski	Child,	Malignant ovarian tumor	The author considers this to be a rare tumor	Ueber einen seltinen Fall von malignen Ovarialtumor bie einen Kinde, Berlin, 1905, xliv, 8
Brown	Child, age not stated	Encephaloid of ovary	.....	Lancet, 1858, i, 456
Amann	Child, age not stated	,Carcinoma	Exploratory incision	Weil, Ibid

TABLE II.—Malignant Disease of the Uterus in Children.

Reporter	Age	Tumor	Remarks	Reference
Hollander	9 mos.	Sarcoma of uterus and vagina	Total extirpation; discharged from hospital in 1 month, cured	Deut. med. Woch., 1896, xxii, 16
Curtis	12 mos.	Sarcoma of cervix and vaginal vault	Operation; death following day	Trans. Obstet. Soc., London, 1904, xlv, 320
Findley	18 mos. first noticed	Primary, small spindle-cell sarcoma of cervix	First noticed on anterior lip of cervix; removed; in 6 weeks return; in 3 months another operation; child died in fourth year of life; whole pelvis filled with mass	Surg., Gynec. and Obstet., Chicago, 1906, iii, No. 4, p. 501
Rosenstein	2 yrs.	Carcinosarcoma	Died	Arch. f. path. Anat., Berlin, 1883, xcii, 191
Steffen	From 5 mos. to 2 yrs.	Eight cases; 3 sarcoma of uterus; 2 carcinoma of uterus; 3 sarcoma of uterus and vagina	All the sarcomas were considered to be congenital; Steffen says that benign congenital tumors are known to become malignant after attempt at radical removal	Die Malignen Geschwülste in Kindesalter, 1905
Virchow's Arch.	Less than 2 yrs.	Primary sarcoma of fundus uteri; extensive in bladder	Death from cachexia	Virchow's Arch., Bd. xcii, 1
Pick, L	2 yrs.	Primary carcinoma of cervix	Polyp in vagina; secondary large growth in abdomen; death due to chronic peritonitis	Arch. f. Gyn., Bd. xlvii, 1
Laidley	2 yrs. and 6 mos.	Carcinoma; post-mortem and microscopic diagnosis	Death	St. Louis Courier of Med., Sajou's Annual, 1891, F. 34, vol. ii
Smith, F. C.	3 yrs.	Small round-cell myxosarcoma of uterus	Subperitoneal uterine growths; vaginal polyp; death in 33 days after removal of vaginal growth	Amer. Jour. Obstet., 1883, xvi, 555-6
Ahlfeld	3 yrs. and 6 mos.	Sarcoma probably primary in vagina; a large tumor was attached to the fundus uteri and occupied the entire cavity of the uterus	Posterior wall of bladder involved	Arch. f. Gyn., Band xiv, 1
Depage	3 yrs. and 6 mos.	Primary sarcoma of uterus; microscopic diagnosis	First operation for cervical fibroid; growth recurred in 6 months; vaginal hysterectomy; in 6 weeks entire pelvis involved; death	Central. f. Kinderheilk., 1902, Bd. vii, S. 103
Markovic	5 yrs.	Large sarcoma of posterior wall of uterus	Death	Liecnieki, 1901, xxiii, ii, 426
Gynghofner	8 yrs.	Anterior lip of cervix growth size of a hazel nut; medullary carcinoma	Almost continuous hemorrhages from genital tract for 2 or 3 years; died	Ztsch. f. Heilk. Prag., 1888, Band ix, 4-5 S. 337, 1 pt
Homans, J.	10 yrs.	Cancer of abdominal organs	Recovered from operation; died 9 months later	Laparotomies for Various Diseases, Sawyer & Sons, Boston, 1887
Bluhm	11 yrs.	Sarcoma; cervical polyp		Arch. f. Gyn., Band lxviii, Heft 62
Agnus Bluhin	11 yrs.	Sarcoma botryoides mucosacervicis	Operation; recovery	Med. Woch., iv, 21, No. 175, Mai 25
Zweifel	13 yrs.	Round-cell sa coma of uterus	Death	Cent. f. Gyn., 1884, Band viii, S. 401
Von Konig	13 yrs.	Krebsbildung	Died	Allg. Wien. Med. Ztg., 1885, xxx, 280
Wells	13 yrs.	Cancer of body of uterus; extension to bladder	Menses suppressed for 8 months, then uterine hemorrhage which persisted until death	Findley, Surg., Gyn., and Obstet., Chicago, 1906, iii, 502

Reporter	Age	Tumor	Remarks	Reference
Little	14 yrs.	Carcinoma of cervix uteri		New Orleans Med. and Surg. Jour., 1896–7, xlix, 322
Jaeger	15 yrs.	Fibrosarcoma of uterus		Central States Med. Monit., Indianap., 1906, ix, 754
Bractz	18 yrs.	Tumor of posterior lip of cervix, 2 cm. in diameter	Kaltenbach removed the uterus by vagina; girl died in 1 weeks; no autopsy; growth was thought to be an endothelioma	Arch. f. Gynäk, Bd. ii, 1896
Coppée	18 yrs.	Encephaloid cancer of rapid growth	Death	Bull. soc. de méd. Gand., 1864, xxxi, 35–39
Tschop	19 yrs.	Carcinoma; microscopic studies	Vaginal hysterectomy; full recovery; vagina and adnexa not infiltrated	Central. f. Gynäk., 1897, No. 2
Pick, L.	Children	Sarcoma of uterus and vagina		Ueber Sarcome des Uterus und der Vagina in Kindersalter und des Primäre Scheidensarcoma des Erwachsenen; Arch. f. Gynk., Berlin, 1894, xlvi., 191–255
Odebrecht	Young, age not stated	Sarcomatous degeneration of a uterine myoma	Recovered	Zent. f. Gynäk., 1904, No. 2
Chase	Young, age not stated	Sarcoma of uterus and ovaries	Mixed toxin treatment	Brooklyn Med. Jour., 1896, x, 442
Marsh	Child	Sarcoma of uterus	Vagina and rectum involved	Findley; Surg., Gyn. and Obstet., Chicago, 1906, iii, 502

indeed, the growth may be so slow that their presence may not be manifested until adult life is reached. In the meantime the ovary will functionate normally. Ovarian cysts, on the other hand, are brought to us while the child is still quite young; it may be as young as three months. Malignant disease of the organs develops very rapidly in the child, and is likely to be accompanied by ascitic fluid; but my experience is that the general health does not become involved as early as we would suppose. Most writers say that it is characterized by early marked affection of the general health. This is certainly not so in my cases. The children with malignant abdominal growth were brought to me because their parents recognized the abdominal enlargement, and not because the child's symptoms were exacting, although in two instances the growths were very large. Of course, later the symptoms are those common to all malignant growths, whether in child or adult.

Sarcoma is the most frequent growth found in the child's ovary. Most of the congenital growths are sarcomas, and they have a strong histological resemblance to embryonic ovarian stroma. Most of those in infancy and childhood are made up of small round cells. The other varieties described in children are: Spindle-cell sarcoma, lymphangioma, endothelioma, and fibrosarcoma.

Emanuel's[29] case of tumor of the left ovary, in a fifteen-year-old

[29] Zeit. f. Geb. u. Gyn., Band xxv.

girl, contained tissue elements of endo-, ecto-, and mesoderm, but its stroma was sarcomatous. The child made an excellent operative recovery, but died four months later from recurrence.

The prognosis of malignant growths of the ovary at the present time is rather appalling. This is due, however, to the fact that the cases are not submitted early enough to radical removal of the incipient growth. The age of the child has little to do with it; very young children have recovered from abdominal operations of all sorts. I agree with Baginsky that the prognosis becomes better day by day as the technique of operations in this region progresses; as a writer has said, the age of the patient does not, as one would suppose, contribute any valid reason for refusing to operate; and, as Kelly says, on account of the large percentage of malignant tumors in children and the consequent dangers of delay, the operation should be performed as soon as the patient can be suitably prepared for it. Henning recognized twenty-seven years ago the advantage of early operation in children.

If the tumor is a dermoid or cyst the prognosis is extremely good, even at the most early age of infancy; but if it is sarcoma or carcinoma the operative prognosis at the present time is appalling and the chances of non-recurrence are remote. Thus, of Sutton's 21 cases of ovarian sarcoma, 11 died within a year after their operations, a mortality of 52.4 per cent. The mortality in cysts will be about 8 per cent., in dermoids, between 13 and 21 per cent.; and in solid tumors (malignant), between 33 and 52 per cent. The literature, however, contains a number of examples in which the children survived for years and were well when last heard from. Bode's[30] case of a girl, aged thirteen years, with large round-celled sarcoma of the right ovary, growing under the sheath of the abdominal muscles, was in perfect health four years after its removal. Gage[31] removed a two and one-half pound, round-cell sarcoma from a fifteen-year-old girl, who five years afterward was well and married. Kelly's patient with ovarian sarcoma, aged twelve years, lived two years after operation and died from dysentery, without return of growth. Harris's case of twenty-two months of age recurred, as did Byford's, aged four years and eight months, Page's,[32] a six-year-old girl, Croom's,[33] a seven-year-old girl, Forbes'[34] at nine years, McBurney's[35] at ten years, and Wagner's[36] at ten years. The record in carcinoma in children is most unsatisfactory, with here and there a gleam of hope. Redner's case, a nine-year-old child, died one year later from recurrence. She made a good operative recovery. One of this operator's cases in a child was well four years after the removal of the ovary; another of the children died in three months from

[30] Central. f. Gyn., Band xviii, S. 1171.
[31] Jour. Amer. Med. Assoc., December, 1894.
[32] Lancet, December, 1895.
[33] Edin. Med. and Surg. Jour., 1893, p. 689.
[34] Australian Med. Jour., 1894, xvi.
[35] Annals of Surgery, 1895, xxi, 706.
[36] Arch. f. klin. Chir., xxx, 704.

recurrence. Homan's[37] case, aged ten years, of cancer of the abdominal organ recovered nicely from the operation, but died nine months afterward from recurrence. Michel lost a case in a sixteen-year-old girl two years after operation from recurrence in the liver and kidneys. Weil's case of adenocarcinoma of the ovary at five years of age was well at the last report, March, 1905.

In true carcinoma of the uterus in children our therapeutic measures are as yet of little avail. Rosenstein's[38] case, aged two years, classed as a carcinosarcoma of the uterus, died fourteen days after operation. Ganghofner's girl, aged eight years, with a uterus 2.6 cm. long, containing a cancerous tumor the size of a crown piece, died from an intercurrent variola. Blum's[39] case, aged eleven years, with sarcoma of the cervix, died from recurrence. Agnus Bluhin's[40] case of sarcoma botryoides cervicis in an eleven-year-old girl recovered. Bradford's[41] case, a girl aged nine years with papillary cystoma, the size of the head of a full-term fœtus, was well three months after a complete hysterectomy. Odebrecht's[42] case in a young girl (age not stated), with sarcomatous degeneration of a uterine myoma recovered after operation. Worrall's[43] case of adenoma of the uterus and double ovarian tumor recovered completely. I do not know the termination of Little's[44] case of carcinoma of the cervix uteri in a fourteen-year-old girl, but imagine it too was fatal.

Of the greatest interest are the cases of chorio-epithelioma in children, the so-called deciduoma metastica malignum of Sänger. Schlangenhofer, of Vienna, in 1902, reported one in a virgin, aged thirteen years, who had never menstruated; Ahlfeld[45] one at seventeen years of age; and Champneys[46] one at eighteen years; while Brock's[47] case was a mole passed by a girl twelve and one-half years old. It has been aptly said that a mother fatally invaded by the trophoblast of her own child would be a person of one generation killed by a tumor belonging to a person of the next generation—a matricide; but these little girls slain by an embryoma in their own uterus are the victims of a potential brother or sister, not of their own child. It is now a fratricide.

For purposes of illustration I will detail the notes of but one case of malignant disease of the ovary.

A. B., aged eleven years, was brought to me on May 20 by her father, because he noticed a reluctance on the child's part to go through the "setting up" exercises which it was the custom of the

[37] Three Hundred and Eighty-four Laparotomies for Various Diseases, 1887.
[38] Arch. f. path. Anat., 1883, xcii, 191.
[39] Arch. f. Gyn., Band lxviii, Heft 62.
[40] See table.
[41] Arch. Pediat., 1892, ix, 508.
[42] Zent. f. Gynäk., 1904, No. 2.
[43] Australasian Med. Gaz., Sydney, 1893, xii, 308.
[44] New Orleans Med. and Surg. Jour., 1896–97, p. 322.
[45] Monatsschr. f. Geb. u. Gyn., 1895, i.
[46] London Pract., January, 1896.
[47] Stone, Chorio-epithelioma, Amer. Jour. Obst., October, 1907.

other children to do every morning. This child said it gave her an uncomfortable sensation to stoop forward and touch the floor with her hands. This was the only symptom that she complained of. There is absolutely nothing of interest in the previous history of the child or in the family history. The mother thought the child's waist measure was increasing. Examination at this time showed a large tumor occupying the right iliac, lumbar, and hypogastric region to the line of the umbilicus. The tumor mass was rather movable. It was readily outlined and was apparently extrapelvic; that is, it did not seem to have a pelvic origin; its physical signs were rather those of kidney enlargement, but the urine was absolutely normal and remained so. The inguinal, axillary, and cervical glands were not enlarged. The mammæ were normal and the pubic hair was not developed. The edge of the tumor was easily outlined.

In a few days (May 24) the temperature became slightly elevated and irregular, ranging between 99.3° and 100° F. This continued up to the time of operation, the temperature occasionally but rarely below the normal. This fall might occur in the morning or the evening; its time of elevation was irregular also. The highest temperature recorded from the first observation, May 20, to the date of operation, June 18, was 101.2°, which occurred on May 27, and seemed to be due to an intestinal toxemia. The lowest temperature was 98.2° F. During the pre-operative period of observation, extending over twenty-nine days, the child not only did not lose weight, but gained one pound between May 24 and June 7.

Operation: Right salpingo-oöphorectomy, with the coöperation of Drs. Lasher, Dunbar, Magee, and Parsons; ether was used and 65 grams was administered. The operation consumed thirty-eight minutes and the anesthesia forty-five minutes. Median incision. The growth was found to be a large sarcoma of the right ovary. A portion of it was wedged tightly between the uterus and the bladder, the adhesions being very intimate, but easily broken up. There was some free hemorrhage. The major portion of the growth was above the brim of the pelvis and occupied about the site of an enlarged kidney; its contour was not unlike that organ. The portion that extended out of the pelvis was pedunculated. The uterus was not enlarged. There was no evidence of metastasis at the time of operation. The child was shocked somewhat during the removal of the growth, but readily responded to the usual methods of combating such a condition.

The child had an absolutely uneventful convalescence, suffered little pain, and speedily arose from bed. Within a month a return of the growth was noted in the abdomen, and in six weeks the child succumbed to what seemed to be the most extensive involvement of the organs in the abdomen that I have ever seen. At the post-mortem, metastasis was seen to be universal; no structure seemed to have escaped its invasion.

Primary tumors in the vagina are rare at all ages; they are usually

fibroma, fibromyoma, myoma, sarcoma, or carcinoma. All except the last two are very rare in children, and these even are of very infrequent occurrence. Sarcoma of the vagina, however, is more frequent in children than in adults; in one of the series in the literature, that of Starfinger, of 26 cases, 24 were in children under five years of age; indeed, 10 were under one year of age. Malignant disease of the vulva and vagina constitutes but 1 per cent. of all malignant disease of the genital tract at all ages. The growths may apparently be congenital or appear early in life, before the fifth year, many, in fact, before the first year. They may develop from the walls of Gärtner's canals or from an obliterated Müller's duct. Primary sarcoma of the vagina is the most frequent malignant growth in childhood and is a specialized form of malignant disease which may affect any or all of the connective tissues which are involved in the complicated developmental processes associated with the formation of cloaca. Sarcoma of the vagina in childhood is usually microscopically the characteristic structure of racemose sarcomas. Striated muscle fibers are sometimes seen in the primary tumor, the secondary growths, and in the metastases, fibers which Piquand[48] says resemble the striated muscle found in a three-months' fœtus. The sarcoma usually grows in the connective tissue of the pelvic organs, and may extend in all directions, into the uterus, vagina, bladder, and urethra. In some cases it may remain circumscribed in the vagina; its usual clinical form, however, is that of a polyp, either vaginal or uterine, often both.

In children vaginal sarcoma shows very distinctly this tendency to become pedunculated, polypoid, and multiple. It pursues a comparatively slow growth and ulcerates very slowly, if at all; the lymphatic glands are affected, if at all, late in the case, and in consequence it dissiminates very slowly. The growth may be so slow that Herndon,[49] who has written a most valuable article on the pathology of the reproductive organs, thinks that the tumors that were not observed until the sixth and fifteenth years, respectively, are probably congenital, but do not show active growth until this later period.

There is usually little trouble in making the diagnosis, but unfortunately the general practitioner is apt to look upon the polyps as benign growths, and the diagnosis is then not made until it is too late to remove the entire growth. The prognosis at best is grave, but recently some happy results have been reported, cures lasting for several years (see Table III), as much as ten in one instance, without recurrence, the children then passing from observation. Unfortunately, however, recurrence and speedy recurrence is still the rule, and the children die early from malignant toxemia or from pressure on the bladder and rectum and absorption toxemia.

[48] Sarcoma of the Uterus, Rev. de gyn., 1905, ix, 579.

[49] Kelly and Noble, Gynecological and Abdominal Surgery, i, 98.

TABLE III.—Malignant Disease of the Vagina in Children.

Reporter	Age	Tumor	Remarks	Reference
Heckford	2 mos.	Vaginal and vulval villous tumor, "medullary"	.....	Trans. Obstet. Soc., 1868, x, 224
Hauser	6 mos.	Vaginal tumors; multiple; anterior vaginal wall	Round and spindle-cells and striped muscle fibres; operation; recurrence at short intervals; death	Virchow's Archiv., Bd. lxxxviii, S. 168
Rabe	6 mos.	Tumor size of small nut at site of vestibule; involved urinary meatus; sarcoma	Removal of uterus and vagina by laparotomy; death from bronchopneumonia	Surgeon General's Catalogue, Holmes, Pediatrics, 1907, xix, No. 2, 99
Frick	7 mos.	Right anterior vaginal wall; round and spindle-cell sarcoma	Operation; rapid recurrence and death	Surgeon General's Library, Holmes, Ibid
Schuchardt	7 mos.	Vaginal tumor	Mixed-cell sarcoma; operated on by Volkman; at report cure had lasted 10 years	Verhandl. der Deut. Gesellsch. f. Gynäk. 1868, Bd., ii, S. 239
Hollander	9 mos.	Vaginal polyp; large	Posterior wall of uterus also involved; spindle and round-cell sarcoma; operative cure by Israel	Deut. med, Woch., Vereins Beilage, 1876, xxii
Johannersen, A.	11 mos.	Sarcoma	Whole pelvis involved	Jahrb. f. Kinderh., 1897, N. F. xliv, 114–122
Kolisko	12 mos.	Left vaginal wall, number of large and small polyps; round and spindle-cell sarcoma	Repeated operation, with rapid recurrence after each; death 6 months after first noticed from pyometritis and suppurative peritonitis	Wien. klin. Woch., 1889, ii, 109, 130, 159, 182, 202, 222; Kolisko abstracts twelve other cases from the literature and thinks that early and radical extirpation of primary tumor justifies hope for successful issue
Weinlechner	12 mos.	Vaginal polyps: sarcoma	.....	Wien. klin. Woch., 1889, S. 109
Holmes, O. L.	17 mos.	Round-cell sarcoma; anterior vaginal wall	Three incomplete operations; death due to uremia and septicemia	Trans. Med. Assoc., Georgia, Atlanta, 1906, 304-311; Pediatrics, N. Y., 1907, xix, 95-100
Starfinger	18 mos.	Large round and spindle-cell sarcoma posterior vaginal wall	Removal by curette; death in 3 weeks; duration of disease 6 months	Steinthal, Virchow's Archives, Bd. iii, S. 449
Thomas	18 mos.	Tumor of left labium majus; sarcoma	.....	Amer. Jour. Obstet., vii, 51
Strassman	18 mos.	Patly gangrenous tumor protruding from vagina; large round and spindle cell sarcoma	Hemorrhage and foul discharge from vagina for 4 months; curretted and tanponned with gauze; result not stated but suggests to remove vagina and uterus per sacrum	Library Surgeon-General's Office, Holmes, Ibid.
Billroth	18 mos.	Fibrosarcoma; warty growth in vagina and bladder	Vesicovaginal septum infiltrated	Wien. klin. Woch., 1889, No. 8, S. 159
Pick	24 mos.	Vaginal tumor; spindle-cell sarcoma	Infiltrating pelvic organs	Arch. f. Gynäk., Bd., 1894, xlvi, 192
Frick	24 mos.	Tumor originated on posterior vaginal wall near vaginal entrance; cylindrical; round and spindle cell sarcoma	Operation; recurrence; sacral operation; tumor same character; recovered and well 3 years after second operation	Holmes, Pediatrics, N. Y., 1907, xix, No. 2
Korner	24 mos.	Vaginal polyps; vesicovaginal septum infiltrated	Fibrosarcoma	Archiv. f. Gynäk., Bd., 1894, xlvi, S. 220, quoted by Pick, Ibid.
Marsh	24 mos.	Polyps in vagina and bladder; vesicovaginal septum infiltrated	Small round-cell sarcoma	Trans. Path. Soc., 1874, xxv, 178

Reporter	Age	Tumor	Remarks	Reference
Power	2 yrs. and 4 mos.	Tumor of right wall of vagina; round-cell sarcoma; large portion are fibro-sarcoma and myxo-sarcoma	Admitted to hospital for retention of urine caused by swelling of vagina said to have followed measles 14 months previously; 5 months before admittance polyps noticed in vagina; died of uremia	St. Bartholomew's Hospital reports, xxxi, 121 135; Trans. Path. Soc., xlvii
Schuchardt	2 yrs. and 6 mos.	Vaginal polyp; mixed-cell sarcoma; round and spindle-cells	Some pain and hemorrhage; tumor removed; recurred in 7 months; removed; no recurrence when case was reported	Verhandl. der Deut. Gesellsch f. Gynäk., Bd., 1888, ii. S. 239
Lee	2 yrs. and 6 mos.	Vaginal polyps; vaginal and urethral entrance involved; rectovaginal and vesicovaginal walls involved	Sarcoma; elastic tumor felt in abdomen reaching to umbilicus; died 13 months after first seen	Holmes, Ibid.
Kolisko	18 mos.	Myofibrosareoma; tumor protruded from vulva for one month before admittance to hospital	No symptoms for 2 weeks; vagina very long; removed tumor, but vagina soon filled again; death; autopsy; origin in anterior vaginal wall; grew to size of walnut in 6 weeks	Wien. klin. Woch., 1889, ii; child was admitted to Billroth's Clinic in 1875 and I think it is the same case as the one that immediately proceeds it in this table.
Kolisko	18 mos.	Polyps; vaginal sarcoma	Died 2 weeks after admission to hospital; autopsy; nodular tumor of vestibule infiltrating vagina, urethra base of bladder and cervix; cystitis, pyelitis, pyelonephritis and pyometra	Ibid.
Weinlechner	18 mos.	Vaginal polyps; myofibrosarcoma	Bladder, cervix and urethra involved	Wien. klin. Woch., 1889, S. 109
Wrede	21 mos.	Cauliflower growth in vagina; round and spindle-cell sarcoma	Removed by ligation; recovered in 2 weeks; repeated operation and recurrences; cystitis, continued fever; death	Holmes, Ibid.
Steinthal	24 mos.	Vaginal tumor; round-cell sarcoma; anterior vaginal wall	Myxosarcoma; duration of disease 12 months; tumor removed after 8 months' duration; recovered in 8 weeks; death in 6 months from uremia	Virchow's Archiv., Bd. iii, S. 449
Piechand, and Guyot	24 mos.	Vaginal tumor on posterior wall; sarcoma	Serious hemorrhage; inoperable on account of size; uterus not involved general suppurating peritonitis cause of death	Holmes, Ibid.
Soltman	2 yrs. and 6 mos.	Vaginal and bladder polyps	Round-cell sarcoma in vagina; spindle-cell in bladder	Jahrb. f. Kinderheilk., Bd. xvi., S. 418
Kelly, H. A.	2 yrs. and 6 mos.	Rhabdomyosarcoma	Death due directly to urinary obstruction	Gynecology and Abdominal Surgery Kelly and Noble, 1907, i, 820
Sathman	2 yrs. and 6 mos.	Anterior vaginal wall and polyps protruding from vagina; round-cell sarcoma	Posterior wall of bladder involved; painful urination; intermittent fever; hemorrhage	Hesterman, Holmes, Ibid.
March	2 yrs. and 7 mos.	Tumor on anterior vaginal wall; polyp	Metastasis on posterior wall of bladder; course of disease 6 months; operation; seven recurrences; death	Hesterman, Holmes, Ibid.

Reporter	Age	Tumor	Remarks	Reference
Marshall	2 yrs. and 7 mos.	Vagina	.....	Brit. Med. Jour., 1889, i, 27
Sänger	2 yrs. and 8 mos.	Polypoid masses in vagina; round-cell sarcoma	Bladder, broad ligaments, deep lumbar glands infiltrated; recurrence in 2 months; death; autopsy; ascites, suppurative peritonitis; large sarcoma; anterior vaginal wall; isolated polyps posterior wall perforating abdominal cavity; dilatation of bladder and ureters	Arch. f. Gynäk., Bd. xvi, S. 58
Brown	3 yrs.	Vaginal sarcoma	.....	Obstet. Soc. of Baltimore, Holmes, Ibid.
Smith, T. C.	3 yrs.	Vaginal and uterine polyps	Myxosarcoma; uterine subperitoneal growths	Amer. Jour. Obstet., 1883, xvi, 555-668; Ibid., 1893, xxvii, 577; when this case was first reported in 1883 it was erroneously considered to be the first case on record
Ahlfeld	3 yrs. and 6 mos.	Vaginal polyps	Fibrosarcoma filling pelvis; inguinal glands enlarged	Arch. f. Heilkunde, Bd., 1867, xvi, S. 135
Schustler	4 yrs.	Vaginal polyps; vesicovaginal septum involved	Myxosarcoma	Wien. klin. Woch., 1888, SS. 148, 225
Demme	5 yrs. and 6 mos.	Vaginal polyps	Fibrosarcoma	Pick (quotes in) Arch. f. Gynäk., xlvi, S. 218
Demme-Gränchier	6 yrs.	Sarcoma	At birth a pea-sized nodule; active malignant growth at 6 years	Gränchier Inaug. Dis. München, 1888
Power, D. A.	Child	Primary sarcoma vagina	.....	Brit. Med. Jour., 1895. ii, 973
Power, D. A.	Children	Primary sarcoma of vagina	.....	St. Barth. Hosp. Report, London, 1395, xxxi, 121, 135
Rollin, M.	Infant	Sarcoma of vagina	.....	Rev. de gynec. et de chir. abd., Paris, 1906, x, 3–20
Peyrache, Jean	Little girl	Malignant tumor of vagina and vulva	.....	Des tumeurs maligin de la vulve et du vagin ches la petite fille. Paris, 1905, 101, p. 80
Ward, G. G.	Under 20	Primary epithelioma of the vagina	This seems to be the youngest case on record of primary epithelioma	Contrib. Soc. Med, and Surg. Twenty-fifth Anniv. Founding of N. Y. Postgrad. Med. School and Hosp., N. Y., 1908, 159–166, 1 pt.

As in malignant disease of the ovary, so in similar disease of the vagina, as a rule, the first thing noticed is the tumor; the child's general health is not affected until very late in the disease. The tumor appears between the labia; whereupon the child may complain of some pain, and the evidence of obstruction to urination and defecation will soon become more marked. Another anology is that the nutrition of the child will not be impaired until the disease has progressed to an almost fatal stage; of course, those cases in which the growth early becomes necrotic from pressure will early show the symptoms of septic absorption.

They are said to grow usually from the anterior vaginal wall, but an analysis of the reported cases does not entirely bear out this statement. Carcinoma of the vagina in childhood is still extremely rare. I have elsewhere called attention to Winckel's report of Smith's case of cancer of the rectovaginal wall in a child aged fourteen months; that of Guersant, who saw a carcinoma 20 cm. long and 28 cm. in circumference growing from the introitus of the vagina in a child three and one-half years old. Johannovsky, in the pathological collection at Strasburg, discovered a carcinoma the size of a hen's egg situated in the vaginal vault in a preparation from a girl aged nine years. In the light of present-day knowledge the probability is that these cases classed as carcinoma were sarcoma. Curtis[50] says that there is a case on record of cancer of the vagina in a girl, aged nine years, but he fails to give the reference. Mann has reported two cases between fifteen and twenty years.

Primary epithelioma of the vagina is a disease almost entirely confined to adult life; it is very rare at all ages, and almost unique in childhood. To show the rarity of primary vaginal involvement by cancer it is customary to quote the statistics of W. Roger Williams, who found that of 9226 tumors in the genitalia of women, but 54 were in the vagina. Other statistics bear this out: Reiche, of Hamburg, in 7498 deaths from cancer, found 29 vaginal; Hecht, in 4507 cases, found 50 vaginal. In view of these figures the case of primary epithelioma of the vagina in a patient under twenty years of age, reported by George Gray Ward,[51] becomes of great interest, and will undoubtedly be the cause of further cases being recognized at even earlier periods of life. The girl had never sustained an injury, nor had she been sick at all previous to the discovery of the vaginal growth, which microscopic examination showed to be an infiltrating epithelioma. The growth occurred in the posterior vaginal wall and was inoperable when first seen. It was treated with radium, without result.

The treatment of these forms of malignant vaginal growth is, of course, radically surgical. It must be fully understood that

[50] International Encyclopedia of Surgery, Ashhurst, vii, 293.
[51] Post-Graduate, New York, 1908, vol. xxv.

simple excision of the vaginal polyp, the usual early form of vaginal sarcoma in children, or of the more diffuse tumor growth, will inevitably be followed by recurrence sooner or later, usually soon. After thorough and complete removal a number of children have remained free from the growth for many years; indeed, until they have passed from observation. This is true in Schuchardt's report of a cure lasting ten years; and of Hollander's, after extirpation of the entire genital tract of a child aged two years. Power's patient was free from return for three years, and others for a shorter time. All the cases of carcinoma were fatal.

A CASE OF VIPERINE SNAKE BITE (OF UNDETERMINED KIND) TREATED WITH CALMETTE'S SÉRUM ANTIVÉNIMEUX (OR ANTIVENIN).

By W. F. Arnold, M.D.,
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On October 16, 1908, Mrs. McG., aged twenty years, a native of Georgia, but for twelve years past a resident of Florida, was bitten on the flexor aspect of her right forearm at the junction of its upper and middle thirds, while plucking jessamine blossoms from a tree-like shrub, her arms being extended and raised so that her hands were as high as her face. She felt the slight blow upon her bare arm, but she was unable to perceive the snake, in spite of somewhat careful search for it. Being accustomed to be circumspect about dangerous reptiles almost all her life, she had examined the ground well before approaching the jessamine tree; but she had been less careful in her inspection of the tree itself, because she believed that poisonous snakes were unlikely to be found in trees.

If the snake were a moccasin (Ankistrodon piscivorus)—as seems to be most probable—almost anyone in the least off-guard would have failed to have seen it, just as she did; for its white buccal surfaces would have been turned straight toward her, and they would readily have been taken for jessamine blossoms. Some of the symptoms suggest also that this was the species of snake concerned; for example, the pain was accompanied throughout with numbness; there was no ecchymosis, but puffiness about the fang-punctures appeared immediately; and swelling, involving the upper two-thirds of the affected forearm and extending almost entirely around it, had developed within one-half hour when I saw her.

I found this forearm was one inch (25 mm.) larger in circumference at its largest part than the other. (I ascertained afterward that in health both forearms were of the same size.) It was almost entirely anesthetic. Some part of each of these manifestations must

have been due to a ligature that had been applied snugly above the elbow of the bitten arm, but it was not tight enough to suppress the radial pulse, which was 80 per minute. Attention to the anesthesia was accentuated when her husband cut her skin slightly while removing the ligature about one-half hour after I had injected 12 c.c. of antivenin (Calmette's *sérum antivénimeux*) subcutaneously in three doses at points a short distance around the fang-marks; she did not know it until he told her.

These fang-wounds showed plainly, and each had bled a large drop or more, and bleeding continued slightly after turpentine—a good styptic—and kerosene had been applied. I explored each puncture with a big sewing-needle after having injected the antitoxin, as described above, and I found that the one on her forearm's outer aspect, which had been made by the snake's left fang, had penetrated the integuments of this region, and that the other had gone well into the derma, but in a direction downward toward the hand. No other marks were observed on the forearm. They were less than three-eighths of an inch (9 mm.) apart, which proves the small size of the snake and shows it to have been a viperine one.

I do not think that the other symptoms observed are noteworthy, especially as she had partaken liberally of whisky—about a pint, she thought. She was prepared to add corroboration to the current popular belief that victims of poisonous snakes cannot be intoxicated, but I prefer to reject this testimony along with all of her subjective symptoms.

With the understanding that it be received for what it may be worth, I shall add that she said that the pain rapidly diminished and soon disappeared altogether after the injection of serum. I left with her relatives the remainder of the *flaçon* to be used, if required by symptoms arising later; but it was not administered. The next day there was a patch of dense infiltration as large as a man's palm around the bite and the swelling had not abated notably. It disappeared altogether in a week. For three days its brawniness was notable. The fang wounds healed perfectly without scarring by the process of scabbing.

Owing to the evident small size of the snake concerned with this case, and the almost necessarily small dose of venom that it inserted, it is improper to claim here anything more than that antivenin appears to neutralize in human beings to some extent, as Calmette has claimed, viperine venoms. This fact is entitled to some consideration, I think, in view of the undoubted efficacy of antivenin in the treatment of the attendant bitten last summer in the Washington Zoölogical Gardens by a large Florida diamond-backed rattlesnake. I think also that its potency in removing symptoms of neurotoxic absorption in a case of rattlesnake bite that I have reported[1] deserves

[1] American Medicine, September, 1906, 310.

to be borne in mind, especially in connection with Prentiss Wilson's[2] academic deductions, that the antitoxin treatment of the bites of indigenous venomous serpents is impracticable.

It can, I think, be shown that the deaths from bites of venomous snakes in certain of our Southern States approach in numbers those caused there by lightning; and I feel sure that many of the former are quite readily preventable, if Calmette's, Noguchi's, and other work—now neglected—be applied as it has been applied in Japan, for instance, and that, too, in connection with a viper.

SOLITARY TUBERCULOSIS OF THE BREAST.

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Tuberculous disease of the breast is apparently very exceptionally met with in hospital practice. Indeed, until comparatively recent years there is scant mention of the condition in English literature, the bulk of the cases reported being those of French and German observers. With the one exception of Sir Astley Cooper, to whom is due the honor of having first, in 1829, in his "Illustrations of the Diseases of the Breast," described clearly under the term "Scrofulous Swellings" cases belonging to this category, there is no report in English literature of the observance of the condition prior to 1888;[1] this, too, in spite of exhaustive treatises upon the subject by Klotz[2] in 1879, by Richet[3] in 1880, Dubar[4] in 1881, Ohnacher[5] in 1882, Orthmann[6] in 1885, and many other extensive monographs and text references within the next five or six years by well-known Continental clinicians. And, even subsequent to the review of Shattock,[7] in 1889, the condition remained unrecognized, or, at all events, was not reported, in the large English clinics for some time.

Since the establishment by Dubar,[8] in 1881, of the diagnosis of this condition upon a histological and bacteriological basis, and its recognition as a clinical entity, there have been in all about forty cases reported, which conform to the term "Solitary Tuberculosis of the Breast." This term, suggested by Geissler[9] to cover those cases in

[2] Archives of Internal Medicine, June, 1908.
[1] Transactions of the Pathological Society, London, 1888, xxxix, 446.
[2] Arch f. klin. Chir., 1879, Band xxv. [3] Gaz. des hôp., 1880.
[4] Des tubercules de la mamelle, Thèse de Paris, 1881.
[5] Arch. f. klin. Chir., 1882, Band xxviii. [6] Virchow's Archiv, 1885, c, 365.
[7] Trans. Path. Soc., London, xl, 391.
[8] Loc. cit.
[9] Deut. med. Woch., 1906, xliv, 1780.

which the breast only was found to be diseased, excludes not only those giving a history or evidence of earlier tuberculous disease elsewhere, but also those in which the breast condition might be considered secondary to existing disease of the lungs, joints, etc., or as a direct extension from infected axillary lymph nodes.

While no case of primary tuberculosis of the breast has been confirmed by autopsy, solitary tuberculous infections of the genital tract do occur in women (such as the fifteen cases accepted by Veit[10]). It is, therefore, a reasonable presumption that such lesions may also be met with in the breast.

It has been a matter of no small difficulty to reach a satisfactory estimate of the number of reported cases of tuberculosis of the breast which conform to the term "primary" or "solitary." Only those cases have been included in this series in which careful search has failed to detect evidence of tuberculosis elsewhere. This is the only reasonable or possible ground upon which a classification can be based, in view of the total absence of autopsy records.

Pathogenesis. It is somewhat difficult to dogmatize upon the exact method of infection of the mammary gland, but if we adhere to the limitations of the term solitary mammary tuberculosis as defined above, we are restricted to the consideration of the three following as the most common: (1) Indirect—hematogenous; (2) direct—(*a*) through the lactiferous ducts and their lymphatics; and (*b*) through wounds, fissures, or abrasions (cutaneous lymphatics).

Predisposing Causes. *Age.* There is no reported case before puberty, while the number decreases rapidly after the menopause, the most advanced age recorded being fifty-three years, a case observed by Remy and Noel.[11] Thus the period of reproductive activity embraces the vast majority of these cases.

Lactation. In addition to the maturity of the gland, lactation would seem to be a predisposing factor, possibly the most important, as the incidence of the disease in married and unmarried subjects has been in the proportion of 4 to 1. No doubt the susceptibility to infection during gestation and lactation is analogous to that recognized in pulmonary disease, in which infection or recrudescence is, unhappily, a frequent sequel of childbed.

Inflammation. In those cases occurring in glands which have functionated, there is a history of inflammation complicating lactation in 42 per cent., and suppuration in 20 per cent.

Traumatism as a predisposing factor has rarely been mentioned, although noted by Poirier,[12] and Sabrazès and Binaud.[13]

Clinical Forms. Two clinical forms of the disease are generally recognized: (1) nodular—discrete—disseminated nodular; and (2) confluent.

[10] American Gynecology, September, 1903.
[11] Bull. de la soc. anat. de Paris, 1893, p. 412.
[12] Contrib. à l'etude des tumeurs du sein chez l'homme, Thèse de Paris, 1883.
[13] Arch. gén. de méd., 1896; Arch. de méd. expér., 1894, p. 838.

In the *nodular form* of the disease, one or more nodules are found, generally deeply embedded in the parenchyma of the gland. The disease tends to run a protracted course, the nodules frequently remaining the same size for years; eventually they gradually increase in size and proceed through the stages of degeneration, suppuration, and fistula formation. In a case reported by Scudder,[14] the condition ran a course of five years, and in a case of Mandry,[15] four years. Such a prolonged course is, however, exceptional, caseation and disintegration occurring, as a rule, within a year from the onset. Not infrequently, nodules coalesce, forming abscesses of considerable size. As might be expected in the nodular form of the disease, running as it does a chronic course, the onset is extremely insidious. Pain is generally absent in the earlier stages, and the breast may remain for some time unchanged in appearance. When foci occupy the central zone and the walls of one or more ducts are involved in the connective tissue changes, retraction of the nipple gradually develops.

In the *confluent form,* the onset is more acute; there is greater enlargement of the breast, pain is an early symptom, and degenerative changes, with fistula formation, develop early. It is this form of the disease which is most frequently met with during lactation; in the majority of cases it is centralized beneath the ampulla, suggesting an infection through the lactiferous ducts.

In all cases the breast is freely movable upon the pectoral muscle, and fixation of the skin over the foci develops only with their degeneration. Rarely more than one gland is involved, even when the disease is extensive and fistulas have existed for some time.

In both forms of the disease the axillary glands in the majority of cases are involved to a greater or less extent, the process being either tuberculous (75 per cent.) or simple hyperplastic adenitis. Where the primary lesion has existed as a solitary nodule in the upper and outer quadrant, running a chronic course, the secondary infection of the axillary lymph nodes may so outstrip the primary focus as to give rise to the belief that the glands themselves were the starting point of the disease. This fact has proved a point of difficulty in classifying the reported cases. While the direction of the lymph stream would suggest that the lymph glandular involvement, in the majority of cases, follows the breast infection, the frequency with which disease of the breast has been noted in the outer quadrants suggests the possibility of a retrograde infection, quite apart from those cases in which the process in the lymph nodes and breast can be shown to be contiguous.

Atypical Clinical Forms. Atypical clinical forms of solitary tuberculosis of the breast have been met with. Roux[16] describes a form

[14] Amer. Jour. Med. Sci., 1898, cxvi, 75. [15] Beit. z. klin. Chir., 1891, viii, 179.
[16] De la tuberculose mammaire, Thèse de Genève, 1891.

of intraglandular cold abscess, considered by Schley[17] as a terminal product, usually of the confluent type. It is of slow development and associated with diminution in the size of the breast. Orthmann[18] reports a case in which the disease apparently began as a subcutaneous infection, resembling an ordinary furuncle, with subsequent induration and extension of its base, resulting ultimately in the formation of a tumor of considerable size. Poirier describes a case in which two small cutaneous vesicles developed into indurated tuberculous masses. In the case of Kramer,[19] a primary lesion was noted, in the form of an ulcer, near the nipple, followed by the development of nodules within the breast, connected with the ulcer by bands of indurated tissue. In Demme's[20] case, tuberculous infection was ingrafted upon the margin of a pre-formed sinus, the result of a pyogenic mastitis. Finally, there is the case of Pluyette,[21] frequently cited, that of an unmarried woman, aged twenty-three years, in whom a swelling of the breast followed within a few days a slight wound of the nipple, and proceeded to suppuration. Tubercle bacilli were found in the nodule beneath the areola.

Retraction of the nipple has been noted as a primary symptom in three cases: in the case of Dubreuil,[22] retraction occurred two years before the observation of a tumor; in the case of Verneuil,[23] it was noted after confinement five years before the detection of an intraglandular mass. Warden[24] also reports a case in which a patient following her third gestation observed retraction of the nipple, whereas the glandular tumor only became manifest twenty-one months later.

It should be noted that the course of the disease in the confluent form is not infrequently hastened by the presence of a secondary (pyogenic) infection, and to this category belongs that group of cases running a rapid course and diagnosticated as simple pyogenic mastitis, which, after incision and drainage fail to heal, with the final development of widespread tuberculous infiltration and the persistence of multiple sinuses.

Pathological Anatomy. The pathological picture of tuberculous disease of the breast presents no striking differences from that of tuberculous processes elsewhere. While the degree and extent of the inflammation surrounding the nodule or nodules vary, there is always the classical picture of peripheral infiltration with lymphoid and plasma and more centrally epithelioid and giant cell formation, and a central zone of degeneration showing defective chromatic staining or actual caseation. In advanced nodular or acute confluent cases there is, of course, abscess formation, the pus sometimes

[17] Annals of Surgery, 1903, xxxvii, 510.
[18] Virchow's Archiv, 1885, c, 365.
[19] Cntrlbl. f. Chir., 1888, p. 867.
[20] Schmidt's Jahrbuch, 1891.
[21] Gaz. heb. de méd. et de chir., 1900, No. 103.
[22] Gaz. méd., Paris, 1888; Gaz. heb. des sci. méd., 1890.
[23] Prog. méd., 1882.
[24] Medical Record, 1898, liv, 479.

containing, as in my case, numerous polymorphonuclear leukocytes, in addition to tissue detritus. The walls of the abscess cavities usually contain numerous secondary tubercles varying in size from a millet seed to a pea, in various stages of growth and degeneration.

In all cases included in this series the diagnosis was confirmed either by histological or bacteriological examination or by animal inoculation. The case which I have to record is the first reported instance in which tubercle bacilli in pure culture have been grown primarily from the lesion in the breast.

Diagnosis. In view of the fact that the majority of cases of solitary tuberculosis of the breast have been noted in individuals of robust health, the diagnosis, especially of the discrete or nodular form, presents innumerable difficulties. When the tumor has been deep seated and retraction of the nipple present, without inflammatory signs in the axillary nodes, the clinical diagnosis of carcinoma has invariably been made. Happily, as the most approved local treatment (that is, wide and complete removal of diseased tissue), is the same in both conditions, no harm can have resulted; but, in view of the growing conservatism in the treatment of localized tuberculous infections, and the substitution of tuberculin therapy for radical operation in selected cases, it is possible that in the future one may consider operation inadvisable, especially in cases in which the possibility of lactation can be excluded. In such cases, the importance of differentiation is apparent, and may be achieved through the administration of sufficient tuberculin to produce a local reaction. When the latter occurs, one can be satisfied that the mass is not carcinomatous, although exceptions may be possible, the co-existence of carcinoma and tuberculosis having been reported by Pilliet and Piatot,[25] and Warthin.[26] In cases in which the gland is active, careful search should be made for tubercle bacilli, and animal inoculation should be carried out.

From acute pyogenic mastitis, tuberculous disease may, as a rule, be differentiated by its more chronic course, exceptional cases being those, as before stated, in which the two infections co-exist. From simple cysts tuberculous disease may be readily differentiated, the former being more regular in outline, elastic, painless, and without adenitis. Aspiration may be practised in confirmation. Fibromas and fibro-adenomas present greater difficulties. They are, however, free from inflammatory signs or evidence of lymphatic involvement. The pure fibromas, especially, are distinctly movable within the gland, and neither react to tuberculin, although, in a case reported by Davis,[27] tuberculous disease was found engrafted upon a pre-existing adenomatous tumor. In sarcoma the disease may

[25] Bull. soc. anat. de Paris, 1897, p. 424.

[26] Amer. Jour. Med. Sci., 1899, cxviii, 25.

[27] Medical News, 1897.

remain stationary and encapsulated for a prolonged period, followed by rapid growth unassociated with inflammatory symptoms, early involvement of the skin, and enlargement and engorgement of the cutaneous veins. Gummatous tumors are usually to be differentiated by the history, the presence of other lesions, or response to antiluetic treatment. In chronic mastitis, the process is diffuse. There is an absence of lymphatic infiltration and of a definite mass. Here, too, in doubtful cases the use of tuberculin may lead to a satisfactory differentiation. Actinomycosis of the breast is extremely rare, only five or six cases having been reported. In the earlier stages, differentiation from tuberculosis without local reaction from tuberculin is probably impossible; once sinuses are formed, however, the nature of the discharge in cases of actinomycosis at once gives a clue to the diagnosis.

TREATMENT. The treatment of this affection may be considered under the headings of (1) local, and (2) general.

Local. While it is conceivable that a partial extirpation might be the rational practice in cases in which a single nodule has developed in the periphery of the gland without lymphatic infection, it is the consensus of opinion that complete amputation of the breast should be carried out in all cases, together with the resection of the axillary glands when these are thought to be the seat of tuberculous disease. From a somewhat extensive experience in the treatment of localized tuberculous infections, with the aid of tuberculin as an adjuvant to general hygienic and dietetic measures, I am forced to the opinion, however, that under specific conditions one may be justified in staying operative interference until the effect of a course of tuberculin has been ascertained. Such cases may be considered favorable for this plan of treatment in which the disease is discrete, when chronicity is a prominent clinical feature, and when the possibility of lactation can be safely excluded. This latter point is of the utmost importance, as will be shown subsequently, but I think it may be here stated, without ground for contradiction, that a breast the seat of chronic tuberculous disease, altered anatomically by the development of scar tissue, and especially one in which there is retraction of the nipple, should not be allowed to functionate.

General. Though one may be reasonably sure that the breast infection is of the solitary type, there can be no certainty that infection of the viscera, joints, etc., may not subsequently develop, even in those subjects in whom total extirpation of the palpable disease has been practised. It is, therefore, of the greatest consequence that, not only in those cases in which operative interference is held in reserve, but also in those in which the local treatment has been radical, some plan of general treatment should be followed, as those operated upon may still remain susceptible to re-infection, and the non-operative cases to the development of metastases. To this end both classes of patients should be urged to effect a radical change in

the environment in which the disease was contracted. If an actual change of location is beyond the means of the individual, she should be relieved of all household work and the innumerable exactions upon time and energy which the direction of a household and its affairs entail. For a period of at least three months, absolute rest should be insisted upon. The greater part of each day and every night should be spent in the open air, the benign effects of fresh air being quite as important in the treatment of surgical infections as in pulmonary disease. One should not be satisfied with any proposed substitute. However liberal room ventilation may be, it should be recognized that "outside air" cannot be imported.

In addition to three meals of properly cooked and nutritious food —including rare meat and green vegetables at least once a day— milk and fresh eggs should be taken between breakfast and the mid-day meal, during the afternoon, and before retiring. If eggs are too costly, or are unobtainable owing to the season, milk alone may be taken, beginning with one quart a day in addition to that taken at mealtime.

Further, the patient should be instructed to keep an accurate record of the hours spent each day in the open air, the quantity and nature of food taken, together with the weekly gain in weight. These records should be scanned by the surgeon at each return of the patient for inoculation.

Finally, tuberculin in some form should be administered regularly. It is important that constitutional symptoms should not follow such inoculations, as, in the event of the development of a hypersensibility to tuberculin, its discontinuance might be necessary for some months. Inoculations should be repeated at intervals of ten days to two weeks, extending over a period of one year. If T. R. (tuberculin) be employed, the initial dose to an individual of average weight should not exceed $\frac{1}{1500}$ milligram. The value of increasing the dosage to more than $\frac{1}{1200}$ to $\frac{1}{1000}$ milligram is doubtful. Under no conditions should the increased dosage be permitted when injections are followed by constitutional symptoms, such as headache, malaise, or fever.

I think it is now generally accepted that in the treatment of localized tuberculous infections the use of tuberculin in some form, as an adjuvant to the measures above enumerated, yields results which are unobtainable when the exhibition of this agent is withheld. The bacillary product recently invented by Professor Vaughan, of Ann Arbor, containing, as it is thought, only the non-poisonous elements of cultures of the tubercle bacillus, has yielded excellent results in the treatment of localized infections, and its further exploitation may prove that we have in it an agent which may be administered in increasing amounts, without risking the serious danger of a prolonged hypersusceptibility or "negative phase."

Prophylaxis. I feel that a review of this subject would be lacking in force and deprived of one of its most important aspects if the question of the infectivity of the milk of tuberculous subjects were not briefly touched upon. Our knowledge of the comparative pathology of mammary tuberculosis conveys such a forceful lesson that even if we were without definite cognizance of cases of transmission of the disease from mother to child we should feel compelled in all cases of mastitis during lactation to consider seriously the possibility of the existence of a tuberculous infection. There are, however, cases described in which the evidence of direct transmission is most startling. The first in English literature, that observed and reported by Hebb in 1888, is a striking instance of the danger to the offspring, five out of eight children having died of tuberculosis. It should be stated, however, that this woman, in addition to mammary disease, had been treated for some years for pulmonary tuberculosis.

While bacteriological observations upon the secretion of glands known to be tuberculous have been scanty, women with tuberculous breasts have been seen nursing tuberculous infants.

Prognosis. Material on which to base an opinion as to the prognosis in solitary tuberculosis of the breast is, unfortunately, very scanty, owing, no doubt, to the difficulty experienced in inducing patients to return from time to time for examination after they have been discharged well. If an analogy to other forms of localized tuberculosis in otherwise healthy individuals may be invoked, the prognosis should certainly be favorable. We are, however, in possession of a record of after-examinations, chiefly upon patients treated surgically in the Tübingen clinic, and reported by Braendle[28] in June, 1906. This author's table embraces the after-histories of fifteen cases which may be accepted as primary infections. Of these 15, two died several years after operation from phthisis without evidence of local recurrence. Of the remaining 13, none had shown local recurrences or evidence of disease elsewhere, although several had been operated on four, eight, and eleven years previously; two patients remaining free from the disease sixteen and nineteen years after operation. Further, of these 13 living patients, 10 were either at the end of the fortieth or the beginning of the fiftieth year at the date of last examination. It is the opinion of Braendle that, in comparison with tuberculosis of other organs, primary tuberculosis of the breast may be considered a less unfavorable affection.

Case Report.—K. M., aged twenty-four years, single, dietitian. Complains of pain and swelling in the right breast.

The *family history* was negative.

Personal History. She had pneumonia at seventeen, with an uneventful recovery. In the spring of 1904 a nodule developed in

[28] Beit. z. klin. Chir., 1906, Band I.

the right breast at the site of the present swelling, which gradually increased in size, burst at the end of three months, and continued to discharge for a further period of four months. The condition was diagnosticated at the time as "cold abscess." Since 1904 the patient has remained well.

Present Illness. On March 20, 1908, the patient first noticed pain and the presence of a swelling below and to the outer side of the right nipple.

Present Condition (March 27, 1908). The patient is fairly well nourished and looks healthy. Mucous membranes are of good color. The left breast shows no enlargement or diminution compared with the right. Below and to the outer side of the right nipple, at the margin of the areola, there is a swelling the size of a pigeon's egg over which the skin is of a bluish gray color. The nipple shows a moderate grade of retraction. On palpation the whole breast appears to be firmer than normal, is freely movable upon the pectoral fascia, and is without evidence of localized disease except at and about the site of the aforementioned swelling, which is found to consist of a central portion, tense and fluctuating, surrounded by an area of infiltration adherent to the skin and shading off gradually into the surrounding gland tissue. Tenderness is not a marked feature, and there is no redness of the cuticle nor local increase of temperature. The nipple cannot be withdrawn. In the right axilla there is a soft palpable gland the size of a bean. Examination of the thoracic and abdominal organs is negative. There is no enlargement of the superficial lymphatic glands apart from that mentioned in the right axilla. Urine analysis is negative. Temperature is 98.2° at 4 P.M.

March 30. There has been a slight increase in the size of the fluctuating area since last seen. Under aseptic precautions the abscess was evacuated and 2 c.c. of viscid grayish pus withdrawn. Cultures on serum and hemoglobin-agar after twenty-four hours are negative. Smear preparations show numerous polymorphonuclear leukocytes, but no bacteria. A portion of pus was also inoculated subcutaneously into a guinea-pig.

March 31. At 4 P.M., tuberculo-ophthalmic inoculation (1 per cent.) was given.

April 1. At 9 A.M. no reaction was present.

April 4. Under ether anesthesia, amputation of the right breast was performed without opening the axilla. Convalescence was uneventful.

PATHOLOGICAL REPORT. *Macroscopically*, on cross-section the body of the gland looks like sarcomatous tissue, the cut surfaces having a glairy appearance, evidently due to a diffuse fibrosis. Below and to the outer side of the nipple the skin is adherent over a small abscess cavity which contains grayish mucoid pus. The walls of the cavity are irregular and villous in appearance, and of a

dirty gray brown color. Surrounding the abscess cavity is a dense area of acute inflammatory infiltration the size of an English walnut. Only one caseous area the size of a pea was found, the contents of which were transferred to slants of modified Dorset egg medium.

Microscopically, smear preparations show innumerable polymorphonuclear leukocytes; no bacteria. Stained sections from the wall of the abscess cavity show numerous recent tubercles with epithelioid and giant cells as well as central necrosis. Dr. Duval thinks it probable that the bacillus was of the bovine type, owing to the rapid course of the disease, the large number of polymorphonuclear leukocytes present in the pus, and the acute nature of the process histologically. The culture, however, was lost before titration observations could be carried out. The difficulty in maintaining the growth upon artificial media is an additional point in favor of a bovine infection.

After-treatment. Since operation, the patient has followed in a general way the line of treatment suggested in the body of this paper. Injections of T. R. (tuberculin), $\frac{1}{1500}$ milligram, have been administered every ten days. When last seen, in October, 1908, the patient was in excellent condition, and reported a gain in weight of eighteen pounds.

TUBERCULOUS PULMONARY CAVITIES IN INFANTS.

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The presentation of this paper on cavity formation in tuberculosis of the lungs in infants under two years of age has for its object, first, to record six cases; and second, to call attention to the frequency and location of pulmonary cavities at this age. In reviewing the literature the fact impresses itself that many writers on diseases of infants treat the subject in a very indefinite manner. Some do not refer to it, while others speak of it as if tuberculous cavities in infants followed the general course of events so frequently seen in the adult. This is somewhat surprising, because most of the authors who have written especially on this subject point out how difficult it is to make an antemortem diagnosis of a cavity in comparison to the condition in later life. We have had difficulty in selecting our cases from the literature, because many authors have failed to make the distinction between infancy and childhood. For our purpose, the subject may be divided into the following heads:

(1) The frequency of cavity formation in the lungs of infants; (2) the pathology of the condition; (3) the location of the cavities in the lungs; and (4) the report of six cases.

1. The Frequency of Cavity Formation in the Lungs of Infants. From the literature, we have been able to find special mention of cavity formation in quite a number of instances. These cases have been sifted from general discussions of the subject, rather than special reports of cases. We believe many cases have not been recorded because observers have not deemed it of sufficient importance to report them. Clinically, in the vast majority of cases cavity formation in the lungs of infants is entirely overlooked. We believe, therefore, that by calling attention to its frequency and localization, cavities should be more often recognized during life.

Leroux, in analyzing Parrot's cases, found cavity formation present in 57 cases out of 219 autopsies on patients under three years of age; that is 26 per cent. of the cases. He particularly mentions 5 of these cases which were under three months of age, but unfortunately no reference is made of the number under two years.

Warthin and Cowie quote Hugunin as reporting 2 cases, a seven-month premature infant and an infant seven weeks old; Berti, 1 case nine days old; Demme, 1 case eleven weeks old; Queyrat, 1 case three months old; and Flesch, 8 cases out of 500 autopsies on children in the early months of life. Demme reports 1 case, aged four weeks; F. Weber, 1 case, aged three months; Henock, 5 cases, aged four, seven, eight, ten, and eighteen months; Comby, 4 cases, aged four, six, eight, and nine months; and Deliarde, 1 case, aged six months, and speaks of the rareness of cavity at this age. Symes and Fisher, in reporting 500 deaths at various ages from tuberculosis, refer to two cases with cavities at five and eight months of age. Shennan reports 23 cases out of 355 cases (6.5 per cent.). Carmichael reports 2 cases, aged eight and fifteen months; Toulmin, 1 case, four months old; Donkin, 1 case, aged 1 year; Fry and Shaw, 1 case, twelve months old; Price-Jones, 3 infants, thirteen, sixteen, and twenty-two months old; J. Lewis Smith, 1 case, aged seventeen months; Green, 6 cases under two years; and Barthez and Sanne report 10 cases under two years.

Zuber says, "All authors agree that cavities are rarer in children than in adults." Baginski states: "Chronic tuberculosis with cavity formation in young children in characteristic form, as in the adult, is an infrequent disease." Other authors, as Rotch, Monti, Jacobi, Still, Osler, and Ashby, speak in general terms of the rarity of cavities in infants under two years compared with older children. The above mentioned cases, in all likelihood, do not represent the observations of these men as to the frequency of cavity formation, but simply some of the cases which they have had occasion to refer

to in reports. On the other hand, the following observers have found cavitation in infancy of more frequent occurrence: Northrup, in a personal communication, says: "Tuberculous cavities are not rare in infants as young as a few weeks even. They follow the law which would seem to apply to adults. The lesion is common." Rilliet and Barthez report cavities in the proportion of one to three, in cases from one to two and one-half years. Holt says, "Areas large enough to deserve the name cavities were present in 35 of 72 autopsies upon tuberculous patients, two years and under." De Rothchild states that pulmonary cavities are observed in one-third of the cases from one to two and one-half years. Hervieux reports that one-fifth of the cases in the course of the first year of life have pulmonary cavities. J. Francis Condie even went so far as to say, "Tuberculous cavities are much more frequent in very young children than in adults." Warthin and Cowie quote Weber as making the statement that he had many times observed tuberculous cavities of the size of half a lobe in children under the age of three months.

From the above resume of the literature, it will be seen that cavity formation in infancy has been observed by some observers in a high percentage of cases, while by others it has been found but rarely. From the comparatively few cases which we have been able to collect from the literature, the earliest case occurred in an infant premature at the seventh month, while the majority were found beyond the third month, with increasing frequency toward the end of infancy.

In order to study the frequency of tuberculous cavities in infants at the Children's Hospital of Philadelphia, the records for the past sixteen and three-quarters years have been reviewed. We have found that during this time 4518 infants have been admitted to the wards of the Hospital. Of this number, 1140 infants have died. The number of autopsies performed was 371, of which 75 disclosed tuberculosis. Of the total number of deaths, 6.5 per cent. were tuberculous. However, these figures are only approximate, because only one-third of the children dead were autopsied. Again, when we compare the number of tuberculous autopsies with the total number of admissions during this period, it will be found that 1.6 per cent. were tuberculous.

It will be seen from these statistics of the autopsy records that with us tuberculosis in infants under two years of age is an infrequent disease. This can partly be explained by the fact that the great number of infants coming to the hospital have some form of advanced gastro-intestinal disorder. If the actual number of cases of gastro-intestinal disease be studied from our autopsy reports, it will be seen that 155 of the 371 autopsies, or 41.7 per cent., were grouped under this head. Therefore, if these cases of advanced gastro-intestinal disease be omitted from our comparison, it will be seen that of the remaining autopsies (216) there were 75

cases, or 34.5 per cent. tuberculous. This latter comparison really emphasizes the high mortality of gastro-intestinal disorders (41.7 per cent.) far more than it does that of tuberculosis (34.7 per cent.) at this age. We believe, from the clinical aspect at least, that our former percentage of 1.6 per cent. of fatal cases is nearer the truth as far as tuberculosis at this age is concerned.

Below we have tabulated the ages of the tuberculous autopsies on infants:

First Year.	Cases.	*Second Year.*	Cases.
1 month old	0	12 months old	7
2 months old	1	13 " "	3
3 " "	2	14 " "	6
4 " "	1	15 " "	4
5 " "	5	16 " "	3
6 " "	9	17 " "	3
7 " "	5	18 " "	6
8 " "	3	19 " "	4
9 " "	2	20 " "	1
10 " "	5	21 " "	1
11 " "	2	22 " "	0
		23 " "	2
	35		40

Number of tuberculous autopsies on infants under one year, 35.
Number of tuberculous autopsies on infants between one and two years, 40.
Total number of tuberculous autopsies on infants under two years, 75.

To study the frequency of cavity formation in the lungs of infants dead of tuberculosis, we have gone over the records of the Children's Hospital, and found that cavity formations were present in 12 of the 75 subjects dead of tuberculosis (16 per cent.).

As we stated above, we encountered difficulty in trying to estimate this frequency from the literature. In only a few reports were actual statements as to percentages obtainable: Holt reported cavity formation in the proportion of 1 to 2; Rilliet and Barthez, and De Rothchild, in the proportion of 1 to 3 (these cases were one to two and one-half years old); Hervieux, 1 to 5 (during the first year of life). In our cases 1 in 6 showed excavations, which is a considerably lower percentage than the observations of the above authors. Nevertheless, it will be seen, even in our cases, that cavity formation at the autopsy table is not uncommon.

If we consider the frequency of this process in older children, as compared to our findings in infants, it will be seen that in the former cavitation is not only more frequent, but follows the rule applying to adults. The usual explanation of this condition is conceded to be the greater resistance offered as the child grows older, allowing the process to become of a more chronic nature, and thus giving mixed infections greater opportunity to act.

Table Showing the Ages of the Cases Under Two Years of Age with Tuberculous Cavities at the Children's Hospital.

5 months old	1
6 " "	4
7 " "	1
11 " "	1
12 " "	1
15 " "	2
17 " "	1
19 " "	1
Total	12

II. The Pathology of the Condition. In considering the pathology of tuberculosis at this age, a brief resume of the types of lesions may be of some value. It is not our purpose, however, to consider in detail these various lesions. Briefly the general pathology of tuberculosis at this age may be divided into three types: (*a*) Acute miliary tuberculosis; (*b*) chronic tuberculosis with fibrous changes; and (*c*) bronchocaseous pneumonic tuberculosis.

(*a*) *Acute miliary tuberculosis,* or acute disseminated miliary tuberculosis, of the lungs is considered rare by the majority of authors at this early age of life. When it does occur the miliary tubercles, either gray or yellowish gray in color, are found scattered throughout both lungs, and to a less extent in other organs of the body. These tubercles vary in size and number according to the degree of infection and the duration of the disease.

(*b*) *Chronic tuberculosis with fibrous changes* in the lungs can be very briefly considered here, because it scarcely enters into the discussion of our paper from the fact that many of the changes in the tissues are of a chronic nature. This type of tuberculosis at first is generally localized to a small area or areas, and the process is accompanied by the production of fibrous tissue. The process may consist of caseating areas containing small cavity formations, surrounded by more or less fibrous tissue. Distinct chronic tuberculosis of the lungs with cavity formation within the first two years of life, compared to the adult type of the disease, is rarely found at autopsy.

(*c*) *The caseous bronchopneumonic* type of tuberculosis is the most frequent form of the disease in the lungs at this age. It runs a more or less subacute course, and is almost always fatal. The advanced caseous lesions are usually confined to one lobe, or to one lung, with less marked lesions in the other lobes or lung. In a section of a lung, showing tuberculous lesions of this type, there are usually large caseous areas with intervening areas of bronchopneumonia, congested lung tissue, or normal lung tissue. The multiple lesions so frequently seen in these cases have their origin from some older process and are disseminated by way of the blood, lymphatics, or by insufflation. They increase in size from peripheral extension, and by the fusing of closely situated smaller areas. This method of

extension is especially to be seen when the lesions are near the opposing surfaces of the lobes and it is not uncommon to see the greater part of one lobe with the adjacent parts of another lobe, involved in the caseous process. The interlobar pleura in such cases is obliterated by the tuberculous process, or stands out prominently as a fibrous band stretched across the caseous area. It is in these large caseous areas that cavity formation most frequently begins at this age. Cavities are usually the result of acute softening of the central parts of the caseous mass, and when this softened area communicates with a bronchus the fluid parts are thrown off, leaving a space or cavity. The softening of the caseous mass is generally the result of secondary infection, and is usually due to Streptococcus pyogenes, Staphylococcus pyogenes aureus and albus, Micrococcus tetragenous, or Diplococcus pneumoniæ. A pure tuberculous process, as a rule, does not tend to liquefy, it being a dryer, harder, and more cheesy process. This in itself would explain the infrequent cavity formation in our group of cases.

In examining the lungs of this type of tuberculosis one frequently finds small softened areas in these caseous lesions. These have been recorded by authors as instances of cavities; they in all likelihood would produce cavities in the course of time. We have not included such areas as cavities in our cases, because as antemortem excavations they had not existed.

The walls of the cavities are usually irregular in outline and somewhat darker in color than the surrounding lung tissue; especially is this true if the cavity has existed for any length of time. A distinct communication with a bronchus may be found on some part of the cavity wall; it may, however, be small and difficult to locate. The size of the cavity varies usually with the chronicity of the case and the size of the caseous lesion. From the microscopic examination of these cases it would seem that the process is rapid, as the walls of the cavity consist almost entirely of caseous material. Fibrous tissue formation in the walls of the cavities does not take place to any great degree in this group of cases, compared to the formation of this tissue in the adult.

The fact that in early infancy the tuberculous process in the lung is generally of an extensive bronchocaseous type, running an acute or subacute course, explains to a certain extent the absence of fibrous tissue in the lesions. It is a fact that the younger the individual, the more extensive the involvement and the more acute the course. This statement is the result of autopsy examinations, and does not take into consideration the numerous infections with the tubercle bacillus which undoubtedly occur in early life, and are held in abeyance or cured. As many of these latter cases are not diagnosticated, the occurrence of the disease can only be surmised from the widespread prevalence of tuberculosis. It is only in the susceptible cases at this age that the process becomes diffuse and of an acute or sub-

acute course, ending fatally. It may also be noted here that in infants dying of tuberculosis, the lungs are involved in 100 per cent. of the cases.

III. The Location of the Cavity. The literature on the subject of the localization of the cavity is not very extensive. The majority of authors state that cavity formation usually takes place in the lower lobes, and generally at the root or central part of the lung. Thus, D. Francis Condie says: "There is an important modification that should guide the practitioner when he seeks to determine the existence of a cavern in young children, viz., that under five years of age, the cavernous excavation is generally seated in the lower or middle lobes, and is almost always confined to one side of the chest." James M. Coley found cavities in infants much more frequently in the inferior lobes, and most often on the right side. Zuber also found the right lung most commonly affected, and generally only one lung. Again, both J. Walter Carr and S. Vere Pearson have observed that in infants the usual location of cavities is at the root or central portion of the lobe. Berti and Demme each report cases with a cavity in the lower lobe of the right lung. Fry and Shaw report a case with a cavity in the lower lobe of the left lung. Price-Jones reported an infant with cavities in the same location; but report another case with a cavity in the upper lobe of the left lung. Hugunin reported an infant with a cavity in the apex of the left lung. Henock reported four cases:

An infant, four months old, right upper lobe, two cavities.
An infant, seven months old, right upper lobe, two cavities.
An infant, four months old, right upper lobe, two cavities.
An infant, eighteen months old, both lungs, numerous small cavities.

J. Lewis Smith reported one case at seventeen months, with cavities in both upper lobes. P. Bennis Green reported 6 cases as follows:

An infant, two years old, summit of right lung.
An infant, two years old, middle lobe of right lung.
An infant, two years old, upper lobe of left lung.
Three infants, two years old, lower lobe of left lung.

On the other hand, Eustace Smith says, "The masses situated nearest the apex are commonly the earliest to liquefy, but not always."

Barthez and Sanne, in 77 cases of cavity formation in infants, found:

Cavities within the right lung, 47.	
Right upper lobe	34
Right middle lobe	9
Right lower lobe	16
Cavities within the left lung, 51.	
Left upper lobe	51
Left lower lobe	18

The location of the cavities in the cases at the Children's Hospital may be tabulated as follows:

Left lung, 6.	
Left lung, upper lobe	2
Left lung, anomalous middle lobe	1
Left lung, lower lobe	2
Left lung, location not specified	1
Right lung, 6.	
Right lung, upper lobe	2
Right lung middle lobe	1
Right lung, lower l be	3

To summarize: Upper lobes, 4; middle lobes, 2; lower lobes, 5; location of cavity not specified, 1.

This small table shows the greater frequency of tuberculous cavities in the middle and lower lobes in infancy in our cases. If we consider the location of cavity formation in our own cases and of those collected from the literature, it will be seen that the lesion does not occur at the same location as it does in adults. In the majority of cases cavities in infants are deeply seated and usually at the root of the lung. In our cases cavities occurred more frequently in the lower and middle lobes than in the upper lobes.

IV. Report of Cases. Of the twelve autopsies performed on cases of tuberculous pulmonary cavities in infants at the Children's Hospital in the last sixteen and three-quarters years, 6 have come under our personal observation. It is these 6 cases we desire to report.

Case I.—Lillian R., aged six months, was admitted to the hospital on April 4, 1904. The family history was negative. The infant had been fed at different times on breast milk, condensed milk, and cow's milk. Although the patient had always been a delicate child, she had never had any serious illness. She was taken sick three weeks before admission to the hospital, with diarrhœa, weakness, and sweating at night. Four days before admission she developed a cough, coughing severely at times, and became extremely weak. The physical examination disclosed a bronchopneumonia in an emaciated, rachitic infant. The patient became weaker, the bowels continued loose, and the temperature irregular, until the child died, nine days after admission.

The *autopsy report*, abstracted, is as follows: Bronchocaseous pulmonary tuberculosis; tuberculous enteritis; tuberculosis of the liver and spleen. An emaciated colored infant with slight petechial eruption on abdomen. The left pleura shows a few tubercles on its parietal surface.

The left lung measures 11.5 x 6.5 x 3 cm., and weighs 70 grams. It is everywhere studded with grayish yellow tubercles, most of them small (millet seed), some few obtaining to larger size. Portions of the lower lobe are so thickly studded with tubercles as to sink

in water. The lung is mottled gray and dark red. The air-content is especially diminished in the lower lobe. There is hypostatic congestion in the lower lobe and the lower part of the upper lobe. The surface section is granular, moist, and glistening, of a mottled grayish color. Crepitation is diminished, while the consistence is firm and tenacious, with a bloodstained fluid exuding. The right lung weighs 108 grams. The air content is practically absent in the lower lobe, and a portion of all three lobes sinks in water. The lower lobe is consolidated, but is less thickly studded than the left lung with miliary tubercles. The lower part of the middle lobe also shows pneumonia. The upper part of the middle lobe and the upper lobe show much more thickly scattered tubercles. There is a cavity in the lower portion of the lower lobe posteriorly, containing caseous detritus. The bronchial lymph glands are everywhere enlarged. There is tuberculosis of the cervical, mediastinal, and retroperitoneal glands. Miliary tubercles in liver and spleen. Also a fatty liver and chronic follicular colitis.

Case II.—John T., aged fifteen months, colored, was admitted to the hospital on April 5, 1904. The family history was entirely negative. Baby was fed since birth on skimmed milk. Until four days before admission to the hospital he was apparently well. During these four days he had profuse sweating cough, fever, and evident pain on touching the anterior chest wall. Examination showed a diffuse bronchopneumonia. Infant died three days after admission.

The *autopsy* findings, in brief, were as follows: Caseous bronchopneumonia; miliary tuberculosis of the lungs, pleura, omentum, liver, spleen, and the hepatic, mesenteric, bronchial, and retropharyneal glands.

Body of an emaciated, colored infant. The left lung measures 11.5 x 7.5 x 3.5 cm., and weighs 71 grams. The color is mottled gray; the air content is diminished in the lower lobe, but in the upper it is normal. In the upper part of the middle lobe there is a cavity 2.5 cm. in diameter, surrounded by tuberculous caseous infiltration. The walls of the cavity are irregular and bordered here and there with tuberculous caseous masses. The contents of the cavity are purulent. The pleura directly over the above area is adherent with recent fibrinous adhesions to the chest wall; on breaking these adhesions the cavity was ruptured. Scattered through the lower lobe, and less numerous in the upper lobe, are many yellow tubercles. The right pleura shows a few pearly tubercles on the parietal layer. The right lung measures 10.5 x 8. x 2.5 cm., and weighs 49 grams. This lung is not affected except for a number of yellow tubercles.

Case III.—Moses C., aged six months, colored, was admitted to the hospital January 8, 1905. The father and mother are living and well; they have had two children, the patient and one other child which is dead. The cause of death given was teething(?). The

patient had been breast fed up to the time of admission. He has always been well until three months before coming to the hospital; during these three months the infant had a severe cough, but two days before admission cough became worse and great quantities of mucus were coughed up. The examination elicited the physical signs of a bronchopneumonia. The temperature was not hectic. In two days after admission the infant died.

The *autopsy* revealed: Caseous bronchopneumonia; general miliary tuberculosis; tuberculosis of the liver and spleen, and of the bronchial and mesenteric lymph glands.

The left lung weighs 145 grams. The entire lower lobe and the lower part of the upper lobe are consolidated. There is a large cavity 3 x 2 x 4 cm. in the upper third of the lower lobe posteriorly. It is superficial, and in one place a thickened pleura forms part of the wall. The bloodvessels and bronchi bridge across the cavity, and the walls are very irregular and dark in color. A small cavity, 0.5 x 0.5 x 0.5 cm., is also found on the lower surface of the lower lobe. There is an extensive obliterative diaphragmatic pleurisy at this location which brings the cavity close to the diaphragm. The whole lower lobe shows advanced caseous pneumonia, and also the greater part of the upper lobe, especially posteriorly. The air content is absent, except along the anterior border and at the apex. On both sides the bronchial glands are very much enlarged and caseous. Miliary tubercles in the liver, spleen, and intestines. The mesenteric glands are caseous.

Microscopic section of the lung close to the cavity wall shows diffuse cellular infiltration of tuberculopneumonic type. The type of cells present is lymphocytic, with a very few polymorphonuclear cells. There is congestion of the capillaries and giant-cell formation. The tissues in the immediate vicinity of the wall of the cavity show numerous large areas of typical caseation, but there is very little fibrous tissue formation. The large bronchial tubes in the neighborhood show marked bronchitis, the cellular exudate consisting of polymorphonuclear cells and lymphocytes. The walls of the bronchi show the infiltrating process.

Case IV. Robert N., aged eleven months, colored, was admitted to the hospital November 28, 1905. The family history was negative; the mother and father living and well. There was only one other child, and it was well. There was no history of tuberculosis in the family. The baby's birth was normal, and up to the time of admission it had been fed exclusively on breast milk. It had always been well, with the exception of an attack of gastro-enteritis when six months old. For several months before coming to the hospital the infant had had a cough, which in the last two weeks had become much more severe. On admission the infant presented the physical signs of a bronchopneumonia. Two days later it became much worse, cough very severe, especially at night. It did not take its

feedings well and the bowels became loose. It began to vomit, and on one occasion the vomited material was streaked with blood. The infant died twenty-two days after admission.

The *autopsy* report was as follows: Caseous bronchopneumonia; general tuberculosis; tuberculosis of the liver, spleen, mesenteric and bronchial glands; tuberculous enteritis and peritonitis; chronic fibrinous pleurisy.

The left lung weighs 62 grams. It is slightly congested, and shows a slight amount of œdema. This lung contains a few scattered gray miliary tubercles. The bronchial glands are slightly enlarged. The right lung weighs 205 grams. The whole lung is consolidated, it is firm in consistency and grayish yellow in color. The surface section is smooth, dry, and dull. There is a cavity at the upper part of the middle lobe, external to the mid-clavicular line. The cavity measures 2 x 1 x 3.5 cm. in size and extends upward into the upper lobe. The walls of the cavity are dark in color and covered with granulations. In the upper lobe there is an excess of fibrous tissue. Over the right lung there is a chronic adhesive pleurisy. There is tuberculosis of the intestines, peritoneum, spleen, liver, appendix, and mesenteric glands.

A microscopic examination of the section from the wall of the cavity shows fibrous tissue and a cellular exudate consisting of polymorphonuclear cells and small lymphocytes. The tissue in close proximity to the cavity wall consists of an overgrowth of fibrous tissue which extends into the alveolar walls. Embedded in this fibrous tissue there are collections of typical tubercles; they are for the most part small and show a small amount of caseation. Giant cells are very prominent. The surrounding lung tissue shows the capillaries congested. Widespread caseation of the caseopneumonic type is not present. The process is a chronic fibrocaseous tuberculosis, with the presence of many giant cells.

Case V.—Lillian P., aged one year, colored, was admitted to the hospital May 27, 1907. The parents were living and well. There had been no other children. The patient's birth was normal, and until the time of her admission she had been breast fed. She had never had pertussis, measles, or any contagious disease. She had had fever for one month before coming to the hospital. Baby had been vomiting sour milk immediately after feeding several times a day. The bowels were constipated. For three days before admission she had a number of general convulsions lasting from five to ten minutes. Examination revealed harsh breathing all over the lungs, with many large moist rales. She had slight rigidity of the neck muscles and a positive Kernig's sign. By lumbar puncture 45 c.c. of slightly cloudy fluid was withdrawn, in which tubercle bacilli were found. Child died forty-eight hours following admission, after having had a number of convulsions.

The *necropsy* follows: Caseous bronchopneumonia; tuberculous

enteritis; tuberculosis of liver and spleen; tuberculous meningitis. The left lung is 10 x 8 x 3.5 cm., and weighs 80 grams. The pleura is normal. The surface section of the lung shows numerous small gray translucent tubercles, without bronchopneumonia. The tubercles are isolated. There is slight hypostatic congestion in the dependent parts. The lung is crepitant and exudes bloody fluid on pressure. Bronchi and pulmonary vessels are normal. The bronchial glands are greatly enlarged. The right pleura is congested and thickened. There are firm adhesions from the right upper lobe to the anterior axillary line, and from the middle lobe to the fourth rib. There are adhesions posteriorly over the upper lobe. The right lung measures 13 x 9.5 x 3 cm., and weighs 95 grams. Generally, this lung is the same as the left, with the exception of the upper lobe. The upper lobe contains a cavity 1.5 x 1.5 x 2 cm., situated 1 cm. from the apex, and another cavity below this at the base of the lobe measuring 2.5 x 2 x 1 cm. Surrounding these cavities the lung tissue in this lobe is consolidated by a caseous pneumonia. On pressure mucopus exudes. The liver shows fatty change, and there are translucent gray tubercles distributed throughout. The spleen contains a few gray tubercles. The small and large intestines show numerous tuberculous ulcers especially in the lower part of the ileum. The mesenteric glands are greatly enlarged, and some are caseous.

Case VI.—Braxton R., five moths old, colored, was admitted to the hospital February 6, 1907. His father, mother, and two sisters are living and well. Infant's birth was normal, and it had never been sick until six weeks before admission. During this time he had a severe cough. Mother states that a dark-colored discharge comes from the nose during coughing attacks. The physical examination revealed an emaciated colored infant, whose lungs showed numerous moist rales both anteriorly and posteriorly. A smear made from the throat, in order to obtain some expectoration, showed no tubercle bacilli. Heart and abdomen were negative. There was a small amount of blood in the bowel movements. Infant died eleven days after admission.

The *necropsy* report follows: Caseous bronchopneumonic tuberculosis; general miliary tuberculosis; tuberculous pleurisy and peritonitis; tuberculosis of liver, spleen, kidneys, and intestines; fatty heart and kidneys.

The pleura on the left side is normal, but on the right side there are tuberculous adhesions. The left lung measures 9 x 5 x 3 cm., and weighs 45 grams. The air content is greatly decreased, and there are a number of large yellow tubercles scattered throughout the lung. The right lung measures 10 x 6 x 3.5 cm., and weighs 85 grams. This lung shows extensive pneumonia with many yellow tubercles, and tuberculous infiltration around the small bronchi. Crepitation is absent; the lung is firm in consistency, and the amount of blood is increased. At the right apex posteriorly the pulmonary

tissue is broken down, leaving a tuberculous cavity, 2 x 1 cm. On the parietal peritoneum are seen a number of discrete yellow tubercles, 3 mm. in diameter, having the appearance of bovine tuberculosis. There are miliary tubercles in the liver, spleen, kidneys, and intestines.

The six cases of cavity formation all occurred in negro infants between the age of five and fifteen months. Our experience bears out the well-known fact that the colored race is especially susceptible to tuberculosis, and the cases autopsied usually showed widespread lesions of an advanced type. Miliary tubercles were present in the lungs especially, and to a less extent in other organs. These lesions were usually small, in all likelihood an evidence of terminal infection, and were secondary to the larger lesions, either of the lungs or lymphatic nodes. The bronchial glands were tuberculous in all these cases. The pleura in our cases showed widespread adhesions or distinct miliary tubercles. No data were obtainable, either in the family or personal histories of the cases, which would indicate the source of the infection. In only one case had there been any disease prior to the fatal illness, namely, one infant having had an attack of gastro-enteritis (Case IV). Three of our cases had been fed exclusively on the breast until admitted to the hospital. The cavity formation in the lungs was with one exception of short duration, and occurred in the larger caseous areas of the lungs, and was not surrounded by fibrous tissue so commonly found in older children. In one case only was the process of longer duration, showing the formation of fibrous tissue around the borders of the cavity.

In conclusion we may say cavity formation does not occur with the same frequency in infants as in older children. In our own autopsies 16 per cent. of the infants dead from tuberculosis showed cavity formation: a far lower percentage than is reported by other observers. The type of lesion observed by us is not of the variety which lasts long enough to produce cavity formation. The infants usually succumb to the widespread process before the larger lesions have time to soften and their contents to be thrown off. In our cases tuberculous cavities in infants occurred most often at the root of the lungs, in the middle and lower lobes.

TWO CASES OF MYXŒDEMA.[1]

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The nervous symptoms of myxœdema are so characteristic and often so profound as to lead one to consider whether the disease is not essentially one of the nervous system. In this respect the term myxœdema is not so good a designation for the disease as is hypothyroidea; and in general treatises if it is not classed among diseases of the thyroid it should have no place other than with nervous diseases. Even in complete cases, notwithstanding the fact that there are marked changes in the skin, the fundamental disability and real pathological conditions to be described and treated are in the central nervous system. It is probably some serious change in the nutrition of the nerve elements, and most of the symptoms are those of nervous exhaustion and malnutrition.

Case I.—With a diagnosis of neurasthenia, Mrs. F., a large, fattish woman, aged forty years, pale, careworn, lacrymose, and feeble, was admitted to the wards of St. Timothy's Hospital, Roxborough. Not only was she physically weak, but it was quite evident by a glance at her face that she was the subject of a profound neurosis. Her eyes were sunken, her eyebrows were arched upward, and her forehead was wrinkled. Indeed, the term "sorrowful fatigue," so aptly applied by Ewald, describes her expression so completely that it is not necessary to say more except that she was pale and her flesh flabby.

On pulling down the bedclothes, it was at once seen that the yoke of myxomatous tissue encircling her thorax was marked. Large pads of such tissue covered the abdomen, buttocks, and thighs. Her bones were small and her muscles ill developed and flabby. The skin of the legs and feet was somewhat dry and scaly.

She was of that type of middle-aged woman who had borne four children and had largely supported them by labor in a mill and as a washerwoman. It was quite apparent that the left breast was the seat of a slow-growing carcinoma. This fact, together with the subnormal condition of her blood and general nutrition, was responsible for her neurasthenic condition, which was indeed pitiable. She worried over trifles, cried at a look, nor could she sleep; her nights were restless and unrefreshing. Her memory was very poor and her carcinomatous breast was the source of much mental perturbation. Her knees creaked and crepitated when they were bent, and, what was highly significant, gave way under her from weakness to

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such an extent that she was prevented from walking very much. Of all the asthenic symptoms of this disease, this one may be singled out as perhaps the most marked and the one of which all patients complain the greatest. She had deafening tinnitus and visual hallucination, very common things for myxomatous patients to experience, as is the sensation of being cold constantly, even in summer; from the latter she suffered, inasmuch as her temperature was subnormal and her skin sensations deranged. She had no spade-like deformity of the hands, no supraclavicular pads, but her abdomen, breast, and thighs were covered with myxœdematous tissue that trembled when it was palpated; her skin was dry, bluish, lusterless, and scaly. She had a history of profuse uterine hemorrhages that were prolonged and exhausting. She has always been large, weighing at one time two hundred and ten pounds.

Her circulation was weak and her heart sounds clear but feeble, and while there were no adventitious sounds, and the size of the heart and its position about normal, the sounds had no tone. Her blood pressure was 110 mm. Hg. Her chest was emphysematous, her breath sounds weak. The liver and spleen were normal; the abdomen was pendulous and distended. She suffered from angenoid pains under the left breast. Her stomach was normal; there seemed to be some hypoacidity. She was always obstinately constipated and her tongue was thick and yellow. Her urine showed casts and albumin; there was no sugar. In 40 per cent. of these cases albumin and casts appear, only to disappear under thyroid treatment. Her blood examination was as follows: The red cells were 3,500,000, the white cells 12,400, the hemoglobin 44 per cent. A differential count of the leukocytes revealed: Polynuclears, 62.6 per cent.; small lymphocytes, 33.6 per cent.; large lymphocytes, 1.6 per cent.; and myelocytes and eosinophiles (each), 1 per cent. The blood platelets were increased. Normoblasts, poikilocytes, anisocytosis, and polychromatophilia also were noted. The presence of the carcinoma largely contributed to the anemia, and altered the blood picture.

Violent paresthetic pains in her calves prevented her from sitting or walking with any comfort. The major reflexes were normal, her pupils reacted normally, and her eye grounds were normal. There was no tremor. Her uvula was œdematous, as was her pharynx, and her voice was high pitched and strident.

Under two grains of thyroid extract and arsenic and iron for her anemia, opium for her insommia, aspirin for her pains, her condition improved wonderfully. She lost seventeen pounds, lost her depressing nervous symptoms and pains, she became stronger, slept well, was cheerful, and happy. Her voice became more soft and less strident, and with the disappearance of her myxœdema the sensation of cold gave way to normal sensations. Under the thyroid treatment she suffered from thyroidismus to the extent that

this drug had to be discontinued occasionally. This was characterized by fever, tremors, and palpitation of the heart, as it usually is.

Myxœdema is a disease of many symptoms, so much so that one may be confused and feel that he is merely dealing with a neurasthenic, rheumatic, fat woman, while in reality he is dealing with a disease that yields most readily to treatment. I have seen five cases of myxœdema, all of which were misdiagnosticated and accordingly mistreated for years. This is true also of the following case, that is typical of this disease in every particular:

Case II.—Mrs. G., aged sixty-two years, married, consulted me for an overpowering weakness of the knees, dyspnœa, rheumatic pains, most pronounced œdema of the face and trunk, and all but total deafness. Upon seeing her for the first time I was impressed by the fact that her face was not only œdematous, but was stolid, wooden, and what little expression illuminated her countenance was that of woful fatigue. The upper eyelids were so œdematous that they at times obscured her vision and hung over the palpebral fissure. They were so translucent as to suggest true dropsy, and many physicians had been led to believe that she was far advanced in renal disease. In order to attempt to see she was compelled to wrinkle her forehead so that her brows were arched continually. Her face was squarish and there were reddish patches over each cheek bone.

She told me that her weight was two hundred and two pounds and she was five feet two inches in height. Not only was she so deaf that I was compelled to shout in her ear, but her mentality was dull and slow; and she suffered extremely from the cold. She is the mother of nine children, and her illness began shortly after the last child was born, near the menopause. She had previously been a robust, healthy woman of good family and personal history.

A careful physical examination showed, in addition to the myxœdematous face, deafness, and dull mind, solid œdema of the trunk and extremities. Her skin was dry and scaly; especially over the extensor surfaces it was thickened, dry, and coarse. When she removed her stockings the floor was often strewn with scales. She suffered from roughness of the skin and dandruff. In places the skin was waxy, translucent, and jelly-like. Above the clavicles and in the anterior axillary folds there were marked pads of myxœdematous tissue. Vast masses of this tissue formed a yoke over her thorax, her abdomen was distended and pendulous and covered with a myxœdematous pad. The axillary and pubic hairs were all but gone, and her eyebrows were scanty and thin. The outer half was characteristically (as Hertoghe has shown) thinner than the inner half. Palpation of the thyroid showed a very small organ; indeed, it is very doubtful if it was felt at any time. There was true œdema (dropsical) of the legs, due to her weak heart. Her joints were the seat of violent pains at times, and creaked and crepitated when they

were flexed and extended. Her voice was raucous and harsh and her speech slow and scanning. Her uvula and pharynx were œdematous and translucent.

Examination of her heart showed that its impulses were weak and feeble, though rythmic; while it was difficult to define the area of dulness, it was quite apparent that she suffered from a dilated heart and a degenerative myocarditis. There were no adventitious sounds; the systolic sound was feeble and the second decidedly unaccentuated, showing a very weak vascular tone, as the blood pressure (90 mm.) showed. Her lungs were slightly emphysematous, but there were no other evidences of any changes in the structure. The liver and spleen were normal in size. Her digestion has always been poor, and she evidently has suffered from hypoacidity. She has suffered from most obstinate constipation at all times.

Her nervous symptoms were striking and gave her the most trouble. She had a dull brain as well as dull hearing; indeed, her memory was so poor and perceptions so slow that her daughter told me that she had to serve as brains and ears for her mother, and it quite wore her out to have to talk to her. Not only was her hearing bad, but she suffered from tinnitus to such an extent that she often summoned her family to answer the telephone when in reality no bell rang; she often heard pistol shots in her ears; she had hallucinations of sight, and frequently thought she saw people in her room when none were present.

Her temperature was 97° when first seen, and in the summer she hugged the fire and wore extra wraps in order to keep warm. This is as characteristic of myxœdematous cases as pyrexia is of typhoid fever. She perspires at times unevenly; the perspiration will stream from one area of the skin and nowhere else, dropping at times from her forehead to the floor.

The major reflexes were normal, there was no Babinski's or Kernig's sign. Her pupils were small, equal, and sluggish to light; her station is fair, her gait unsteady; her sensations were diminished, and motor apparatus weak. All fine movements were clumsy and awkward. She fell at the slightest provocation, and her knees gave way under her. She had constant backache and joint pains. Under her left breast she had characteristic anginoid pains. Her gait was awkward and hippopotamus-like. Her urine was absolutely normal in every particular, it had a specific gravity of 1020, was acid, and devoid of sugar, albumin, and casts.

Her blood was in surprisingly good condition considering the length of her illness: Hemoglobin, 88 per cent.; red cells, 3,560,000; white cells, 7600; the color index 1.1. A few normoblasts were found. I have found these in all four cases of myxœdema.

She had no history of anything amiss with the pelvic organs, except about the menopause there was a period of severe uterine hemorrhages which lasted for weeks.

Her eyes were carefully examined by Dr. Wendell Reber, who found nothing abnormal for a woman of her years. Dr. Reber also found that the poor hearing was due to poor air conduction and probably to some labyrinthine involvement. Myxomatous infiltration of the pharynx and Eustachian tubes was probably the cause. Her nose and throat were highly myxœdematous. She was troubled for some time by attacks of constant sneezing.

It was as if a large heavy blanket had been cast all over this woman, weighing her down and interfering with all movements and with her senses of sight and hearing, and instead of keeping her warm it made her cold and caused her to be dyspnœic and oppressed.

Under 2 grains of thyroid extract three times a day this woman improved wonderfully; at the end of a month she was well except for her weak heart. Her hearing was restored to the extent that she could take part in all conversations in her rooms in ordinary tones. Her voice lost its raucous tones and the scanning speech disappeared. The pains in her back and joints were dispelled, her countenance lost its stolidity and œdematous appearances, and she became bright and animated. Her weight was reduced to one hundred and seventy-eight pounds in a month. The thyroid had the effect of causing her to feel warm and nervous. Once or twice she suffered from thyroidismus with fever tremor and rapid pulse. After six weeks' treatment with thyroid, digitalis, and massage her myxœdema disappeared completely, and save for a weak heart and pulse of 92 and slight tinnitus in damp weather, she has no symptoms. The thyroid caused her hair to fall out and her hands to scale excessively, but her hair began to grow immediately.

After thyroid extract nothing does these patients so much good as massage; it was the only thing that seemed to help this woman before she took the thyroid extract.

FATAL ANEMIA OF UNKNOWN CAUSE IN A CHILD OF FIVE YEARS, WITH UNUSUAL CELLS IN THE BLOOD.

By Williston W. Barker, M.D.,
of Dorchester, Massachusetts.

On December 8, 1908, I was asked to examine the blood of a patient with the following history:

C. B., a female, aged five years, white, adopted, but of known and healthy parentage, had always been a particularly healthy child, except for a capricious appetite. Three months ago she had a mild attack of measles without evident sequels. For four or five weeks she had been pale and listless. Ten days ago three small

purplish spots appeared on her face, fading gradually. Four days ago a blood stain was found on her pillow, and its source was determined to be an oozing gum which she had picked. Next day, by advice of the family physician, a dentist packed the gum, without result; on the following day, a gutta-percha packing did control the bleeding over night, but on December 8 it began again. On December 7 fresh purpura was observed on the legs. The whole amount of blood lost was very slight, there never being any active hemorrhage or clots seen.

Physical examination showed a well developed and nourished child, with moderate, slightly yellowish pallor of the skin. The mucous membranes were of fair color. No glands were felt. The teeth were good; the throat was normal. Thin blood was adherent to the left upper gums, which were not swollen or necrotic. A loud systolic murmur was heard all over the cardiac area and in the neck; the heart was not enlarged. The pulse was of notably low tension. The lungs, liver, and spleen were normal to physical examination. There were three small greenish purpuric spots on the face, two on the back, one on the front of the chest; many large purple spots were found on the legs and thighs. The temperature was 99°, the pulse 80, the respirations 20. The urine was pale, alkaline, had a specific gravity of 1006, and albumin, sugar, blood, diacetic acid, and acetone were absent. The stool was free from blood by the benzedin test.

The child failed progressively, and died January 27, 1909.

Treatment, consisting of out-door life, forced feeding, orange juice, electricity, and various drugs, was entirely ineffectual. The temperature was constantly elevated, reaching 103.5° at night. From January 5 to January 15 there was an attack of chicken-pox, with vomiting and rapid loss of strength. There was temporary diplopia about December 15. There were a few more purpuric spots at intervals, and once there was slight epistaxis. During the last two weeks of life there was slight œdema of the face and ankles and an extraordinary swelling of the gums, which almost completely covered the teeth. No glands were ever felt, except a few under the jaw after the swelling of the gums. The spleen was never demonstrably enlarged. The urine contained a trace of albumin at one examination. Death in coma followed twenty-four hours of active delirium. An autopsy was not permitted.

The blood (see table) showed a marked and progressive poverty of all the myelogenic elements (red cells, polynuclear leukocytes, and platelets). Toward the last there were many very young basophilic cells, and many erythroblasts. There was marked macrocytosis from the first, but comparatively little variation in the shape of the red cells. Polychromatophilia was extreme, but stippling was absent. No eosinophiles or mast-cells were ever seen. The coagulation time on December 8 was the same as my own—three and one-

Fig. 1

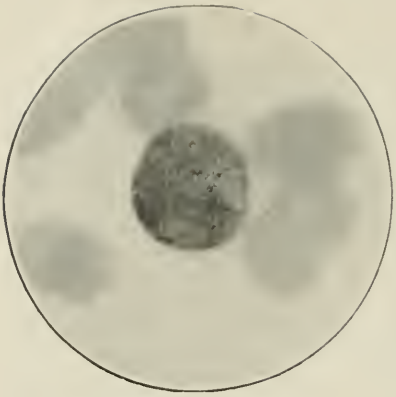


Fig. 2

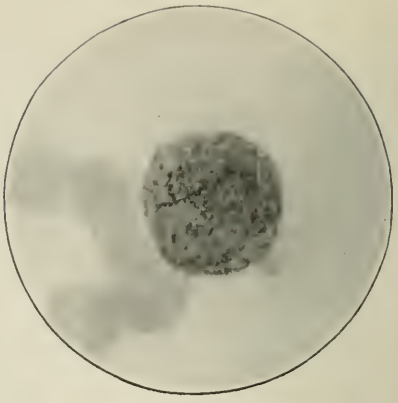


Fig. 3

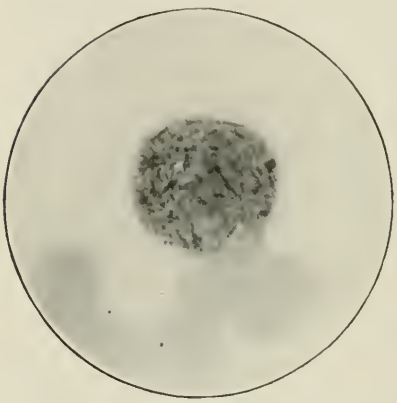
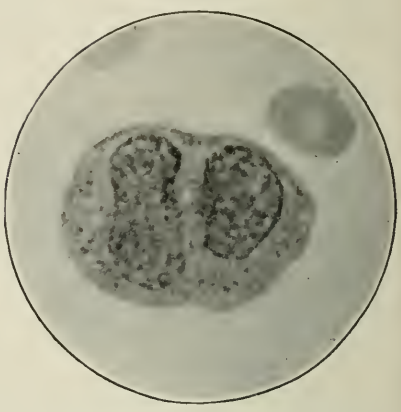


Fig. 4



Figs. 1 to 4 are cells from the blood. In No. 1 two rods are shown plainly, forming an *X*, and several above that are less distinct. In No. 2 the rods form a *Y*, while in No. 3 they are scattered all over the cell. In No. 4 the rods are less clear, but there is a large double nucleus.

Fig. 5

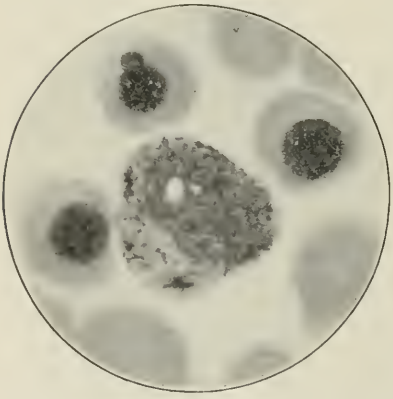
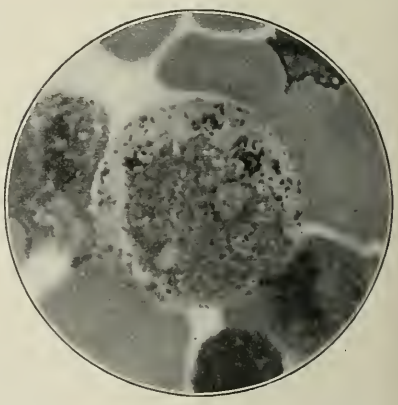


Fig. 6



Figs. 5 and 6 are cells from crushed fœtal liver tissue, prepared and stained like the blood smears. They both show scattered rods, and No. 6 shows a nucleus in division. × 1500 diameters. (Wright's stain.) (Photomicrographs by L. F. Brown, of the Massachusetts General Hospital.)

half minutes. A marked serum ring appeared on the Tallqvist paper, on January 5.

Blood Examinations, C. B.—1908-1909.

	Hemoglobin per cent.	Red cells	White cells	Platelets	Polynuclear neutrophiles per cent.	Lymphocytes per cent.	Young basophiles, per cent.	Myelocytes, per cent.	Special cells, per cent.	Erythroblasts per 100 whites	
										Megaloblasts	Normoblasts
Dec. 8, 1908	65	3,360,000	2100	Very few	15.0	82.0	0.0	3.0	0.0	0.0	2.0
" 15, "	60	2,216,000	1900	Very few	5.0	93.0	0.0	2.0	0.0	3.0	0.0
" 22, "	60	2,212,000	1300	Absent	2.5	95.5	0.0	2.0	0.0	1.0	0.0
" 29, "	65	1,776,000	1500	Absent	6.0	90.0	1.0	3.0	0.0*	8.0	3.0
Jan. 5, 1909	45	1,568,000	3100	Absent	1.0	90.0	9.0	0.0	0.0*	0.0	0.0
" 20, "	20	944,000	4300	Absent	2.5	42.5	53.0	0.0	2.0	7.0	6.0

* In count of 200 whites.

In the smears taken January 20, and rarely in those of January 5 and December 29, there were found certain cells of which I have not been able to find a published description. Dr. J. H. Wright, who was kind enough to examine the smears, had observed similar cells in the liver of a four months' fœtus about two years ago. These cells (Figs. 1 to 6) are large, rounded or oval, with from one to three vesicular neutrophilic nuclei and pale basophilic protoplasm; in the protoplasm there is more or less coarse granular material staining a deep red; in some of the cells this material takes the shape of straight or slightly curved rods, some of them beaded. That these rods are not artifacts is proved by their appearance in many smears, stained with different bottles of stain and by different persons. They are similar to rods seen in the protoplasm of lymphocytes in a case of acute lymphatic leukemia reported by Auer,[1] but the cells are not lymphocytes, nor prelymphocytes. In the absence of postmortem evidence I venture no hypothesis as to the place of these cells in blood formation. They are obviously young cells, many of them in active division, but none of them show evidence of phagocytic activity. It is equally futile to discuss the classification of the case. There was certainly grave impairment of the cell-producing activity of the bone-marrow, but not of the aplastic type. Leukemic metaplasia of the bone-marrow is not an impossible explanation.

The case is reported partly because of the rarity of fatal anemia in early childhood similar to the "pernicious" anemia of adult life, and partly to place on record the appearance of the cells described. I wish to acknowledge my indebtedness to Dr. J. T. Sherman, of Dorchester, for allowing me to make this report; to Dr. J. H. Wright and Dr. R. C. Cabot for helpful advice and information; and to Mr. Brown for the illustrations.

[1] Amer. Jour. Med. Sci., 1906, cxxxi, 1002.

THE OCULAR COMPLICATIONS OF NASAL SINUS DISEASE.[1]

By Arnold Knapp, M.D.,

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The most striking and probably the best known orbital complication of nasal sinus disease is that due to a dilatation of the frontal and ethmoidal sinuses without inflammatory signs, the so-called mucocele, which causes a mechanical displacement of the orbital contents. The eyeball is generally uninvolved. The distention which the sinus undergoes is associated with rarefaction of the bone, usually of the inferior wall. On the other hand, in a case which I recently operated upon, the upper or cerebral wall of the frontal sinus was absent and the altered mucous membrane of the dilated sinus was directly adjacent to the dura of the anterior cranial fossa. The only treatment is operation, and is usually simple, as the long-standing displacement of the eyeball permits very free access to the nasal structures. The operation[2] consists in the removal of the lower wall of the frontal sinus and of the entire ethmoid labyrinth, thus permitting free drainage into the nose.

Inflammation of the sinuses is associated with external or orbital inflammatory signs whenever the inflammation invaded the intervening bony wall. In the mild cases there is no macroscopic change in the bone, but a periostitis is present over the floor of the frontal sinus with swelling and redness of the eyelid or over the ethmoidal os planum, causing exophthalmos. This condition may resolve of itself or on intranasal treatment. If the involvement of the bone is more active a discoloration or a circumscribed defect in the bone will occur usually in a typical site in the floor of the frontal sinus or at the ethmoidal os planum. The pus thus gains its way to the periosteum and forms a subperiosteal orbital abscess which either remains encapsulated or may extend forward and perforate the skin of the eyelid; the suppuration may then cease, the wound heal, or a purulent fistula remain. The orbital contents are generally not involved in these cases beyond slight venous congestion at the optic disk, as the periosteum is easily detached from the bone and protecting granulations spring up.

Subperiosteal orbital abscesses are not infrequent in children, the infection being transmitted by the ethmoid labyrinth. In operating upon these cases a curved incision is made along the inner orbital margin; on retracting the periosteum the small collection of

[1] Read at a meeting of the New York Academy of Medicine, December 3, 1908.

[2] Arnold Knapp, The Surgical Treatment of Orbital Complications in Diseases of the Nasal Accessory Sinuses, Jour. Amer. Med. Assoc., July 25, 1908.

pus will be encountered between the periosteum and the bone and usually a small irregular defect in the os planum. The opening in the bone should be slightly enlarged and the contiguous ethmoidal cells curetted; the case then heals without deformity.

If the orbital periosteum itself is invaded, which is most likely in the places where it is normally adherent, as for the passage of vessels, involvement of the orbital structures results, cellulitis or abscess. The optic nerve becomes inflamed and the ophthalmoscopic changes at the nerve head may be more pronounced on the nasal side. This was especially noticed in one of my cases in which the lesion in the orbital periosteum corresponded exactly to an area of necrosis just below the posterior ethmoidal foramen. The orbital condition recovered after the ethmoidal focus was removed.

The important and preponderating part played by nasal sinusitis in producing orbital cellulitis and abscess has, strange to say, only been recognized within the last years. Birch-Hirschfeld,[3] in an elaborate statistical research, concludes that nearly all orbital inflammations are caused by nasal sinus empyema. Under these circumstances the usually practised blind incisions into the orbital cavity are not surgical; the treatment should be based on the lines of the orbital operation for nasal sinus empyema, aiming at broad exposure by an incision along the orbital margin, careful inspection of the orbital walls, and proper drainage according to the individual case.

The importance of the maxillary antrum in causing orbital complications has in my mind been exaggerated. Disease of the antrum itself rarely seems to cause an orbital complication unless through the intermediation of the ethmoidal cells. The extension much more frequently follows a periostitis of one of the walls, as after an alveolar abscess. Thrombophlebitis of the orbital vessels, according to Uhthoff, is unusual after accessory sinus disease, and is more frequent from extension of a process in the face.

A very important group of ocular complications of nasal sinus disease are those of the optic nerve. That the optic nerve sometimes suffers in sphenoidal sinusitis has been known for some time, and brilliant cases of restoration of sight have been recorded by Holmes and others by timely treatment of the sphenoidal sinus. The recent investigations of Onodi[4] have shown that the optic nerve frequently is in close relation to the posterior ethmoidal cells and that the extreme thinness of the intervening wall makes the involvement more likely than in the case of the sphenoid. Cases have been observed that bear out the frequency of this involvement. Of especial interest is that the optic nerve is involved in the form known as retrobulbar neuritis in which a central scotoma is the characteristic functional defect. It is not known why this particular bundle of optic nerve

[3] Versamml. Deut. Naturf. und Aerzte, Dresden, 1907.

[4] Der Sehnerv und die Nebenhöhlen der Nase, Vienna, 1907.

fibers is first and principally involved. The optic nerve shows in the beginning no ophthalmoscopic changes unless the nasal infection is situated in the anterior ethmoidal cells, as in a recent case of well-marked papillitis with central scotoma, which I had the opportunity of following together with Dr. Coakley. The prognosis in these cases is excellent, and now that ophthalmological attention has been focussed on this interdependence of the nose and the eye, some of the hitherto obscure cases of acute retrobulbar neuritis will be recognized and nasal treatment promptly instituted.

Ocular paralyses are not infrequently the only manifestations of orbital complications. In a case of grippe with ethmoidal involvement which came under my observation the internal rectus for a time was paretic.

Neuralgia, especially ciliary and retrobulbar, is frequently due to sinus affections, and asthenopia is a common symptom. According to Kuhnt,[5] muscular asthenopia, accommodative asthenopia, and contraction of the visual field, are the functional disturbances observed in nasal empyema in the order of their frequency; he believes them to be due to absorption of toxins from the purulent focus in the sinus.

There is a group of eye affections comprising iridochoroiditis, glaucoma, and detachment of the retina, with which certain authors have brought sinus disease in etiological relation. Some over-enthusiastic observers have even cured these conditions by surprisingly insignificant operations on the nasal sinuses. This is a subject upon which we require additional information. Kuhnt, whose experience is surely great, states in the above-quoted article that he has never seen a case of iritis or choroiditis caused by sinus disease, but thinks that cyclitis, especially the form chiefly characterized by opacities of the vitreous, can depend on this cause. He has never observed glaucoma to be caused by nasal disease, but believes that in predisposed eyes an attack may be brought on by a sinus affection. A number of cases of this kind have been observed. The reported cases of detachment of the retina associated with nasal sinus disease are not convincing.

That the list of ocular disturbances which can be produced by sinus affections is not exhausted there can be no doubt, and a great deal of careful work needs still to be done to complete our knowledge of the relation of nasal sinus disease to disease of the eye.

[5] Deut. med. Woch., 1908, No 37.

THE CAMMIDGE REACTION IN EXPERIMENTAL LESIONS OF THE PANCREAS.[1]

By John Speese, M.D.,
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AND
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(From the William Pepper Clinical Laboratory of the University of Pennsylvania, and the private laboratory of Dr. John H. Musser, Philadelphia.)

Although abundant clinical proof has been afforded his reaction by Cammidge, it has thus far remained for others to publish results of work conducted along the fundamental lines of chemistry and experimentation. Reports of experimental studies have by no means kept pace with those derived from clinical observation, which is the more to be regretted since the value which would accrue from the establishment of a diagnostic measure in pancreatic disease is inestimable and demands a firm experimental basis before it can be accepted.

In a preliminary report[2] we published results of some studies of the Cammidge reaction in experimental pancreatitis, using the "C" reaction, which had given good clinical results in the hands of one of us.[3] Up to that time no report of experimental work had appeared in which this modified reaction had been used, but since our first report Eichler and Schirokauer[4] have published results of some experimentation in dogs, using the "C" method. Their work has left them somewhat in doubt as to the usefulness of the test, the crystals appearing at irregular intervals even when definite pancreatic lesions existed.

The work which we report at this time was undertaken to confirm or to refute Cammidge's claims, that by means of chemical analysis a specific substance could be demonstrated in the urine of individuals suffering with pancreatic disease. This specific substance was thought by Cammidge to be glycerin, absorbed into the circulation during the process of fat necrosis and excreted finally in the urine. This hypothesis was soon abandoned for the present belief that the substance which is isolated by means of phenylhydrazin is a pentose, not found as such in the urine, but obtained from some mother substance by hydrolysis with hydrochloric acid. What this mother substance may be is not stated, but its origin is thought

[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 11 and 12, 1909.

[2] Amer. Jour. Med. Sci., January, 1909.

[3] Annals of Surgery, February, 1909.

[4] Berl. klin. Woch., 1909, p. 352.

to be from the pancreas, which, as is well known, contains a relatively large amount (2.48 per cent.) of pentose-yielding material.

We have endeavored in our series of cases to imitate the acute and chronic types of pancreatitis met with in human beings. The first is easily done by the injection of oil into the pancreatic duct, while the morbid changes seen in the second type of pancreatic lesion were closely simulated by simple ligation of the duct of Wirsung. All of our experimental animals were dogs, and before operation they were confined for from one to more days in a cage used for metabolic studies. The food consisted of meat, potatoes, bread, and water. The urine was collected for twenty-four hours preceding operation, and in every instance, with the exception of three of our early animals, it was examined for albumin, sugar, and the Cammidge reaction. Albumin was tested for by the Heller's test, and if present was removed by heat and acid. Sugar was tested for by Fehling's and Nylander's methods, and the Cammidge reaction was performed according to the directions given in Robson and Cammidge's book.[5] This is the so-called "C" reaction. It may be stated parenthetically that albumin was almost a constant constituent of the dogs' urines; sugar was never found before, and with the exception of our extirpation series, never after operation; and the Cammidge reaction was found but once before operation, this dog, of course, being discarded as valueless in our experiments.

Operation was performed under aseptic conditions, ether being used as an anesthetic. Postoperative suppuration was never seen, although no dressing or protection was given the wound. The urine obtained after operation was examined for albumin, sugar, and for the Cammidge reaction. Seventeen dogs were studied—six for acute pancreatitis, four for the effects following ligation, two for the effects following extirpation of half of the pancreas (tail), three for the effects following crushing of the tail of the pancreas, and in two dogs a total extirpation was performed.

Series I.—The pancreatic lesions in this series were induced by the injection of from 10 to 30 c.c. of cotton-seed oil into the pancreatic duct, a procedure which causes rapid necrosis of the pancreas.

Dog 1. The urine before operation was not examined. The dog was killed eight hours after operation, and the urine collected up to that time gave a positive reaction. Macroscopic examination of the pancreas showed a diffuse necrotic process which involved most of the organ. A microscopic study revealed complete destruction of the tissue.

Dog 2. The urine before operation showed no Cammidge reaction. The dog was killed eight hours after operation, and the urine failed to show a positive reaction. The pathological examination of the pancreas showed neither macroscopic nor microscopic

[5] The Pancreas, its Surgery and Pathology. 1907.

lesions, as the attempt to inject oil into the pancreatic duct had been unsuccessful.

Dog 3. The urine before operation was not examined. The dog was killed eight hours after operation, and the urine collected up to that time showed a positive reaction. The postmortem examination of the pancreas revealed a diffuse necrotic process; lesions similar in nature were seen microscopically.

Dog 4. The urine before operation showed no Cammidge crystals. The urine after operation gave a positive reaction. The dog was killed eight hours after operation, and the macroscopic and microscopic examination of the pancreas showed a diffuse necrotic process.

Dog 5. The urine before operation was not examined. The urine after operation gave a positive reaction. The animal was killed eight hours after operation, and the pancreas was found to be necrotic except at the tail. Microscopic examination of the head revealed complete necrosis of the tissue, with hemorrhagic and leukocytic infiltration. In the tail there was marked parenchymatous degeneration and a few areas of necrosis.

Dog 6. The urine before operation showed no Cammidge reaction. The urine the first hour after operation was negative; four hours after operation it was positive. The dog was killed at this time, and the usual necrotic appearance following oil injections was seen. On microscopic examination, normal tissue was nowhere visible except in the tail.

Series II.—The pancreatic lesions in this series were caused by ligation of the pancreatic duct.

Dog 7. The urine before operation was negative for the Cammidge reaction; the urine the first day after operation was negative; the urine the second day after operation gave a positive reaction; on the third, fourth, and fifth days, positive reactions were obtained. The dog was killed seven days after operation, and an area of induration was found in the head of the pancreas opposite the point of ligation. The lesion, which was the size of a large walnut, was distinctly hard, and cut with greater resistance than the remaining portions of the gland. Microscopic study of the indurated tissue showed a subacute inflammatory process, which had caused small areas of necrosis in the acini. A leukocytic infiltration, consisting for the most part of round cells, was seen in the stroma and, to a less extent, about the acini. Proliferated connective tissue cells were also noted.

Dog 8. The urine before operation showed no Cammidge reaction. Positive reactions were obtained on the first and second days following operation. The dog was killed on the second day, and the pancreas was found to be indurated in an area about the size and shape of a hickory nut, the lesion being directly above the ligature, which still surrounded the duct. Microscopic examination

revealed changes similar to those seen in the preceding case, but not so extensive.

Dog 9. The urine before operation was negative for the Cammidge reaction. Negative reactions were obtained on the second, third, and fourth days following operation. On the fifth and sixth days after operation an attempt was made to open the pancreatic duct at the point of ligation, but was unsuccessful, owing to the induration about the duct. The first day after the second operation the urine was lost; on the second and third days the reaction was negative. On the fourth day typical crystals were found, and on that day the dog died. The urine collected on this day up to the time of death gave a negative reaction. Owing to advanced decomposition the pancreas was not examined microscopically. We are unable to explain the irregularity in the appearance of the crystals in this case.

Dog 10. The urine before operation was not examined. The first day after operation it was negative, but positive reactions were obtained on the second and third days. On the third day the duct was again exposed, the ligature cut, and the intestine opened. A probe was inserted in the duct and a point of resistance was encountered 1 cm. from the ampulla. Gentle pressure overcame the resistance and the probe was advanced still farther. On withdrawing it the same resistance was again met with. The head of the pancreas felt decidedly indurated. The urine following this second operation was so scanty that it could not be examined. On the second and third days negative reactions were obtained; on the fourth day a positive reaction, on the fifth a negative, on the sixth and seventh days positive reactions, and thereafter negative reactions were obtained. On the fifteenth day after the first operation, or twelve days after the second operation, the abdomen was again opened, and the omentum was found to be so adherent that it was impossible to locate the pancreatic duct. The dog was killed five days later, or on the twentieth day after the first operation; the pancreas was very much indurated and the duct could not be found. A microscopic examination was not made.

Series III.—In this series the tail of the pancreas was extirpated to a point immediately below the pancreatic duct.

Dog 11. The urine before operation gave no Cammidge reaction. The day following operation no reaction was obtained, but on the second day typical crystals were seen. The animal died three days after operation, and the autopsy disclosed a necrotic condition of the pancreas. The duct was found to have been ligated, so that this case can not be properly regarded as an example of the results following partial extirpation, but rather as an instance of incomplete extirpation plus ligation. Microscopic sections from the head of the pancreas showed well-preserved cells, the nuclei being slightly obscured by cloudy swelling. Small areas of liquefaction necrosis

were seen, and considerable hemorrhage was present in the tissues.

Dog 12. Urine before operation was negative. No pancreatic reaction was obtained on the first, second, third, fourth, fifth, and sixth days. Thereafter, until the nineteenth day, positive reactions were found. On that day the dog was allowed to run out of doors, and through carelessness the animal escaped. Examination of the excised portion of pancreas revealed a normal gland.

Series IV.—In all the cases in this series the tail of the pancreas was crushed with a hemostat in six places.

Dog 13. The urine before operation was negative. On the first, second, third, fourth, and fifth days positive pancreatic reactions were obtained. The dog was killed six days after the operation. The pancreas showed induration of the head and some injection about the tail, but no fat necrosis was apparent. Microscopic examination of the head of the pancreas showed some increase of connective tissue around the acini. The parenchyma seemed normal in appearance.

Dog 14. The urine before operation was negative. No urine was obtained the first day after operation. On the second day the urine was negative, but on the third and fourth days positive reactions were obtained. The dog was killed on the fifth day after operation. The tail of the pancreas was hemorrhagic and necrotic. The head was slightly indurated, the duct patulous. On microscopic examination the tail showed advanced necrosis of the cells, the head being practically normal.

Dog 15. The urine before operation was negative. Up to the sixth day after operation no Cammidge reaction was obtained; on the sixth and seventh days positive crystals were seen. The urine was not studied again until nineteen days after operation, when it was negative for the pancreatic reaction. Two days later, when we intended to kill the animal for the purpose of macroscopic and microscopic examination of the pancreas, we found that the dog had been removed from the cage and had been disposed of, so a pathological report on this case cannot be made.

Series V.—In this series a total extirpation was attempted.

Dog 16. The urine before operation was negative. The animal died the day following operation, and the urine collected up to that time gave a positive pancreatic reaction. Examination of the pancreas revealed a normal condition of that gland.

Dog 17. The urine before operation was negative. On the first and second days after operation positive reactions were obtained. The animal died on the second day. The pancreas seemed normal on microscopic examination.

We were led to believe from the results obtained in Series I, and from those seen in Dogs 7 and 8 (Series II), that occlusion of the pancreatic duct was the principal causative factor in the production

of the Cammidge reaction. We tried to establish this fact by removing the obstruction in the remaining two dogs of the second series, but were unsuccessful in Dog 9, and in Dog 10 positive reactions were obtained after two negative days. This may have been caused by a closure of the duct, but we have no means of verification, as there were so many adhesions that the pancreatic duct could not be located.

The view that occlusion of the duct might be the main cause of the appearance of Cammidge crystals had to be abandoned after we had completed Series III and IV, as the duct was left patulous in these cases, except in Dog 11 (Series III), and positive reactions were obtained in each case.

Cammidge has asserted that pentose is the specific substance which he isolates from the urine by his method, and that the pentose has its origin in the pancreas. We are not in favor of this view, for, were this correct, we should not expect to find typical crystals after total extirpation of the gland. We are inclined to believe that the substance found by the use of Cammidge's technique is but an expression of disturbed carbohydrate metabolism, this disturbance being referable to morbid changes in the pancreas. We have made no investigations along the lines of chemistry, but the melting point of the crystals found in one case was between 150° and 155° C., which is about the melting point of the phenylhydrazin compound of glycuronic acid.

Conclusions. 1. The Cammidge reaction is a constant feature in hemorrhagic pancreatitis, in mechanical injuries of the gland (crushing of the tail, partial extirpation), and in total extirpation.

2. In certain cases of the subacute type of pancreatitis the reaction is inconstant.

3. The nature of the phenylhydrazin compound is not definitely established. If pentose, it is apparently not derived from the pentose-yielding material of the pancreas.

4. A positive reaction is indicative of altered carbohydrate metabolism due to disturbance of the internal secretion of the pancreas.

THE TONSILS AS ELIMINATIVE ORGANS.

By William W. Ashhurst, M.D.,
of Chihuahua, Mexico.

The true functions of the tonsils and other adenoid structures abutting on the mucous membranes is a problem nearly as obscure today as it was twenty years ago. They were then supposed to secrete a substance which facilitated swallowing the bolus of mas-

ticated food, and also to prevent in some obscure way the entrance of infectious materials into the body.

Francis R. Packard[1] writes: "The faucial tonsils are really a large pair of lymphatic glands, the functions of which are but imperfectly understood. As with other lymph glands they probably protect the organism from infections of various kinds by acting as filters, and by in some way lessening the toxicity of microörganisms which come within their sphere of activity."

This seems to represent the sum and substance of the ancient and modern teaching in regard to the matter. But it is somewhat incomprehensible why the microörganisms should enter the tonsils at all, when it is to be supposed that they might just as easily slip by unharmed, into the pharynx and œsophagus. If there were such a thing as ciliated epithelium in the follicles and crypts of the tonsils, one might imagine a continuous current such as exists in the Fallopian tubes, sucking them in against their will. And yet the crpyts of the tonsils even in health are found loaded with a varied flora of pathogenic and non-pathogenic germs—Loeffler's bacilli, pneumococci, streptococci, and staphylococci in pleasing variety. The natural supposition would be, since the nose is constantly exposed to atmospheric infections, and the mouth to those brought by the food and otherwise, that many of these microörganisms failing to penetrate the mucosa of the nose and mouth find lodgement in the tonsillar crypts. This, however, is a very unsatisfactory explanation of their presence there.

The tonsils are composed as we know of adenoid tissues, resembling that of the internal lymphatic glands, as distinguished from the superficial, or those abutting on the mucous membranes, such as the lingual follicles, the tonsils, the solitary and agminated glands of the intestine. The great and evident difference is this, that the superficial lymphatic glands have no direct connection with the lymph vessels, while the deep lymphatic glands are directly in the course of these, and are penetrated by them in all directions. The superficial lymphatic glands are, however, surrounded at their bases by wide lymph spaces, and are described in works of anatomy and physiology as "hanging into" these lymph spaces which communicate by small channels with the larger vessels. That the superficial lymph glands are different in their functions from the deep glands may be inferred from their different arrangement in this respect and from the fact that the deep glands are often found enlarged without any corresponding enlargement of the superficial glands, which may be supposed to be connected indirectly with the same system of channels. The superficial lymph glands have a free blood supply, thus being connected intimately with the blood circulation.

Physiologically there is a certain similarity between the superficial

[1] Osler's Modern Medicine, 1907, iii, 596.

and the deep lymph glands, in that they both have "germinating centres" whence issue enormous numbers of wandering leukocytes. In the deep glands all these leukocytes are poured into the lymph channels whence they are carried into the venous circulation. With the superficial glands the course is different. Some of them are poured into the surrounding lymph spaces, whence they may eventually reach the lymph channels and the venous circulation; but a vast number of them penetrate between the epithelial cells covering the glands, thereby causing an outward current. It is with these escaping leukocytes and the part they play that we are now concerned. Their outpouring into the crypts, and their being found there together with innumerable microörganisms is probably more than a coincidence. It might be regarded as a never-ending succession of sorties by the defenders of the body against the invading horde of microörganisms, and the crypts and surfaces of the glands might be regarded as the battle ground where the infection or protection of the body is decided. But why should the invading microörganisms select such a well-defended part for their onslaught? There lies the whole improbability of the idea.

W. G. McCallum[2] says of the mode of invasion in primary intestinal tuberculosis, "the bacilli adhere to the mucosa, and work their way into its substance. Orth holds that the lesion begins always in the lymph nodules. . . . The first change is of the appearance of tubercle nodules in the substance of the mucosa and very frequently in the lymphoid nodules, or Peyer's patches."

Now so far as we have any positive knowledge, there is no proof that the superficial lymphatic glands ordinarily absorb anything. Of foods, fat is the only one ordinarily absorbed through the lymph channels, but there is not the least evidence that the superficial lymphatic glands have anything to do with this. The fat in the form of soap passes between the epithelial cells to the lacteals, which penetrate the villi, and are nowhere near the follicles and have no connection with them.

Russel H. Chittenten and Lafayette B. Mendel[3] say "there is some evidence that the lymphatic tissue elements are concerned in these synthetic transformations" (re-conversion of soaps into fats), but as the lacteals in the villi are surrounded by adenoid structure unconnected with the lymph follicles, it is the adenoid tissue in the villi that is more likely to be concerned than any other. Fat carrying leukocytes are found in the lacteals in the villi, but so far as I can learn they have never been observed in the lymph spaces surrounding the follicles, nor in other lymph spaces.

So that it seems so far we have no evidence that the superficial adenoid glands absorb anything during the digestive processes. Now if they are only capable of absorbing microörganisms, it would

[2] Ibid., p. 230.

[3] Ibid., i, 695.

seem that we should be better off without them. But by observing and studying their pathology some new light seems to be thrown on their functions, which will explain many things otherwise difficult to understand, such as McCallum's statement quoted above in regard to the invasion of the intestine by tubercle bacilli, that the tubercle nodules appear "sometimes in the lymphoid nodules or Peyer's patches."

Why is it that in diphtheria, scarlet fever, and other exanthemas the tonsils are often affected after other manifestations of the disease have been present for hours or days? This question first forcibly presented itself to me while I was attending a fellow-physician who, in opening a postscarlatinal abscess, received a scratch or prick on the dorsal surface of his right middle finger. The following day he performed an operation on another septic case, and while operating noticed discomfort in the finger. The following day the finger was worse and lymphangitis appeared on the forearm. On the fourth day he called me in. The finger was rather painful and moderately swollen, the swelling extending to the wrist. The red line of the lymphangitis reached the elbow. There was an enlarged and slightly tender anterior axillary gland; and slight fever and general malaise. He had himself freely incised the finger, and was treating it in approved antiseptic style. There was nothing wrong with the throat, nor any symptom of scarlet fever, which he had already had. About two days later the tonsil on the affected side (the right) became inflamed, and presented the typical appearance of scarlatinal tonsil. Two days afterward the left tonsil went through the same process. Convalescence was rather slow, but without notable incident. He himself examined a swabbing from the tonsils with the microscope to see if Loeffler's bacillus was present, but did not find it. Many staphylococci were present. I regret that no bacteriological cultures were made either from the pus from the finger or from the membrane in the throat. When first called I was rather surprised to find nothing apparently wrong in the throat, and was again surprised when the throat trouble appeared. Since the infection was both local and general when I first saw him, it appeared probable to me when the throat trouble developed that this was a form of elimination, like a diarrhœa as the result of an indigestion. The toxins or the toxins and the bacteria together were coming out through the tonsils thereby causing a local manifestation at their point of exit. It seemed that elimination was twofold: local, whereby the nearest tonsil was involved first, and general, whereby the other was involved secondarily. This seemed at first rather hypothetical, but in the light of further thought and observation it seems continually more probable, as explaining points otherwise obscure. Adopting this as a hypothesis, it explains the analogous condition of the solitary follicles and Peyer's patches in typhoid fever, a general and local infection (for the typhoid bacilli are probably absorbed

principally from the intestine) with elimination through the nearest superficial lymph glands. It explains the problem of tuberculous nodules in the lymph follicles in primary or secondary tuberculosis of the intestine. It explains the condition of the adenoid structures of the tongue in gastric and œsophageal diseases, of the pharynx in laryngeal affections, and in general the condition of chronic hypertrophy of these organs in all its forms.

It has been observed for years that tonsillar hypertrophy is not simply a local disease, but that the hypertrophy accompanies a diathetic taint or a condition of malnutrition; and that the removal of the hypertrophied organs often greatly benefits the general health is explained in two ways: First, the removal of a mechanical obstruction to respiration and deglutition, and, second, an improvement of the function of the tonsil, whose crypts and recesses have become so deeply situated that the normal outward flow is greatly impeded, and accumulations take place in them, to the detriment of the organ itself and of the whole body.

To some extent the different forms of hypertrophy may be regarded as indicating different forms of overstimulation. The hyperplastic form would indicate excessive elimination of some irritating substance from the general circulation, probably brought to the tonsil by the blood, and the true hypertrophic form would indicate local elimination, probably directly from the lymph space surrounding the tonsils, as in the case of nearby tuberculous disease. To some extent the true hypertrophy must be a compensatory condition, but nevertheless one requiring correction when excessive, as when the lacunæ are so deep that the eliminative function is interfered with.

The obscure relation between rheumatism and follicular tonsillitis will find its explanation in this hypothesis, rather than in the old belief that the rheumatism might enter through the tonsil. Some light ought to be thrown on the causes of appendicitis by this probable function of the adenoid structure in the appendix. Nor shall I be surprised if eventually some explanation along these lines may be found for gastric and duodenal ulcers and various other diseases of the digestive organs whose causes are still entirely obscure.

REVIEWS.

The Prolongation of Life; Optimistic Studies. By Elie Metchnikoff. English translation by P. Chalmers Mitchell. Pp. 343. New York and London: G. P. Putnam's Sons, 1908.

Several years ago M. Metchnikoff's book, *The Nature of Man*, became known to the English-speaking public in its translation by Dr. Mitchell, and excited no little pleasure by its broad and pleasant philosophical views of life, and varied comment for its propositions concerning the limitation of human existence and the proposal to employ certain microbes to interfere with certain toxic influences which the author held as largely responsible for the appearance of senile changes of the body. The present volume is not precisely a sequel, but at least an elaboration, of the former book. Throughout the work, dealing mainly with the matter of senescence, its development, the question of natural death, and the propriety and means of its postponement in man, but also devoting attention, in a number of semidetached essays, to phases of individual and socialistic life, to the material side of morality, an analysis of pessimism, and to an illustrative discussion of the life of Goethe and an interpretation of his "Faust," there runs a thread of argument sustaining the value of human life to the individual as well as to the community, appealing to the recognition of an ideal or standard life cycle in which the end of existence must come with a sense of satiety, if not of pleasure, to the man who has run his race. The general line of his argument is well known: that, owing to a variety of circumstances of accident and disease, the life of man but rarely reaches its fullest measure of years, and that old age, while evasive of thorough analysis and appreciation, is premature to the most of us from the influences of toxins absorbed largely from putrefying material in our large bowel and from the activity of phagocytes with the higher cells thus influenced. The proposition to avoid such all-too-early changes by preventing putrefaction in the alimentary canal through the agency of lactic-acid producing organisms (notably the Bulgarian bacillus) ingested for the purpose, is likewise well known; it is further discussed in the volume, its value sustained by argument from analogies and from a small amount of illustrative but mainly incomplete experience.

One finds much to admire in the genial consideration of what is so widely regarded as a disaster and an anticlimax, the loss of power

and cessation which succeed the highest purposes and efforts of life, and in the author's deliberate ignoring of that infantile acceptance of stock-immortality for the real desideratum of personal con inuation which has become so much the vogue in scientific circles. As a materialist, he accepts the condition of life, but seeks to improve it and expand its usefulness; and his lines are not laid in the lamentations of failure and the hysteria of poetic revilement of the existing state, but in the practical efforts of a wisely persuaded man to recognize his limitations and to obtain all the good they permit. One recognizes well the personality of the illustrative case of "the man of science" whose development into the efficiency of contentment Metchnikoff draws with absorbing care in his study of pessimism and its relation to health and age, a tale adding sympathetic interest to the whole growth of the author's views and one suggesting, in its evolution, the possibility of further steps of hopefulness were the surroundings of the subject to permit. Orthobiosis, the normality of the life cycle, the author's desideratum, is perhaps chimerical, but none the less to be sought for and approximated. That even in the best regulation of life, age and death are sure to come, Metchnikoff does not seek to evade; but he speculates whether even normal sensescence and death may not, like age and death from disease, depend upon material causes whose principle may run through all species, an autointoxication comparable to the effects of disease, appearing in the close of the cycle and in some way connected with fulfilment of perpetuation of species. Be that as it may, human life, as we ordinarily estimate it, is, in the vast majority of instances, subject to the decaying and thanatizing influence of avoidable disease. Men rarely die of old age. They die (sometimes in old age) from violence, from the immediate effects of acute disease, and from the long-drawn influences of lesions induced by survived attacks, and from slowly advancing effects of poorly appreciated but almost ubiquitous and unnecessary faults of environment. One of these last, the bacteriologist Metchnikoff seeks in the putrefactive changes incident to the retention of putrescible waste in our large bowel, a part of the alimentary canal he traces in evolution by fancied necessities of our progenitors; and his proposition to prevent these changes by the introduction of antagonistic and apparently less harmful lactic-acid producing microörganisms is at least logical, even if as yet unproved beyond a suggestive degree. Taken merely as a part of a general truth, perhaps a very important part, it is not likely that serious denial of the author's ground will be urged; and none can more earnestly look forward, if the remedy succeed, to the measurable prolongation of efficient old age than practising physicians, whose bane of practice is in the effects of constipation and intestinal autointoxication.

In orthobiosis, in Metchnikoff's view, in orderly sequence of the life cycle not too seriously disturbed by adventitious factors, man

lives an ignorant and happy childhood, a violent and reckless youth, a sentient, appreciative manhood, and a deliberate, contented, and judicial old age—a childhood ignorant of death and its meaning, a youth reckless of death and the loss it entails, a manhood first sensing the real meaning of life and conservative of its possibilities, and an old age filled with the measure of accomplishment, wise to the faults which have harmed, and satisfied to relax the hold upon the cup. That pessimism and the embrace of *Weltschmerzen*, denial of life's joy in the overwhelming pain of disappointments, should be the natural portion of the young, of the poet, the artist, and the ill-balanced genius is no wonder to Metchnikoff, who sees in these subjects an absence of the sense of orientation, and an inability to acquire perfect adjustment because of the clashing of their overweening ambitions and ill-judged activities; and he is no admirer of the theory making for elimination of age for the advantage of the world in the strenuous life of the young. He has seen evidence, in the interim between the issue of *The Nature of Man* and that of the present volume, of the fate that would befall us were this the universal acceptance; and his philosophy promises in the orthobiotic cycle the conservation of energy from misapplication and its judicious apportionment to insure the greatest happiness to the individual and greatest advance to the state. It is almost a pity he does not extend his idea of toxic production of old age also to the explanation of no little proportion of the cases of pessimism he deems a not abnormal state of youth; calomel sweetens sour dispositions like a charm in many cases, has lifted a world of woes from melancholic poets, and has doubtless averted many a suicidal act.

Metchnikoff may be very correct in what he has written (the reviewer confesses his acceptance of much of the volume), and indeed, does not fasten himself to intestinal absorption as the sole source of our precocious age and death. He specifically insists in his discussion of a normal old age that there should have preceded it a normal life; it is this which is demanded in orthobiosis. Alcoholism and syphilis he urges as potent faults which hasten our end, and avoidable deteriorants are, of course, to be accepted as always to be eliminated if the normal end is to be expected. There might well have been written a chapter on the avoidable infections, which we treat too lightly from this point of view. An attack of scarlatina in childhood, apparently recovered from and not appreciated in the ordinary course of life, has, however, often left its sequel of potential failure under succeeding stress, and may well in after years, under influences which otherwise would be easily withstood, determine the development of a fatal nephritis. The summation of effects like these, each individual factor in itself perhaps trivial, may, with whatever care the man exert, determine the wearing out which comes in a seemingly inexplicable way. From this view, all infections should be avoided; and the total price for a group of post-

infectional immunities may involve the existence which their protection is relied upon to save. For the matter of that, what does normal senility and normal death amount to? Age and decay are with us from our prenatal period to the end, and much of our body is in continuous loss and replacement. It tallies with Metchnikoff's general view, but widens the view to the utmost, to think of the influences of cell upon cell in the cellular community we speak of as the human body and the harmonic equations which determine or permit the wasting and disappearance of the unuseful.

The natural death contemplated by Metchnikoff really does not occur, and theoretically has no reason for occurrence. If there were such a possibility as absolute orthobiosis there could be no materialistic reason for death. Yet natural death of cells does occur; the regular disappearance of the unuseful by atrophy cannot be doubted as commonly a natural process. It is here and in his inability to cope, whatever his suggestions, with the variations of life-limit seen among different species, that Metchnikoff virtually yields the incompleteness of materialistic views to explain the essence of life and practically acknowledges the predestination he distinctly denies in his pages. The book, however, was not written to defend the monistic theory of life, and it has well fulfilled its purpose of pointing out some material limitations to life; and it would be unfair and at the same time Quixotic to criticise it from this point of view. Yet, page for page, the essays on morality and the social life of man would grow in the optimism the author avows were there but the certain feeling that beyond it all there is a hope without justifiable denial by man that death, in whatever form it come, is not the end, but simply a stage in the evolution of an entity which manifests itself in our imperfect bodies in our present state, but which is not of the earth. A. J. S.

A Text-book of Human Physiology. Theoretical and Practical. By George V. N. Dearborn, A.M., Ph.D., M.D., Professor of Physiology in the Medical and Dental Schools of Tufts College, Boston. Pp. 550; 309 illustrations. Philadelphia and New York: Lea and Febiger, 1908.

Dr. Dearborn has prepared his *Text-book of Human Physiology*, as he states, primarily for medical and dental practitioners and students. In many respects the book follows conventional lines; that is, it comprises thirteen chapters—devoted to protoplasm and the cell, the nervous system, respiration, foods, digestion, nutrition, the blood and the lymph, the circulation, the skin, the senses, muscular action, mental function, and reproduction and development.

But the book is by no means a conventional volume; it contains much that is original, as well as novel—at least in presentation, and almost every page reflects the personality of the author, the enthusiasm of the successful teacher. As the author states in the preface, the book emphasizes the mechanism of the sense-organs, nerves, and muscles as the basis of the individual's efficiency, and it is the first text-book of medical physiology to recognize the more and more insistent demands of the mental process. The chapter on the mental functions is quite unusual in scope and manner of presentation, and well repays careful perusal; it should be read with much interest and profit by students and teachers of psychology and physical education. Special interest attaches also to the chapters on foods, digestion, and nutrition. Dietetic fads receive a well-merited share of condemnation, and emphasis is laid upon the importance of cookery—which is described as the art of preparing food for the pleasurable nourishment of man and other animals, and which is said, as much as anything else, biologically to differentiate man from his "poor relations," the brutes. The physiology of the circulation is well described, aside from the fact that the discussion of the myogenic, as contrasted with the neurogenic, theory of the cardiac activity is inadequate and altogether disappointing. Another of the noteworthy features of the book is the use of many original diagrams to illustrate divers physiological processes, such as cell metabolism, digestive processes, intestinal movements, the functions of the liver, glandular activity, urinary secretion, the functions of the blood capillaries, of the lymph capillaries, and of the skin, kinesthetic nerve impulses, muscular action, etc.; these, together with an appropriate explanation by the teacher, should materially assist the student easily to acquire a knowledge of physiological problems. There is an appendix, of seventy pages, containing directions for performing certain fundamental physiological experiments, with brief theoretical notes; a list of topics suitable for essays and conference discussion; and conversion tables of various sorts—all of which render the book valuable as a laboratory manual.

The book may be cordially recommended: it is of moderate size; it is original; it contains the essential facts of physiology set forth clearly, concisely, and perhaps somewhat dogmatically (which in a book of its kind is by no means a defect); it accentuates matters of prime importance, those of secondary rank being merely mentioned or perhaps omitted; and it frequently contrasts physiological phenomena with related aspects of practical medicine. A. K.

An Index of Treatment. By Various Writers. Edited by Robert Hutchison, M.D., F.R.C.P., Physician to the London Hospital, and H. Stansfield Collier, F.R.C.S., Surgeon to St. Mary's Hospital, London. Revised to conform with American Usage by Warren Coleman, M.D., Professor of Clinical Medicine in the Cornell University Medical College, New York. Pp. 888. New York: William Wood and Co., 1908.

The Treatment of Internal Diseases. By Norbert Ortner, M.D., of the University of Vienna. Edited by Nathaniel Bowditch Potter, M.D., Instructor in Medicine in Columbia University, New York. Translated from the Fourth German edition by Frederick H. Bartlett, M.D. Pp. 658. Philadelphia and London: J. B. Lippincott Co., 1908.

The Treatment of Disease. By Reynold Webb Wilcox, M.D., Professor of Medicine in the New York Postgraduate Medical School. Second edition. Pp. 932. Philadelphia: P. Blakiston's Son and Co., 1908.

The existence of what is sometimes referred to as a revival of interest in therapeutics is well attested by the recent publication of several books devoted more or less exclusively to the subject; of these, the three at present under notice may be considered representative. The *Index of Treatment*, the combined work of some seventy contributors, reflects the advanced teaching and practice of the best English schools; and it has been adapted to the requirements of practitioners in this country by the association of a capable American editor. The majority of the contributors are well known and well qualified to write on the topics that have been assigned them; some even have attained to international reputation. Designed for the practitioner, the treatment discussed is largely medical, but surgical measures have not been entirely ignored; there is, for instance, some good, albeit conservative, advice in regard to the surgical treatment of borderland diseases, such as appendicitis, cholelithiasis, gastric ulcer, etc., although little is said regarding the aberrant phenomena of gallstones or ulcer of the stomach. The work as a whole, however, is of undoubted value, since four editions have been issued in England within a year.

Ortner's *Treatment of Internal Diseases* has achieved a notable success among the German-speaking peoples. In general it reflects the best therapeutic practice of Germany and Austria, although it is quite personal in its tone. There is some mention of pathological physiology as it bears on treatment; and in addition to drug therapy, emphasis is laid upon the importance of mechanical, dietetic, climatic, hydrotherapeutic, and other extramedicinal methods. The value of the book and its adaptability to English-speaking practitioners has been much enchanced by the careful and judicious work of the American editor.

The nature and scope of Wilcox's *Treatment of Disease* is best represented in its subsidiary title, a *Manual of Practical Medicine;* it is, in fact, a manual of medicine in which an unusual share of attention has been devoted to methods of treatment. Many readers will note with ill-disguised concern a certain fondness for divers proprietary remedies; and here and there there is some lapse from the approved practice of many leading American physicians: thus, in the treatment of pneumonia, although creosote carbonate is recommended as a means of limiting the infection, and oxygen is said to be of value, there is no mention of the importance of fresh air (aside from advising a well-ventilated room to be kept at a temperature of 65° to 70°). The value of fresh air is commented upon by the American editor of Ortner's book, but no mention of it occurs in the *Index of Treatment.* In general, however, one may say that Wilcox's book is eminently good, practical, and to the point. A. K.

Medical Gynecology. By Samuel Wyllis Bandler, M.D., Adjunct Professor of Diseases of Women in the New York Postgraduate School and Hospital. Pp. 676; 135 illustrations. Philadelphia and London: W. B. Saunders Company, 1908.

The advances of the operative possibilities in the treatment of the diseases of women during the last two decades have created an undoubted tendency to belittle the importance of many conditions which either are not indications for operative intervention, or which may be quite as well treated by non-operative means. It is, therefore, a matter of congratulation that books of this nature are making their appearance. It of course goes without saying that many cases which demand operation are even now treated without operation either through the ignorance of the medical man or from an even less excusable cause; this book will aid in instructing ignorance; it cannot be expected to teach morality also. The present volume opens with a good chapter of forty-seven pages upon the gynecological examination. As our only criticism of this portion, we would suggest that it is the experience of most of us that one finger is better than two, as a rule, in vaginal or bimanual examinations. We are very glad to find that the cystoscope perfected by Tilden Brown is figured and advocated, as it will render the examination much simpler to the ordinary operator than some of the models recommended in recent books. Next in order is a section of a little less than 100 pages devoted to the methods employed in medical treatment. Under this heading are included, among others, the indications for the use of electricity, pelvic massage, pessaries, and intra-uterine therapy. With regard to the latter,

we are glad to note that while directions are given for its performance, its dangers are strongly enforced. We wish that the half-hearted justification of the use of the sound or stem pessary, as a diagnostic measure in the determination of cervical stenosis as a cause of dysmenorrhea, had been instead a hearty condemnation. The remainder of the volume is taken up by the consideration of the various pathological conditions included within the domain of gynecology. The directions regarding treatment are definite and the symptomatology is clear. We are glad to note that recourse to operative relief is always advised when conditions demand it. In the consideration of the treatment of inevitable abortion, we are sorry to see that the old method of dilatation of the cervix by gauze packs is still given a place, as it certainly seems to us that anyone who can be trusted to pack a uterus, and, if need be, to repeat that packing some hours later, and then remove the product of conception by the forceps or finger, might certainly be considered competent to use the steel dilators, thus avoiding septic infection without, to our mind, any increased risk from instrumentation over the method referred to. We particularly appreciate his treatment of sterility, because of the general fairness of presentation and definiteness in directions; particularly to be praised are his negations as to curettage in this condition.

There is a very satisfactory chapter upon gonorrhœa, special attention being called to the frequency of gonorrhœa without clinical signs. While the author gives the various procedures sanctioned by different authorities for the treatment of intra-uterine Neisserian infection, we are glad to note his own personal avoidance of these manipulations, and also his definite warnings with regard to them. In discussing the question of retrodisplacements of the uterus, the author states his belief that an uncomplicated case of retrodisplacement does not in itself cause symptoms, but that they are due to other associated conditions, standing in no direct relation to the retroflexion. We are especially glad to note his favorable opinion of the pessary and ergot after delivery in cases of displacement. In the chapter following upon uterine subinvolution the author gives directions as to the care of the puerperal patient, which, although possibly a little more radical than we would ourselves favor, are, nevertheless, a step in the right direction; we have long favored the early change of position from the supine to the lateral and prone, but have never been able to convince ourselves that the use of the commode as early as the fifth day is entirely free from danger.

The book can be most heartily recommended to the profession. One of its most praiseworthy characteristics is the conservative spirit which pervades it; this, as may be inferred from the opening paragraph of this review, is, in our opinion, a matter for congratulation.

W. R. N.

ESTIMATION OF THE RENAL FUNCTION IN URINARY SURGERY. By J. W. THOMSON WALKER, M.B., C.M. (EDIN.), F.R.C.S., Hunterian Professor of Surgery and Pathology, Royal College of Surgeons of England (1907). Pp. 295; 75 illustrations. London and New York: Cassell & Co., Ltd., 1908.

DR. WALKER'S book comprises a clinical study of the means of estimating the renal function in surgical diseases of the urinary organs. Part I is concerned with the estimation of the total renal function, and Part II with that of the function of one kidney. Attention is given to the older methods of renal analysis, such as the chemical and microscopic study of the urine; in addition, the significance of cryoscopy, of the electrical resistance of the urine and the blood, of the surface tension of the urine, and of the elimination of certain chemical substances by the kidneys are discussed in detail and judicially. The methylene blue test is believed to be more trustworthy than the quantitative examination of the urea or the appearance of general symptoms of renal inadequancy, especially in the more severe grades of kidney disease. The phloridzin test also is considered to be of value, since a diminished phloridzin glycosuria is said to indicate a depressed renal function which is usually due to disease of the kidney, and a complete absence of sugar should be regarded as a sign of advanced renal lesion. The technique of these and other tests, as well as of divers surgical procedures resorted to in investigating the urinary organs, is fully described. The value of the book is much enhanced by the incorporation of many illustrative cases that form the basis of the conclusions deduced. The book should prove of much value, not only to surgeons, for whom it is intended primarily, but also to general practitioners. A. K.

L'APHASIE DE BROCA. By DR. FRANÇOIS MOUTIER. From the Laboratory of Prof. Pierre Marie (Hospice de Bicêtre). Pp. 769; 175 illustrations. Paris: G. Steinheil, 1908.

THE author, a student of Prof. Marie, in an excellent presentation of 769 pages, again brings up the discussion of aphasia. He begins with a short introduction of the causes of the controversy, and in the first part of the book gives an excellent historical resume of the subject. In the second part is taken up the cortical localization of Broca's aphasia. The author attempts to show that the old or classical view of Broca's aphasia is based upon a fallacy. In a resume of the literature up to the present time he comes to the conclusion that there is no evidence that Broca's aphasia results from isolated disturbances of the third frontal convolution. He quotes

84 negative cases in which Broca's aphasia was present in 57, with no lesion in the third frontal convolution, and 27 cases in which there was no aphasia and yet there was a lesion of the third frontal convolution. In the third division the nature and clinical symptoms of Broca's aphasia are discussed. He reiterates the view advanced by Marie that there is no such thing as motor or sensory aphasia, but that aphasia is a disturbance of the intelligence generally, and principally of that concerned with speech. By the first he means a disturbance in attention, memory, and mimiery; and by the second, a disturbance of understanding and of speech, as illustrated in writing and reading. He then discusses the mental disturbances occurring in aphasia, and concerns himself with word pictures of motor, acoustic, and visual origin, to which he is antagonistic. In the last part of the book there is presented a study of 44 cases of aphasia. In 24 of these the brain was examined, and in one-half serial sections were made. It would be impossible in a review of this kind to give the results of this excellent work, but it might be noted that there were 3 cases with disturbance of the third frontal convolution without aphasia, 4 cases with anarthria, or Broca's aphasia, without a lesion of the third frontal, while in 8 there was anarthria in 1, with a lesion of the third frontal and Marie's zone. Of course, the object of this work is to further Marie's views, and it is well done. At the same time, it must be remembered that throughout there is an evident attempt to justify these views without, perhaps, critical attention being paid to the opposing view. On the whole, the work is well done, and should be read by everyone interested in the question of aphasia. T. H. W.

A Manual of Obstetrical Technique as Applied to Private Practice: with a Chapter on Abortion, Premature Labor, and Curettage. By Joseph Brown Cooke, M.D., Adjunct Professor of Obstetrics in the New York Polyclinic Medical School and Hospital. Sixth edition. Pp. 254. Philadelphia and London: J. B. Lippincott Co., 1908.

The author's idea in preparing this little book has been to give detailed instruction to the younger members of the profession, in their early work among the poor, in regard to the best methods by which they may approximate the hospital technique of the present day. He undoubtedly takes the only tenable ground when, in his preface to an earlier edition, he inveighs against the inferior work too often excused upon the ground of an inadequate fee. The instructions given are definite, the whole subject is satisfactorily covered, and there are but few points with which one may feel inclined to differ. It is, for instance, hardly sufficient to examine the urine

once a month up to the eighth month, as suggested by the author. We cannot see the value of any attempt at urea estimation among the poorer class, since any attempt to estimate the intake of the nitrogen would be worse than useless. Furthermore, the advice that with a conjugate of 8 cm. labor should be induced at the thirty-sixth week and the delivery aided by forceps or version, with the alternative of induction at the thirty-second week and the subsequent use of the incubator, is hardly the attitude of the modern obstetrical specialist. It is true that the book was not prepared for the expert, but since, in the present day, there are men in every town competent to handle major operative obstetrics, there does not seem sufficient reason to advocate such methods. If the beginner were more thoroughly trained in methods of diagnosis, in order that he might know when to summon help, there would be a great improvement in both the mortality and morbidity of childbed. There is no question, in the minds of the majority of obstetrical operators that the use of rubber gloves is an added safeguard in the technique of delivery, and, therefore, it seems that their advantages might have been more definitely insisted upon. It is true they are included in the very complete armamentarium advised by the author, but their use is not insisted upon.

Aside from the above mentioned differences of opinion there is nothing to which exception can be taken, and there are very many most excellent features in the volume. Among the points to which the attention of the younger practitioner may well be directed, is the statement that eclampsia is an absolutely preventable disease provided the patient places herself unreservedly under the care of the physician, and that the "basiotribe is as far beyond the craniocast as the railway train is beyond the stage coach." Both these statements if heeded will tend to decrease the difficulties of practical obstetrics, though we are a little doubtful about the advisability of too easy access to the latter instrument as advocated by the author. We feel that a careful reading of the volume will well repay not only the obstetric tyro, but also many a man of considerable experience.

W. R. N.

Text-book of Diseases of the Nose, Throat, and Ear. By Francis R. Packard, M.D., Professor of Diseases of the Nose and Throat in the Philadelphia Polyclinic College for Graduates in Medicine. Pp. 369; 138 illustrations. Philadelphia and London: J. B. Lippincott Co., 1909.

For the use of students and general practitioners the work before us comprises, it is likely, as fair a summary as can be selected of such diseases of the nose, throat, and ear as are likely to come under the

cognizance and permanent care of the general practitioner. To these are added a few conditions which pertain rather to the domain of the skilled specialist or the unusually skilled general surgeon: mainly the radical procedures for advanced diseases of the accessory sinuses of the nasal passages, and for mastoid operations on the ear. Of the 350 pages devoted to the text, 37 are applied to descriptions and illustrations of the instruments required, and the methods employed for routine examination and treatment, and to remedies and the methods of their application. A scant 100 are devoted to the anatomy and diseases of the ear, malformations of the auricle being very copiously illustrated. Of the 200 odd pages devoted to the anatomy and diseases of the nose, rhinopharynx, pharynx, and larynx, but 20 are occupied with diseases of the larynx.

The teaching throughout is admirable. The illustrations with a few exceptions are excellent, especially the anatomical ones and most of the skiagraphic series. Many of them are new and superior in selection and execution. The language is good and to the point. Students are frequently cautioned against reliance on popular remedies, on the abuse of cocaine, and on the overlust for operative interference. Old established methods of treatment are not discredited. Thus, in discussing remedies for topical applications the long-established value of the too much neglected silver nitrate is fully appreciated; and in discussing minor operative procedures, even the too much despised tonsillotome is advocated in no uncertain terms as the preferable instrument in many cases in which it is often discarded, at least in the hands of the inexperienced operator; and Sajous' snare is appreciatively commended in attacks upon nasal polyps. In describing operative procedures, the accidents possible in execution or in sequence are not ignored; but care is taken to indicate measures for prevention and for control in emergency. Subjects recently brought into additional prominence are discussed, especially diseases of the eye following pathological conditions in the nose and the accessory sinuses. J. S. C.

Surgical Memoirs and Other Essays. By James G. Mumford, M.D., Instructor in Surgery in the Harvard Medical School, Boston. New York; Moffat, Yard & Co., 1908.

Dr. Mumford, whose ability as an interesting writer on medical history has already been proved by his entertaining *Narrative of American Medicine*, has in this volume collected a series of addresses which he has delivered from time to time, together with some of his previously published articles, and out of the whole has evolved a

most agreeable book. The essays on the surgeons of the eighteenth and early nineteenth century are very interesting, particularly those on John Hunter, Sir Astley Cooper, Sir Benjamin Brodie, and John Collins Warren. Although the facts he presents are mostly gathered from the previously published biographies of these worthies, they are retold in such a graphic and pleasing manner that they acquire an original flavor. The essay on "The Teachings of the Old Surgeons" is a really valuable summary of the state of surgical knowledge in the times of various surgeons, from Guy de Chauliac to Baron Larrey. Dr. Mumford possesses the happy faculty of getting at the gist of his subject and dressing it out in a concise and forceful style which compels attention and carries the reader along. He recalls the achievements of the past masters of the art of surgery, and shows us that in surgery, as in all other human pursuits, much that we deem modern was in reality known and taught many years since. This is the kind of book which if placed in the hands of medical students or young practitioners cannot fail to do much good. Its pages are a stimulus to good work, and at the same time cause the reader to see that success can only be achieved in the profession by self-sacrifice and devotion to high ideals.

F. R. P.

The Pathology of the Eye. By J. Herbert Parsons, B.S., D.S.C. (London), F.R.C.S. (England), Assistant Ophthalmic Surgeon to the University College Hospital, London. Volume IV, General Pathology. Part II, pp. 298. New York: G. P. Putnam's Sons. London: Henry Frowde, Hodder & Stoughton, 1908.

This, the concluding volume of Parsons' monumental work, is perhaps the most interesting of the whole to the ordinary reader, being less what may be termed specialized. Its subject matter consists of an exhaustive account of injuries with sympathetic ophthalmia, and panophthalmitis, enophthalmos and exophthalmos, disease of the orbit and symptomatic diseases of the eye; that is, occular diseases dependent upon general affections and diseases of other organs. A short chapter upon heredity closes the work. Each article is richly documented and testifies to a most extensive reading. Upon controverted points Parsons has positive opinions and states them with the reasons therefore after exhaustive statement and criticism of rival views.

Regarding the pathogenesis of sympathetic ophthalmia, after detailing the various hypotheses as classified by Schirmer, he gives the preference to the bacterial metastatic theory, and in this we believe he is in accord with the trend of authority. The advance of

bacteriology during the last few years has weakened the force of the objections which Leber and his followers have urged against it. The intra-ocular signs of general arteriosclerosis so strongly insisted on by Gunn and others are made more intelligible by a number of illustrations. A brief reference is made to the occurrence of Mikulicz's disease; that is, symmetrical enlargement of the lacrimal and salivary glands, but its pathology is not discussed.

Each additional volume as it appeared has strengthened the conviction that this work is to be the standard for years to come upon the pathology of the eye in English. Heretofore the seeker for information upon this subject has been compelled to have recourse to books written in foreign languages, especially German. The entire profession, and not the ophthalmologists alone, will undoubtedly receive with thankfulness a work which so ably and exhaustively puts its reader in possession of the latest views in the rapidly growing field of pathology, which find such striking illustration in the various tissues which enter into the organ of vision. T. B. S.

Cataract Extraction. Being a Series of Papers with Discussion and Comments Read before the Ophthalmological Section of the New York Academy of Medicine, 1907–1908. Edited by J. Herbert Claiborne, M.D., formerly Adjunct Professor of Ophthalmology in the New York Polyclinic. Pp. 169. New York: William Wood & Company, 1908.

This symposium upon cataract extraction by twelve New York ophthalmologists is a fair resume of the present status of the operation. While the opinions vary somewhat regarding details, there is a fair unanimity upon all essential points except upon the question of the simple or combined method. The pendulum is now swinging in the direction of the latter. The 8.61 per cent. of prolapses are the stumbling block in the way of many an operator who would otherwise decide in favor of simple extraction. We confess to some surprise at the statement by some of the participants that the latter is the easier method. Claiborne advocates extraction with a lance-shaped keratome of his devising and also describes a new instrument to scratch the capsule for artificial maturation. The incisions are so fine that the aqueous humor is brought in contact with the lens substance without protrusion of the latter. Lambert contributes a paper upon removal of the lens in high myopia, with a report of two highly successful cases in young women, but does not advocate the practice in all cases of high myopia. The editor justly observes that the operation will probably never be generally employed in this country. Indeed the editor's summing up at the conclusion of each paper is always judicious and interesting. T. B. S.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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Sterile Serous Effusions with Empyema and Lung Abscess.—Königer (*Münch. med. Woch.*, 1909, lvi, 603) mentions the occurrence of multilocular tuberculous pleural effusions, the exudates being walled off and separated from one another. Such a condition is much rarer in acute infections. It is not a very unusual experience in making exploratory puncture to find a thin, clear, serous exudate at one point, a purulent fluid at another. This has usually been attributed to the settling of the cellular elements in the exudate. Studying a number of cases bacteriologically and cytologically, the author has found that the purulent fluid contains microörganisms, while the serous is usually sterile. From observations made on a number of cases, he concludes: (1) That acute pleuritic empyema which becomes encapsulated early, not infrequently leads to extensive serous effusion in the adjacent part of the pleura, which ordinarily does not communicate with the primarily infected area. (2) That such a serous effusion is sterile and is quite rich in cells, the majority of which are polymorphonuclear neutrophiles, with a few lymphocytes, endothelial cells, etc. The cells are very well preserved, few of them showing the degenerative changes seen in the pus cells of the purulent exudate. (3) Bacteriologically and cytologically the findings show that the difference in the fluids at the two points does not result from settling of the cells. The serous effusions probably owe their origin to bacterial toxins and are comparable to the œdema surrounding a phlegmon in the tissues. (4) Similar serous effusions arise in connection with purulent processes in the lung (abscess, gangrene) as well as with other severe suppurations in the neighborhood of the pleura (subphrenic abscess, liver abscess). (5) All the above

mentioned serous exudates have this in common, that they are caused by a localized infection. Their clinical importance lies in the fact that their presence renders much more difficult the recognition of a suppurative process; they are apt to confuse the diagnostician because of their comparatively harmless appearance and their sterility. The cytological examination combined with the bacteriological finding may, however, raise one's suspicion.

The Excretion of Hexamethylenamin (Urotropin) in the Cerebrospinal Fluid, and its Therapeutic Value in Meningitis.—Crowe (*Johns Hopkins Hosp. Bull.*, 1908, xix, 109) first studied the excretion of urotropin in the bile and pancreatic juice in the report a year ago, and he recorded a case of purulent meningitis following a postoperative cerebrospinal fistula which cleared up in three weeks from the administration of urotropin by the mouth. Since then (*Ibid.*, 1909, xx, 102) he has demonstrated that the urotropin which is administered by the mouth in man, rabbits, and dogs invariably appears in the cerebrospinal fluid, a maximum amount in from thirty minutes to one hour after ingestion. In therapeutic doses an amount appears sufficient to exercise a decided inhibitory effect upon the growth of organisms inoculated into the spinal fluid after its removal from the body. Sixty to eighty grains a day in dogs and rabbits will either defer or prevent the onset of a fatal, experimental streptococcic meningitis. In view of these observations he advises its application as a prophylaxis in all clinical cases in which meningitis is a possible or threatened complication, such as fractures of the skull, operations upon and infections of the nasal sinuses and middle ear; even after simple lumbar puncture. Thirty to sixty grains a day may be given.

Toxic Effects from Bismuth Subnitrate.—Beck (*New York Med. Jour.*, 1909, lxxxix, 16) reports seven cases from the literature, in addition to his own experience, with poisoning from bismuth subnitrate. The clinical picture is one of collapse with cyanosis, vomiting, diarrhœa, and abdominal cramps. Böhme has shown that these are symptoms of gastro-enteritis and methemoglobinemia due to acute nitrite poisoning arising from the conversion of the subnitrate into nitrite in the large intestine by bacterial action. Beck points out that bismuth subnitrate is used freely and regarded as harmless, especially by radiographers. In conclusion, he states that small doses by the mouth are harmless. In larger doses sufficient nitrites are liberated and absorbed to produce an acute nitrite poisoning, characterized by methemoglobinemia and collapse. This may terminate fatally. Children and individuals suffering with intestinal putrefaction are very susceptible. This acute nitrite poisoning is a distinct and separate affection from the more chronic bismuth absorption. Beck advises the use of some other salt of bismuth than the nitrate. He finds that iodine neutralizes the nitrite in acute poisoning, whereas alcohol and glycerin hasten its liberation.

The Recognition of Glycuronic Acid in the Urine.—C. Tollens (*Münch. med. Woch.*, 1909, lvi, 652) recognizes the need for a simple test for the recognition of glycuronic acid in the urine. He points out that it is increased after the administration of various drugs, such as phenol,

salicylic acid, chloral hydrate, cresote, camphor, morphine, etc., and this increase may be of assistance in the diagnosis of poisoning with any of these. Furthermore, patients presenting an increase of the paired glycuronic-acid compounds have been looked upon as diabetics and have been refused life insurance, because their urines reduce copper solutions. Again, it has been shown that aromatic substances arising in deep-seated, unrecognized abscesses may become paired with glycuronic acid and thus lead to its increase in the urine. Hoppe-Seyler has demonstrated that excessive intestinal putrefaction may give rise to large amounts of indoxyl-glycuronic acid in the urine. It is evident, therefore, that the recognition of increased quantities of glycuronic acid compounds in the urine may be of aid in a variety of conditions. Of the tests for glycuronic acid in the urine, the orcin test is not reliable, for pentoses also give it. B. Tollens has devised a test which is simple, quite accurate, and easily carried out. It is based on the fact that naphthoresorcin and HCl, when acting on glycuronic acid, give rise to a blue pigment, *soluble* in ether, which produces a well-marked, dark band in the sodium line on spectroscopic examination. The pigments formed in this reaction with pentoses and various sugars are *insoluble* in ether. Method: To 5 c.c. of urine add a bit of naphthoresorcin about the size of a millet seed and 5 c.c. of fuming HCl (sp. gr., 1.190). Heat to boiling over the flame and continue boiling gently for one minute. Now allow the test-tube to stand four minutes and then cool in running water. Add an equal volume of ether, shake vigorously, and set aside till the ether is clear. (The clearing is hastened by the addition of a few drops of alcohol.) If paired glycuronic acid compounds are present in excess, the ether extract is dark blue to violet, while small amounts produce a faint bluish or reddish violet color. Examined spectroscopically, the ethereal extract shows a single dark band near the sodium line. Instead of napthoresorcin in substance, one may employ 0.5 c.c. of a 1 per cent. alcoholic solution. The test, performed as above, is delicate enough to detect the small quantities of glycuronic acid present in normal urine. With large amounts of glycuronic acid it may be necessary to dilute the ether extract before spectroscopic examination. From the intensity of the reaction one may make a rough estimate of the quantity of glycuronic acid present.

Functional Test of the Pancreas.—Gross (*Deut. med. Woch.*, 1909, xxxv, 706) has devised a method for estimating quantitatively the trypsin in the feces. The method is based on the fact that casein rendered soluble in alkali is precipitated on the addition of acetic acid, while the products of its digestion remain in solution. For fecal examination one dissolves 0.5 gm. of caseinum purissimum (Grübler) in one liter of 1 to 1000 soda solution, warming moderately. The feces to be examined are rubbed up in a mortar with three times the quantity of 1 to 1000 solution of soda until a homogeneous suspension is obtained, and then filtered until the filtrate is clear. (Usually this is very quickly accomplished, but if bacteria cause the turbidity, they will settle to the bottom in a short time and the clear fluid may be decanted.) 100 c.c. of the casein solution is placed in a flask and 10 c.c. of the fecal suspension added. The mixture is shaken, placed in an incubator at 38° to 40° C. and from time to time small quantities are withdrawn

and tested with 1 per cent. acetic acid until a precipitate no longer occurs. Employing this technique over 200 stools from patients without demonstrable pancreatic disease were examined, and a proteolytic ferment was found in all. A few drops of chloroform was added to prevent bacterial growth and the probability that the ferment was trypsin was further shown by the lack of digestion after the addition of 0.5 c.c. of human blood serum (antitrypsin). Erepsin was practically excluded by removing the pancreas of dogs. Feces from such animals failed to digest the casein. Of the 200 cases the duration of digestion until complete was eight to thirty hours. The rapidity of digestion and, therefore, the amount of trypsin were found to vary with the variety of food given the patient. Digestion was most rapid after proteid, next after fat, and slowest after carbohydrate food. Therefore, the examination is more satisfactory after proteid nourishment, and here the average time required for complete digestion is twelve to fourteen hours (limits eight to fifteen hours). In various intestinal diseases Gross found normal tryptic digestion, except that in diarrhœa the casein is digested more rapidly. In one patient with complete closure of the ductus choledochus no tryptic digestion was demonstrable. A similar result was obtained in a case of cancer of the pancreas and in this case absence of pancreatic juice was the only definite sign of pancreatic disease. In a patient with cirrhosis of the pancreas and a clinical picture of pernicious anemia normal tryptic digestion occurred.

Thymol as a Source of Error in Heller's Test for Urinary Protein.—Weinberger (*Proc. Soc. Exper. Biol. and Med.*, 1908, vi, 4; *Jour. Amer. Med. Assoc.*, 1909, lii, 1310) considers the sources of error in Heller's nitric acid test for urinary proteins. In urines concentrated beyond a certain degree the test will fail even in the presence of large amounts of albumin. According to Kenny, too much formaldehyde used in preserving the urine will render the test unreliable. In the course of other investigations Weinberger found that thymol also as a preservative was a source of error. If such a urine is floated upon concentrated nitric acid, there forms precisely at the time of junction a grayish white ring 0.5 mm. high, resembling the ring given by a faint trace of albumin. In an excess of urine the cloud will not disappear on shaking. Warming will not prevent the formation of the ring—if anything, will make the reaction more distinct. Dilution of the urine with three or four times its volume of water has no prohibiting effect. It occurs in both acid and alkaline thymolized urines, more pronounced in the latter. In urine containing both albumin and thymol in various amounts each ring may be discerned. The albumin ring is wider and white; whereas, the thymol ring forms underneath it and is gray and thin. However, the albumin ring may be completely covered by the latter. The necessary modification is the removal of the thymol by extraction with an equal volume of petroleum ether. On the other hand, ether will not extract the thymol when it has been given internally and is excreted through the kidneys, a point of some importance.

A New Method for Finding Tubercle Bacilli.—Lange and Nitsche (*Deut. med. Woch.*, 1909, xxxv, 435) have described a new method for the examination of sputum for tubercle bacilli. If a pure culutre of

tubercle bacilli in a watery medium be mixed with a culture of non-acid fast bacilli, the two can be separated almost quantitatively by the use of a hydrocarbon, the tubercle bacilli being found in the latter. This fact has been applied to the examination of sputum, urine, pus, etc. Sputum should be rendered homogeneous. The choice of the hydrocarbon is of some moment. Petroleum ether, benzine, ligroin, toluol, and xylol have proved satisfactory. The best results are obtained, however, with ligroin of a boiling point of 90° to 120° C. It has the advantage of being cheap. The specific gravity of the hydrocarbon should not be too much below 1; otherwise it rises too quickly after shaking. The advantage of the low boiling point is that the reagent quickly evaporates and does not interfere with subsequent fixation of the preparation. There is also less tendency for the drop to "run" when evaporation is rapid. To render the sputum homogeneous the authors recommend normal potassium hydrate as the most satisfactory reagent; 5 c.c. of sputum and 50 c.c. of normal potassium hydrate are well shaken and allowed to stand three hours at a room temperature (shorter in incubator at 37°); repeated shaking hastens the solution; 50 c.c. of tap water and 5 c.c. of ligroin are added and the whole shaken vigorously, preferably in a stoppered graduated cylinder. The separation of the hydrocarbon requires several hours, but warming the emulsion to 65° greatly facilitates this. When the separation is complete, the tubercle bacilli will be found at the bottom of the clear layer of ligroin. Employing this technique the authors have been able to find many tubercle bacilli in sputum which appeared free of them when examined in the usual way. Furthermore, in their preparations there was a striking absence of bacteria.

Iron as a Substitute for Bismuth in Radiography.—Taege (*Münch. med. Woch.*, 1909, lvi, 758) confirms the findings of Lewin that iron may be substituted for bismuth in radiographic work. Two advantages are possessed by the iron: (1) Apparently there is no danger of intoxication, as may happen after using bismuth; and (2) it is much cheaper, costing about one-thirteenth as much as the bismuth. The preparation, which he has found most satisfactory, is red oxydate of iron (*German Pharmacopœia*). Of this Taege himself has taken 50 grams without any perceptible effect.

The "Pulsometer."—Fellner (*Deut. med. Woch.*, 1909, xxxv, 211) has discovered a method for determining the rate of flow of the blood in man, the results of which correspond with those previously determined in animal experiments. An Esmarch bandage is applied to the arm to remove the blood from the capillaries and veins. The cuff of the sphygmomanometer is now applied to the arm at the upper end of the bandage. The pressure is raised 30 to 50 mm. Hg. above the systolic pressure and the bandage then removed. A stop watch which registers 0.2 of a second is now started and simultaneously the pressure on the sphygmomanometer is released. The arm is pale and (before releasing the pressure) pulseless. The fingers are observed, and at the first sign of reddening in them the watch is stopped. The distance is measured and the time is read off on the watch. About 1000 experiments on healthy and diseased persons gave a velocity of 200 to 400 mm. per

second. Fellner has devised an apparatus which he says is especially suited to this procedure.

A Skin Reaction in Carcinoma from the Subcutaneous Injection of Human Red Blood Cells.—Elsberg (*Proc. Soc. Exper. Biol. and Med.*, 1909, vi, 91; *Jour. Amer. Med. Assoc.*, 1909, lii, 1310) attempted to produce a specific hemolytic reaction locally by the injection of red blood cells under the skin of a carcinoma patient. He was led to these experiments by the recent demonstration that hemolysis occurs when the blood serum of a patient suffering from carcinoma is mixed with normal human red blood cells. The technique employed was simple. Blood was obtained aseptically from normal individuals, preferably children, and defibrinated. A 20 per cent. emulsion of the washed red blood corpuscles was used, five minims of this being injected subcutaneously in the anterior surface of the forearm. A "positive reaction" consisted of infiltration, tenderness, and redness at the site of injection, in six to eighteen hours. Simple discoloration or the puncture wound was all that was seen in the negative cases. Thirty-four injections were given to 20 cases of known carcinoma, and every one of the cases had a positive reaction; of four cases with known sarcoma; three gave a positive reaction. The reaction was negative in all but 3 of 100 individuals who were either normal or suffering from such diseases as nephritis, leukemia, syphilis, benign tumors, tuberculosis of the lungs, bladder, and kidneys, and many acute and chronic inflammatory conditions. No reaction was obtained in several patients in whom malignant disease was suspected but not found at operation. An exploratory incision has confirmed the positive reaction in several patients in whom carcinoma was not suspected. The injection of laked bood caused a positive reaction in all patients, whether suffering from malignant disease or not. This seemed to show that the positive reaction in carcinoma from blood cell injection was due to hemolysis. It may prove that this is constant enough to be a characteristic and easily recognized aid to diagnosis.

SURGERY.

UNDER THE CHARGE OF

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A Modification of the Bassini Operation, Especially in Large Herniæ.—Caminiti (*Zentralbl. f. Chir.*, 1909, xxxvi, 244) makes an incision through the skin and external oblique muscle, opening the inguinal canal. The fibers of the cremaster are then separated and the sac of the hernia

exposed and isolated from the surrounding tissues. The separated and, emptied sac is then ligated at its neck and the stump first fixed above or the operation carried further toward its completion. Instead of now taking up together in one bundle, the cord, the common tunica vaginalis, and cremaster, they are separated from one another, and the elements of the cord carefully isolated. Temporarily these are kept together by wrapping them with the cremaster in gauze. If the stump of the cord has not already been drawn upward and fixed in the upper part of the wound this is now done. The internal oblique and transversalis muscles are then sutured to Poupart's ligament. The peripheral edge of the cremaster is removed, so that only its compact portion remains. It is then sutured over the other layers. The cord structures are then spread out over this layer. The external oblique is sutured over the cord and then the skin. The cremaster muscle begins, immediately to functionate and increases the strength of the inguinal region. It still retains its undisturbed relation to its anatomical origin and will undergo no alteration.

Some Disorders of the Cerebral Circulation and Their Clinical Manifestations.—Russell (*Lancet*, 1909, i, 963 and 1031) says that the fundamental factor in the pathology of a fainting fit is a diminution of the volume of blood passing through the brain. Physiologically, two important factors are concerned: (1) Vasodilatation in the splanchnic area; and (2) cardiac inhibition. Probably in some cases only one of these factors is primarily concerned, in others both may be involved. It is difficult to judge as to the degree and frequency with which the heart may be affected independent of the changes in the splanchnic area. Gowers has reported three cases in which repeated syncopal attacks have passed into epilepsy. In all three the attacks were originally brought on by external influences and were typically syncopal. After a lapse of time they appeared spontaneously and with a more sudden onset, in fact, they were undoubtedly epileptic. The explanation of this change which suggests itself is that the increased suddenness of the attacks is due to increase of the rapidity with which the circulatory changes are brought about. Gowers believes that a change in the nerve elements occurs and is due to a sudden diminution of the intracranial pressure in syncope. He suggests that the resulting state of the nerve elements from repeated induction becomes spontaneous, constituting minor epilepsy. A number of cases have been reported in which it has been established beyond question that arrest of the heart preceded the epileptiform attacks. Langendorff made observations on the results of stimulation of the peripheral end of the vagus in rabbits narcotized with chloral. Electrical stimulation caused a prolonged stoppage of the heart with a complete epileptic convulsion, ten or fifteen seconds after the diastolic pause. Precisely similar convulsions are produced by compression of the thoracic aorta at a point nearer the heart than where the great vessels to the head are given off. The circulatory failure offers the most reasonable explanation for the various manifestations observed in an epileptic attack. Leonard Hill produced clonic convulsions in himself by compression of one carotid artery. Epileptic convulsions, petit mal, and infantile convulsions are probably to be explained on the basis of cerebral anemia. The phenomena of Jack-

sonian fits dependent on tumor might be due to spreading vasoconstriction.

Postoperative Pulmonary Embolism—Lenormant (*Archiv. gén. de chir.*, 1909, iii, 221) quotes Selberg, who, upon postmortem evidence, determined that half of the deaths after aseptic laparotomies are due to pulmonary lesions. Von Lichtenberg collected statistics on 23,600 operations, of which 16,000 were laparotomies; and found that the pulmonary complications reached 2 per cent. for all operations and 5.5 per cent. for laparotomies alone. Kelting classified these cases according to the path of the pulmonary infection, into the bronchial (aspiratory pneumonias), and infection through the lymphatics (through the diaphragm from operations in the upper abdomen, especially on the stomach). Von Lichtenberg classified them into four groups: pneumonias consecuti.e to narcosis; hypostatic pneumonias; infectious pneumonias; and the embolic complications. Lenormant would add to this classification a fifth group, the pneumonias of deglutition which occur in consequence of operations on the upper digestive and respiratory tracts and in patients who vomit (intestinal occlusion). Lenormant reports briefly 4 cases of postoperative embolism, occurring in 792 different operations (a proportion of 0.5 per cent.). Three of the 4 occurred after abdominal operations. They are much less frequent after operations on other parts of the body, as the head, neck, thorax, and extremities. The danger of pulmonary embolism from laparotomy, seems to be greater the further the operative zone is from the diaphragm. Pulmonary complications are, it is true, very frequent after gastric operations, but these are pneumonias or pulmonary congestions, and emboli play in these cases, only an exceptional and insignificant role. Sometimes the emboli take their origin from the veins of the operative region, as the femoral and external iliac, especially on the left side. Lenormant believes that the latter result from an infection. As soon as a thrombus develops the danger of an embolus breaking off and being carried away in the blood stream is present. The pulmonary lesions consecutive to a pulmonary embolus vary. Massive emboli of the pulmonary trunk kill in a few minutes, by syncope. Medium and small emboli produce the classic hemorrhagic infarcts. Secondary infection of the infarct is frequent. Sometimes the infection comes from the embolus itself, sometimes from the bronchus. Odinarily the infection remains moderate and is accompanied by an embolic pneumonia. Abscess or gangrene may also result. The prognosis is extremely grave. In a series of 233 cases collected from the literature, Lenormant found 106 deaths, 45.5 per cent. The prognosis is probably less dangerous than this would indicate, since many of the milder cases are overlooked. The chief therapeutic indication is preventive. Patients with weak hearts should receive beforehand proper medical treatment. Ranzi advised preliminary injection of scopolamine-morphine, to reduce the quantity of ether necessary, while Witzel employs in such cases, ether exclusively, because it has not the depressing effect of chloroform on the heart. Strict asepsis, careful handling of the bloodvessels, especially of the veins, in the dissection of large tumors, are necessary. When the signs of pulmonary embolism develop, the usual measures to stimulate the heart and avoid asphyxia should be adopted. The operation

of Trendelenburg upon the pulmonary artery for the direct extraction of the clot can be justified only in the most grave cases in which death is imminent.

Concerning the Healing of Chronic (Tuberculous) Empyema by Means of Artificial Pneumothorax.—Wenkenbach (*Mit. a. d. Grenz. de Med. u. Chir.*, 1909, xc, 842) says that little is to be expected from the internal treatment of tuberculous empyema. The Estlander, Schede, and Delorme methods of treatment are always desperate, and when successful always leave the patient a cripple, usually an invalid. In a few cases Wenkenbach employed a method which he regards as simple and safe. In the first case several aspirations of the pus had been done, and during the last one the patient complained of oppression and pain in the chest, so that the aspiration was terminated. Through the stopcock and rubber tubing attached to the trochar, air was permitted to enter the chest, and then the stopcock was closed. The patient breathed more freely and the oppression and pain were relieved. Ten days later the aspiration of pus and the admission of air were repeated. Similar improvement followed. The treatment was repeated, at first three, and later four and six weeks apart, with complete healing finally. The patient still suffered from tuberculosis of the other lung, and gradually grew worse. In a second case, also severe, a cure was obtained. This treatment can be carried out by every general practitioner. No complicated apparatus is necessary. It is not necessary to introduce oxygen. Air is enough. The extraction of the often very voluminous exudate greatly relieves the patient. By the introduction of air the whole exudate can be removed entirely without danger. The general condition of the patient improves immediately, and the quality of the exudate is favorably changed. The lungs gradually approach the chest wall, so that the strong traction on it is avoided. The condition of the diseased side of the thorax can subsequently be improved by mechanical treatment. After a few months the patient can recover in great part or completely his ability to work.

Concerning Suture of Stab Wounds of the Lung.—Stuckey (*Archiv f. klin. Chir.*, 1909, lxxxviii, 767) says that in recent years most surgeons have contended for radical procedures in stab wounds of the lungs. Their chief reasons for this treatment were to be found in the loss of blood, increasing pneumothorax, and progressive subcutaneous emphysema. In earlier times the fear of a general pneumothorax led surgeons to assmue a waiting position. Stuckey will follow the expectant line of treatment if thirteen hours have passed since the wound was received, unless anemia or a tense pneumothorax is present, when a radical operation will be necessary. When a stab wound is found to lead into the pleural cavity, the edges of the pleural wound should be drawn aside with sharp hooks, in order that one may obtain a view of the cavity of the pleura, and to learn whether or not there is any blood in it. If little or no blood is found, the object has been attained. The edges of the wound in the pleura and muscles should be closed with a few silk sutures. With an hermetically closed pleura, the pneumothorax will be more quickly absorbed. If there is discovered in the pleural cavity a considerable amount of blood, or there is suspected a wound of the

heart or diaphragm, a rib resection is necessary. Recent stab wounds in themselves call for operative intervention. Wounds of the lung should be closed by suture, and the suture should include the floor of the wound and not be too tightly tied. When the suture cannot be introduced and caught in the tissues, the wound should be packed with gauze and the edges of the wound sutured to the margins of the pleural opening, so that it may heal by granulation. The pleural cavity is to be completely closed by the suture, so that the resulting pneumothorax shall not give way.

A Contribution to the Surgical Treatment of Cancer of the Lip.—Steiner (*Deut. Zeit. f. klin. Chir.*, 1909, xcvii, 243) says that cancer of the lip is ten times more frequent in males than in females. Cancer of the lower lip is twelve times more frequent than of the upper lip. On the lower lip there is a predilection of the cancer to attack either the right or the left half, while in the upper lip it is generally found in the middle. Among farmers and, more particularly, smokers, cancer of the lip is especially frequent. It occurs most frequently between the ages of fifty-five and sixty years. Preceding inflammation, psoriasis, leukoplakia, and scars are exciting causes of their development. 84 per cent. of the recurrent and 71.7 per cent. of the non-recurrent cases were admitted within a year of the stated appearance of the growth; 55 per cent. of the primary cancers were admitted within the first year; in 76 per cent. the cancer had already ulcerated, and in 67 per cent. a regional gland infiltration was present; 70.7 per cent. of the cases operated on for primary cancer were free of recurrence more than three years after operation, and 69.6 per cent. after more than five years. In two-thirds of the recurrent cases death occurred in the first year; 10 per cent. of the cases operated on for recurrence remained free of recurrence more than three years. Three-fourths of the cases operated on for recurrence died in the first year.

The Treatment of Acute Inflammations with Bier's Passive Hyperemia.—Lossen (*Deut. Zeit. f. Chir.*, 1909, xcvii, 259) says that Bier's passive hyperemia is a valuable physical aid in the treatment of tuberculous, gonorrhœal, and acute suppurative inflammations. It is indicated, and without incision, in pure fungus, without inclination to softening, in all gonorrhœal inflammations, and in all beginning acute inflammation. It is further indicated in acute inflammations, however, only when there results a relief of inflammatory pressure from an incision or a fistula, and when for a cosmetic or functional purpose a large incision is to be avoided. It is still further indicated in those cases in which, in spite of a wide incision, the granulating wound in unhealthy and the sepsis has not diminished. Passive hyperemia is particularly effective in suppurative periproctitis, para-urethral abscesses, furuncles and carbuncles of the head and neck, suppuration of the joints and tendon sheaths, phlegmonous bursitis with fistulæ, biliary fistulæ, mastitis, softened axillary bubo, and especially in inflammations with advanced softening. It is not effective in infiltrated and fibrous inflammatory tissue (as in many streptococcic phlegmons), in glands without complete softening, lymphadenitis and lymphangitis, in many cases of bone infection, erysipelas, and diabetic phlegmons. This treatment

calls for close attention in order that the presence of retention of pus may be recognized promptly, that excessive œdema may be reduced by other physical methods, and beginning adhesions retarded.

Inguinal Hernia of the Uterus.—Parker (*Brit. Med Jour.*, 1909, i, 947) reports the case of a woman, aged forty-seven years, on whom he did a double herniotomy. During a fortnight previous to operation, symptoms of intestinal obstruction had occurred, lasting three days. The right hernia, which alone had been troublesome, was found at operation to contain an ovary, Fallopian tube, broad ligament, and small, elongated uterus, all enclosed in a peritoneal sac. There was no means of reducing the contents, which were accordingly removed. In doing this a careful search was made for a portion or trace of the vagina, but none was found either then or after dissection of the specimen. But the finger was passed into the vagina at this stage of the operation, without obstacle and without any discovery of note. Both wounds were then closed, and they healed by first intention. Much pain and inability to pass wind, with slight but increasing distention, followed the operation. On the fifth day a median abdominal section was performed. Attached to and blocking the sigmoid flexure was found a hard lump which was taken for a small carcinoma. A loop of bowel with the lump superficial was sewn closely to the peritoneum, and its contiguous sides attached to each other in the middle of the opening. An artificial anus was then made. A careful search was made in the pelvis for the left ovary and appendages, and for the left half of the supposed uterus bicornis, but nothing whatever could be found of such parts. On attempting a vaginal examination the finger could now be passed no more than an inch or so, the vagina being tightly closed beyond this point. The symptoms of bowel obstruction disappeared, but the severe pain continued, requiring the use of morphine hypodermically. Exactly two months after the colostomy was performed a third operation was undertaken, with the view of removing the growth on the colon, which had receded into the wound. After clearing out the rectum by enemas from the fistula and rectum, the finger which passed in both directions from the fistula revealed no tumor. A clamp was applied to the spur and was gradually cut through in a little over a week, when feces were found to be passing the anus, though most passed by the fistula. A fourth operation was done to close the fistula. This did not succeed, but later the fistula gradually closed and the patient became perfectly well.

Tetanus Occurring after Surgical Operations.—Richardson (*Brit. Med. Jour.*, 1909, i, 948) reports two cases in which the symptoms of tetanus developed after operation, one for gallstones, the other for a strangulated, inguinal, omental hernia. Both patients died. Richardson collected notes of 21 cases in which tetanus followed operation. Twenty of the cases have occurred within the past three and one-half years, and one as long ago as 1884. Hamilton has pointed out that there is a group of diseases among sheep, the symptoms of which are closely allied to tetanus and the bacillus of which cannot be distinguished from that of tetanus. These diseases are endemic in certain parts of the British Isles. The 21 cases have occurred only in those districts in

which the tetanic group of sheep diseases is endemic. More than 90 per cent. of the cases of postoperative tetanus followed operations in which the peritoneal cavity had been opened. Richardson suggests that the disease which we call postoperative tetanus is not tetanus at all, but one of the sheep diseases; that it is not introduced by the catgut; that the patient is at the time of the operation the host of the bacillus; and that we must look upon these as cases of idiopathic tetanus, accepting Hamilton's suggestion that idiopathic tetanus is not true tetanus as we understand the term. The most important point is to determine whether the infection, whatever it may be, is conveyed by the catgut. Richardson hopes that the catgut may be acquitted.

Concerning Excrescences of Fatty Synovial Fringes in the Knee-joint.—Rammstedt (*Archiv f. klin. Chir.*, 1909, lxxxix, 173) says that no joint in the body is subject to so many injurious influences as the knee. While much attention has been given in the literature, recently, to many of these conditions, relatively little has been given to the inflammatory overgrowth of the fatty masses and fringes in the joint. Hoffa, who first called attention to them, on opening a joint for a supposed injury to the semilunar cartilages, found this condition, and upon excising the fringes a cure followed. They spring from the ligamenta alaria and mucosa, increase in size from inflammatory hyperplasia, and because of their increased size become caught between the ends of the bone, producing severe symptoms. The causative synovitis may be due to trauma or disease. The catching of the fringes between the ends of the bones may be confused with a dislocation of a semilunar cartilage, a free joint body, or a tearing of a crucial ligament. In the latter group of conditions the severe symptoms are followed by synovitis and effusion into the joint. In the case of catching of the fatty fringes, almost always a chronic swelling of the joint, and especially of the fatty masses at the sides of the ligamentum patellæ, precedes the occurrence of the severe symptoms. One should bear in mind that isolated lipomas and sarcomas have been reported. The lipomas may be of various sizes, from that of a hazel-nut to that of a walnut, and may give symptoms simulating those produced by the catching of the fringes and may also be preceded by trauma. The treatment of cases in which the fringes are caught may be conservative if they give only moderate disturbance. In most cases, improvement and a gradual return to the normal follows the use of massage, heat in the form of sand baths or hot air, elastic compression by bandages, and guarded use of the joint with apparatus. Patience is especially necessary. Operation is justified in the more severe cases with limitation of movement and severe pain associated with locking of the joint. The joint should be opened freely by a curved incision at the outer or inner side over the prominence. The hypertrophic masses are then seized with forceps and excised with suitable scissors. The hemorrhage is slight and can usually be stopped by a gauze pad, which may be left in position until the capsule wound is about to be closed. Drainage is not necessary. If an effusion of bloody serum should form, this may be removed by puncture on the first change of the dressing. In from twelve to fourteen days after the healing of the wound, massage, hot air, movements of the joint, and compression bandages should be employed.

THERAPEUTICS.

UNDER THE CHARGE OF

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The Therapy of Diabetes Mellitus.—Falta (*Archiv. Int. Med.*, 1909, ii, 159) calls attention to the fact that human diabetes mellitus is a highly complicated disease often of widely different etiology. He thinks that the diabetic disturbance of metabolism may be broadly defined as a lack of equilibrium between carbohydrate mobilization and carbohydrate metabolism, arising from deficiency of the pancreas or from overactivity of the chromaffin system, or from both causes together. Accordingly, a causal therapy is out of the question. Attempts have been made to increase the efficiency of carbohydrate metabolism by increasing or supplying the internal secretion of the pancreas, but as yet Falta says that the results are unsatisfactory and impracticable. A diminution of the excessive carbohydrate mobilization might be brought about by checking the activity of the nervous system. Falta thinks that some of the favorable results obtained by the use of sedatives may be thus explained. The theoretical principle of dietetic therapy is to diminish the demands upon the diseased organ or organs and give them thereby the opportunity of recovering.

Falta first considers a small group of cases which manifest a marked disease of the pancreas. In these cases protein and fat are imperfectly absorbed, as evidenced by their presence in increased amounts in the stools, and in addition carbohydrates are excreted, as evidenced by the glycosuria. It is essential, Falta says, to replace the pancreatic secretion in the treatment of these cases. For this purpose the most useful preparation is the pancreatin of the Rhenania factory in doses of 10 grams daily. Alkalies must be used in conjunction with the pancreatin. Von Noorden prefers the use of calcium carbonate, which is less readily absorbed from the stomach than sodium bicarbonate, for this purpose. Emulsified fat (as milk or the fat of egg yolk) is much better utilized by these patients than other fat preparations.

Falta points out that the measures by which glycosuria may be diminished may have to be considerably modified in some of the acute and chronic infectious processes, which frequently complicate diabetes. Thus, for instance, a strict diabetic regimen may be harmful in a complicating pulmonary tuberculosis. However, in the milder forms of pulmonary tuberculosis complicating diabetes, Falta has noted an improvement with the disappearance of glycosuria after appropriate dietetic treatment. He maintains that each individual case has its own peculiar characteristics, and therefore the glycosuria must be combated only after a careful preliminary estimate of the character and intensity of the disease. He makes use of a test diet over a period of three days. This test diet consists of 250 grams of meat, 150 grams of butter, 4 eggs, 300 grams of vegetables with a low carbohydrate value, tea, coffee, bouillon, about

4 deciliters of light white wine, and 75 grams of white bread divided into three equal portions. This diet contains, approximately, 16 grams of nitrogen, 50 grams of carbohydrates, and about 2400 calories. On this diet a certain number of cases will preserve a so-called positive carbohydrate balance; that is, they will excrete a less amount of sugar than corresponds to their carbohydrate intake. Falta gives formulas which help to determine the source of the glycosuria in those cases in which there is a so-called negative carbohydrate balance. The test diet also allows a rough estimation of the ketonuria. The age, occupation, surroundings, duration of the diabetes, and possible complications should also be considered in order to arrive at a conclusion concerning the severity of the disease. In the mild cases, the elimination of bread from the test diet will usually make the patient sugar free. In severer cases, in which the withdrawal of the carbohydrates does not suffice, we decrease the nitrogen content of the food to about 8 grams and increase the amount of butter to about 200 grams. If this also proves insufficient, it is advisable to introduce one or two days of vegetable diet; or, according to Naunyn, one day of fasting; or, according to von Noorden, an "oatmeal cure." Falta emphasizes the necessity of a close watch of the ketonuria, especially in the severer cases. It is easier to prevent the occurrence of a ketonuria than to suppress an existing ketonuria, and therefore Falta rightly says that this prevention is the fundamental point in the treatment. This is accomplished by the administration of alkalies during the period of withdrawal of the carbodydrates. The reaction of the urine, according to Falta, affords an excellent sign of the degree of acidosis. If the urine becomes alkaline, the acids surely have been neutralized. If a marked ketonuria is present, it is better to saturate the body with alkalies as quickly as possible, and when the urine has become alkaline to decrease the dose gradually, but always to keep the reaction of the urine slightly alkaline. At this stage usually a part of the sodium bicarbonate is replaced by sodium citrate. If the administration of alkalies is not sufficient to neutralize a high degree of acidosis, it is necessary to decrease the formation of the ketones. Large quantities of alcohol decrease slightly the formation of ketones. However, the carbohydrates are our main reliance for the reduction of the formation of ketone bodies. Falta says that it is best to give a low diet containing from 8 to 10 grams of nitrogen and not more than 150 grams of fat, with the addition of 100 or 150 grams of carbohydrates in the form of fruit, milk, oatmeal, etc. This naturally produces an increase of the sugar content of the blood, which should then be lowered by the interposition of two or three "vegetable days," with 50 grams of carbohydrates.

For the treatment of diabetic coma, Falta advises a diet consisting almost exclusively of carbohydrates, with the addition of 50 to 100 grams of levulose. Enormous doses of alkalies are necessary, and Falta recommends the intravenous method as the best method of administering them. He advises one liter of a 4 per cent. soda solution for this purpose. In the treatment of those patients who have become sugar free by the withdrawal of carbohydrates, Falta follows Naunyn's suggestion and keeps them two weeks on the diet by which they have become sugar free. Then bread is gradually added to the limit of tolerance. Finally, half the amount tolerated is given to the patient and variety is introduced

into the diet by means of fruit, cream, vegetables, and various kinds of bread. In those cases in which it has been necessary to reduce the amount of proteins, the nitrogen in the diet should first be increased to 12 or 14 grams. When the patient has been sugar free for three or four weeks, carbohydrates should be added. The kind of carbohydrate tolerated by the individual case may differ widely. Levulose and lactose are tolerated by some diabetics, but increase the glycosuria in others.

Falta says that especial care is necessary for those cases who do not become sugar free by the withdrawal of carbohydrates and the reduction of protein. Such cases are at times rendered sugar free by the introduction of "hunger days" or "oatmeal cures." Falta believes that the "milk cure" only diminishes the glycosuria in diabetic patients, who were formerly overnourished and are insufficiently nourished on a strict milk diet. He discusses the "potato cure" as of no value. He believes that the "oatmeal cure" has both theoretical and practical value. He describes the present method of giving the "oatmeal cure." After several days of strict diet come two vegetable days, then three oatmeal days, and finally two vegetable days. After the vegetable days, strict diet with diminished protein is given. On each oatmeal day the patient receives 250 to 300 grams of oatmeal, prepared with an equal quantity of butter and divided into five portions. Black coffee, wine, or a little brandy may be added to the diet. If there is any diarrhœa, Falta advises the use of a few drops of tincture of opium. In all but a few cases the ketonuria is diminished, and the most important indication for the "oatmeal cure," therefore, is the combating of ketonuria. In addition the patient has assimilated a considerable amount of carbohydrate without a corresponding increase in the hyperglycemia.

Falta adds some general points in the therapy of diabetics. He says that the treatment of patients with ketonuria is only possible outside of hospitals if they are very intelligent. A course at one of the many spas is often of great assistance in the treatment. He also includes carefully regulated exercise as a very important element in the treatment.

The Treatment of Contraction in Hemiplegia.—Von Criegern (*Fortschrit. der Med.*, 1909, i, 1) believes that many of the phenomena of contractures are due to congestion in the parts affected. The cramps in the muscles which are caused by unaccustomed exercise are, in Criegern's opinion, analogous to some hemiplegic contractures. The sensation of stiffness in the arms which follows rowing is due to congestion, and is relieved if the exercise is persisted in, or if the blood is allowed to pass out of the limbs by raising the arms above the head. In applying this principle to the conditions found in hemiplegia a month or longer after the onset, several methods may be employed. The contracture subsides after the application of an Esmarch bandage, and then the muscles may be exercised. Sometimes it is found best to defer exercise until after the removal of the bandage, which should always be done with the limb raised. The arm should be worn in a sling or held in an elevated position in order to avoid congestion. Criegern has noted that the paralysis often is not so marked as it had been supposed to be before the congestion had been reduced, and relates in detail the case of a man who had partially recovered from a hemiplegia two years before. His right arm was quite

powerless, and a severe degree of contracture of the hand had resulted. After three months of treatment by induced anemia this patient was able to use his hand in eating and dressing, and improvement was continuing at the time of this report. This method is not so easy to apply to the leg as to the arm on account of the difficulty in keeping the leg raised, and here an elastic stocking is advisable. The treatment is the reverse of the hyperemic method and has yielded excellent results in Criegern's hands.

Phosphorus in the Treatment of Rickets.—The action of phosphorus in rickets has not been satisfactorily studied with reference to the calcium metabolism, and for this reason SCHABAD (*Zeitschr. f. klin. Med.*, 1909, lxvii, 454) has examined the calcium metabolism of healthy and of rachitic children before, during, and after the administration of phosphorus. His results are detailed at great length, and lead to the following conclusions: (1) In therapeutic doses phosphorus produces no change in the calcium metabolism of healthy children, but it leads to a well marked retention of calcium in rachitis. (2) The retention is to be attributed to the increased absoption and the diminished excretion of calcium in the urine and feces. (3) The retention of calcium may be noted soon after the exhibition of phosphorus (three to five days); after the withdrawal of the drug this retention persists about two months (following its previous administration for two and one half months). (4) Phosphorus exerts a specific action on rachitic bones and raises their calcium content, so that the latter approaches the normal.

The Treatment of Chronic Asthma and Emphysema.—BOELLKE (*Med. Klin.*, 1909, viii, 283) has used pyrenol in the treatment of 39 cases of asthma and emphysema. He gives it in doses of from 3 to 4 grains per day. Pyrenol is a combination of Siam benzoic acid and thymol with synthetic benzoic acid and oxybenzoic acid. The Siam benzoic acid gives it expectorant properties, which are enhanced by the thymol. In addition, the thymol has anesthetic properties. The distressing dyspnœa is relieved in three or four days, the cough loses its hard and paroxysmal character, and expectoration becomes looser and easier, generally on the second day. At the same time the disturbances of the circulation disappear. A number of the patients experienced a sense of well being to which they had long been strangers. Coincidently the signs of bronchitis cleared up, in many cases entirely so. A relapse occurred in one case only. Boellke did not observe any untoward effects upon the heart, kidney, or gastro-intestinal tract in the use of pyrenol. He observed no cumulative action and no diminution of the effect of the drug after long-continued use. Heart, kidney, and bronchial complications are no contra-indications for the use of pyrenol. Boellke states that patients who had formerly been treated with atropin or potassium iodide thought that pyrenol gave them the greatest relief.

PEDIATRICS.

UNDER THE CHARGE OF
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An Epidemic of Cerebrospinal Meningitis in Paris; Efficacy of Antimeningitic Serum.—In opposition to the usual belief that there is very little cerebrospinal meningitis in Paris, NETTER (*Bull. de l'acad. de méd. de Paris*, 1909, lxi, 300) reports a small epidemic. He, himself, has seen and treated 22 cases within the short space of three months, most of them in children and infants. Many of the patients lived within a small circle; and 6 of the children attended one school. Several of the children died within a few days and the diagnosis could only be made postmortem through lumbar puncture, the serum being turbid and containing the diplococcus of Weichselbaum. The symptoms varied greatly in the different cases, but the lumbar puncture made the diagnosis a certainty in each instance. Macroscopically, the fluid in all was turbid; microscopically, lymphocytosis existed and the Weichselbaum organism was found to be present. Netter emphasizes the importance of isolation until the nasal and pharyngeal mucus is free of organisms known to bear an etiological relation to the disease. He appreciates the difficulty, but feels that this is one of the most important problems in combating the disease. Fifteen of his patients were treated with antimeningitic serum, and but 3 of these died; these deaths occurred, respectively, in one patient with old otitis media and phlebitis of the ophthalmic vein, in another with bronchopneumonia and enteritis, and in a third who was not treated until moribund. The technique of the treatment, the changes in the symptoms, and the modifications in the cerebrospinal fluid are described minutely. The latter changes form a perfect control to the employment of the serum. He injects the serum into the spinal canal, using doses varying from 20 to 30 c.c. in children, and repeating the injections daily for three to four days. He is very enthusiastic about the results achieved.

Congenital Imperforation of the Œsophagus.—P. GIBERT and J. LEMEE (*Bull. et mém. de la soc. anat. de Paris*, 1909, lxxxiv, 94) report a case of congenital imperforation of the œsophagus in a child aged seven days. Within a few moments after nursing the child regurgitated all the milk spontaneously. This regurgitation was always accompanied by marked cyanosis. Having seen 5 cases of congenital imperforation of the œsophagus within fifteen months, this condition was at once considered, and local examination revealed this to be the true state of affairs. The soft catheter could not be pushed beyond 12 cm. from the buccal orifice. A tracheo-œsophageal fistula was suspected because of the marked cyanosis after nursing and at other periods, when saliva would be vomited up. This condition was verified at the autopsy, the upper third of the œsophagus being represented by a pharyngeo-œsophageal cul-de-sac, the lower two-thirds by a canal

extending from the stomach to the trachea, opening into it at its posterior surface 3 mm. above its bifurcation and there separated from it by a valve.

Grave Intestinal Hemorrhage in the Newborn.—Guerin-Valmale (*Bull. de la soc. d'obst. de Paris*, 1909, xii, 97) reports the following case: A little girl, born without difficulty of healthy parents, was exposed to considerable cold air shortly after her birth. She sneezed and cried a great deal from her birth on. She vomited after every nursing, but took the breast well during the first forty-eight hours; by that time she was so weak that she could hardly cry or nurse. On the third day a hemorrhagic eruption appeared in different parts of her body, especially, however, over the buttocks. The child became very anemic, and was constantly in partial stupor. The stools, which, until now, consisted of meconium, contained considerable blood, and several times during the next few days seemed to be composed of pure blood. Chloride of calcium was given with the mother's milk from a spoon, and adrenalin was ordered by lavage. This condition continued for a week, some blood also being discharged by the vagina. Recovery finally, however, was complete, and a month later the child bore no further traces of the hemorrhages. Discussing the cause, Guerin-Valmale excludes prenatal infection, as the mother was perfectly well during the entire pregnancy; postnatal infection, as no signs of any such existed, even the umbilical cord falling off without any trouble; syphilis, there being no history of any. He considers the case to be one of hereditary tendency to hemophilia, the maternal side being remarkably nervous, the paternal side containing several individuals with a tendency to epistaxis and other bleeding. The chilling immediately after birth is considered the exciting cause.

The Antitoxic Properties of Gastric Juice and its Importance in Infant Feeding and Immunity.—Several years ago A. Schultz published the results of certain researches, which showed him that the gastric juice of the newborn and nursing infants destroys the diphtheria toxin; this property he found to pertain to all stomachs functionating normally and not to present any individual variations, which would depend on age, or the mode and state of nutrition of the child. This was found to be due to an organic substance, some importance also being attributed to the degree of acidity of the solution. New studies along the same line performed by him (*Zeit. f. Hyg.*, 1909, lxi, 115) produced the following results and conclusions: (1) The diphtheria toxin is destroyed by a solution of hydrochloric acid, the amount of free acid of which corresponds to the gastric juice of a nursing infant. (2) The action in vitro appears after a certain reaction time (more than fifteen minutes). (3) In vivo the free hydrochloric acid continues to act subcutaneously on the poison, if the inoculation is performed immediately after they are mixed. (4) Hydrochloric acid fixed by egg albumin is without influence on the diphtheria poison, but when fixed with peptone it destroys the toxin. (5) Hydrochloric acid loses its activity when added to cow's milk and cooked human milk, but when combined with ordinary mother's milk the diphtheria toxin is destroyed by the mixture. (6) Combined hydrochloric acid is weaker in its action on diphtheria poison than is

free hydrochloric acid. (7) The reaction appeared as quickly in most of the experiments, whether combined or free hydrochloric acid was employed, as long as equal quantities of the solution were used. If the amount of free hydrochloric acid was smaller, the appearance of the reaction was correspondingly slower. (8) The amount of the antitoxic power of both free and combined acids equals their antiseptic properties. (9) The concentration of the pepsin solution employed is forty times stronger than the pepsin strength of the gastric juice of the nursling; it destroys the diphtheria toxin as quickly as does the hydrochloric acid solution, and no free hydrochloric acid need be present in the pepsin solution. (10) The destruction is complete after one minute, the reaction beginning immediately. (11) The acid pepsin solution quickens the results reached with combined hydrochloric acid; it increases the digesting power so much that the destroying power of the pepsin solution containing combined hydrochloric acid is almost equal to that of free hydrochloric acid. (12) The alkaline pepsin solution is without power. (13) Rennet does not act on the diphtheria poison; lactic acid destroys it. Hydrochloric acid combined with mother's milk possesses an antiseptic power equal to that of free hydrochloric acid. In the case of breast-fed children the disinfection of the stomach occurs therefore not only toward the end of the digestive period, but also to some degree during it. This is of especial importance in the cases of children, who, while being artificially fed, are seized with intoxications and infections associated with gastro-intestinal symptoms, and explains why such children recover much more quickly from such diseases when their nourishment is changed to mother's milk.

Ovarian Cysts in Childhood.—Cheney (*Pediatrics*, 1909, xxi, No. 3) reports a case of ovarian cyst in a girl, aged sixteen years, who had been previously healthy. Menstruation had been regular and normal for two years. She had noticed a growth in the abdomen for about two months, had no fever and few symptoms except discomfort and occasional pain. A large tumor was found occupying the entire left half of the abdomen and extending down into the pelvis, its upper border not connected with the spleen. The outline was regular and felt solid or semisolid. It partly filled the pelvis and had pushed a normal uterus downward and backward. Examination of the blood yielded negative results. On operation a papillary cystadenoma of the left ovary, weighing nine and one-half pounds, was removed. Cheney states that ovarian tumors are not uncommon in childhood, being more frequent as puberty approaches. They are cystic or solid, and include sarcoma, carcinoma, and dermoid cyst. Of all classes multilocular cysts, or cystadenoma, are the most common. He refers to Howard Kelly's collection of 126 cases of ovarian tumors in children. Of this number number, 55 were cysts, 47 dermoids, and 24 solid tumors. The youngest patient reported was an infant, aged four months, the next were three years and four years respectively. In diagnosis ovarian cysts must be differentiated from dermoids and sarcomas of the ovary; from peritonitis with encapsulated effusion; from malignant and cystic disease of the kidney; and from retroperitoneal affections.

OBSTETRICS.

UNDER THE CHARGE OF
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Convulsions in the Newborn.—Esch (*Archiv f. Gynäk.*, 1909, lxxxviii, Heft 1) reviews the literature of this subject, in which the mortality of convulsions in the newborn varies from 25 to 100 per cent. The frequency of this complication is reported as from 0.13 per cent. to 2.69 per cent. The cause of the convulsions is most frequently umbilical infection, then œdema and asphyxia, then syphilis, and hereditary eclampsia. Among the illustrative cases reported is one by Stöckel, of a female child delivered spontaneously after normal labor. For the first day after birth the child was restless, cried frequently, and showed in the face great pallor. In the morning of the second day it vomited, and the greater fontanelle was protuberant. Spasms of the limbs, fingers, and toes, with stiffness of the back, developed, and pressure upon the internal surface of the arm produced spasmodic closure of the fist. On the evening of this day the child suddenly became deeply cyanosed, and tonic convulsions developed throughout the muscular system. On the third day the convulsions ceased, but there was increased muscular irritability in the arms and legs and the muscles of the back. On the fourth day the symptoms were less pronounced, and the child ultimately recovered. No signs or history of syphilis could be detected. The case was diagnosticated as cerebral irritation occasioned by bleeding above the tentorium without special localization of the lesion. As the hemorrhage gradually ceased, and was not profuse, the child recovered. Auto-intoxication of intestinal origin, may also produce convulsions, as illustrated by the following case: The birth of the child was spontaneous, and the mother apparently healthy. The child cried and vomited excessively during the second day of its life. There was increased muscular and nervous tension of the extremities and back. Meconium was discharged freely. On the next day the bowel movement was yellowish in color, vomiting continued, and the hypertonic condition of the muscles was more pronounced. The abdomen became swollen, and on the day following fluid could be diagnosticated in the abdominal cavity. Death occurred shortly after. There were no clonic convulsions. Upon autopsy a thick, yellowish brown membrane was found lining the abdomen, composed of fibrin and white blood cells. The abdominal fluid contained intestinal bacteria, leukocytes, and erythrocytes. The intestines were matted together. On separating them a perforation of the bowel was found at the jejunum and the borders of the perforation showed infiltration with small cells. The contents of the bowel escaping into the peritoneal sac had caused death. There was also hyperemia in the lungs and membranes of the brain, with petechial hemorrhage in the pleura, and hemorrhage in the uterus, membranes, and stomach. The cause of the intestinal perforation could not be clearly distinguished. Esch calls also attention to pathological conditions in the medulla causing convulsions in the new-

born. He cites the case of a child nursing poorly on the third day, and seeming oppressed or stupid. On the fourth day cyanosis developed with regurgitation of food. Spasms of the muscles of the extremities and back appeared, varied by clonic spasm. The temperature was much below normal, and the child seemed collapsed. The bowels moved freely. On the day following there was a free discharge of mucus from the mouth and nose. The breathing became labored and the child died. At autopsy no jaundice was found, the heart was normal, the lungs contained air, and there was no pneumonia. The spleen was enlarged and dark red; the suprarenals were large but without excessive fat, and with no hemorrhage. The kidneys were engorged with blood; the liver was filled with blood, and large and yellowish brown in color; the stomach and intestines were normal; there was no tumor upon the cranium or scalp; there was no rupture of the superior longitudinal sinuses; and in the subdural spaces there was some thickened blood. The tentorium was distended and tense. The cerebrum showed no hemorrhage and its configuration was normal. In the cerebellum there was pronounced hemorrhage, sufficient to alter the contour of this portion of the brain. The fourth ventricle was greatly compressed, but contained no blood. There was also pressure upon the medulla and pons. The meninges of the cord contained fluid blood, but the cord itself was normal. He also reports the case of an infant born by version and extraction because of prolapse of the cord and transverse presentation in a flat rachitic pelvis. The child was cyanotic after birth. After efforts at resuscitation the child breathed spontaneously, but died soon after. Section showed the occipital bone pressed under the parietals, and hemorrhage in the region of the cerebellum. Esch also calls attention to the fact that in the case of a child dying a few days after birth, disturbances of nutrition, with the development of pneumonia, may produce convulsions similar to those observed in cerebellar lesions in the newborn.

Axillary Milk Glands Developing during Pregnancy.—Seitz (*Archiv f. Gynäk.*, 1909, lxxxviii, Heft 1) contributes a paper upon this subject, reviewing the literature, and giving illustrations of cases with microscopic drawings of specimens removed. He concludes from his studies that the sweat glands of the axillary region increase in size and function during pregnancy, as do the mammary glands. Under conditions not clearly recognized, these axillary glands become greatly enlarged during pregnancy and the puerperal period. In addition to the glands normally found in this region, others may develop to an extraordinary degree. Some take on the shape and function of milk glands and show the characteristic secretion. Microscopic examination shows the structure of these to be practically identical to those of the enlarged sweat follicles. As these glands are without excretory ducts, they become distended, and are largest in size from the fifth to the seventh day of the puerperal period. They usually subside by the fourteenth day. These glands have nothing to do with so-called accessory mammary glands, or with the auxiliary follicles sometimes found about the nipples. They are more often seen in patients who have highly developed mammary glands. When these tumors are examined microscopically, they might readily be mistaken for adenoma. The fact that the swelling occurs during pregnancy and the puerperal state, should

make clear the diagnosis. A somewhat similar swelling is sometimes observed at menstruation.

The Practical Value of Antistreptococcic Serum.—Heynemann and Barth (*Archiv f. Gynäk.*, 1909, lxxxviii, Heft 1) have investigated by bacteriological and clinical studies several of the antistreptococcic sera now employed. They find that those supplied by Höchter, Merck, and Schering give results which are more or less discernible. In none of the cases observed did the serum equal in potency the serum of a normal puerperal patient. In fresh serum obtained by these different substances the potency was never so great as that of normal serum from a normal puerperal patient. With a diluted antiserum 1 to 1000, a more pronounced phagocytosis was obtained than with a diluted normal serum. This result was not altered when fresh normal human or horse serum was added as a complement, or by the use of leukocytes obtained from the horse. The potency of antistreptococcic serum does not depend upon its containing substances causing phagocytosis. With serum of the highest potency in doses of 100 c.c. no results were obtained in puerperal streptococcic infection. Experiments upon animals should be conducted with the hope of obtaining a more efficient serum of high potency, which can be used in the streptococcic infection of parturient women.

Indications and Prognosis of Operations for Gallstones Performed during Pregnancy.—Roith (*Monatssch. f. Geb. u. Gyn.*, 1909, Band xxix, Heft 4) reviews the literature of the subject, finding but 9 reported cases; 8 of these are reported as recovered; in but one did the uterus empty itself fourteen days after the operation, and this patient died; one patient aborted after operation, but recovered. Pus was present in the gall-bladder in 5 out of the 9 cases. All of them were before full term with one exception, and this patient's case terminated fatally, labor occurring fourteen days after the operation; 4 of the cases were treated by cholecystectomy with drainage of the hepatic duct; 4 by cholecystotomy; and one by sewing the edges of the gall-bladder to the edges of the abdominal incision. These cases indicate that pregnancy is not a contra-indication against the operations upon the gall-bladder and bile ducts. The prognosis is not made especially grave by the occurrence of pregnancy. The probability of the interruption of pregnancy depends upon the period of gestation, the intensity of the jaundice and the infection, and the extent and time occupied in the operation. During the first half of pregnancy the danger of interruption is very little. Toward the end of pregnancy the danger of interruption increases, and incisions obliquely along the borders of the ribs, and cystotomy, are the method of choice. By this method the operation is made as brief as possible, and the uterus is disturbed as little as possible. In severe cases at the end of gestation the patient should be delivered before the operation upon the gall-bladder is undertaken. Vaginal Cesarean section seems indicated in these cases, because the incisions for delivery are removed from the area of infection, and infection is less likely to occur. When labor pains develop after operation the uterus should be emptied as soon as possible, because uterine contractions tend to spread infectious matter from the hepatic wound into the peritoneum. Anesthesia in these cases requires especial attention.

GYNECOLOGY.

UNDER THE CHARGE OF

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Non-surgical Treatment of Uterine Displacements.—H. J. Boldt (*Jour. Amer. Med. Assoc.*, 1908, li, 903) makes a plea for treatment, other than surgical operations, for displacements of the uterus. Boldt believes anterior displacement of that organ requires no treatment but that by pessaries; acquired retrodisplacements free of adhesions are successfully treated by properly applied pessaries, preferably of the Smith or Thomas variety. Boldt, after fully describing methods of replacing the uterus so displaced, states that pessaries should never be applied when the uterus is not in proper position.

Injuries to the Bladder during Hernia Operations.—R. E. Skeel (*Amer. Jour. Obst.*, 1908, lviii, 964) states that injuries to the bladder taking place during operation for the ordinary forms of hernia are of two distinct classes, as pointed out by Bruner in 1898. The first and probably the least common form occurs from the presence within the sac of the bladder or a diverticulum therefrom; or in the paraperitoneal form the bladder escapes along the side of the hernial sac and is injured when the latter is incised or ligated; or otherwise damaged when the neck of the sac is dissected from subjacent tissues. The other and commonest form is probably due to traction upon the sac in an effort to ligate it as high up as possible, with the result that the peritoneum passing from the bladder to the pelvic wall is dragged into the incision, carrying the bladder with it, where it is either ligated, incised, or torn. Skeel has collected 325 cases, of which 225 were in males, 80 in females, and in 20 the sex is not given. Of the males, 216 were inguinal, 4 femoral, 1 of both varieties, and in 4 the variety is not definitely stated. In the female 21 were inguinal, 50 femoral, and in the remainder the variety is not given. In Bruner's statistics 59.5 per cent. of the cases in which the bladder was recognized it was injured, while in the cases collected by Skeel since 1896 this rate fell to 27.6 per cent. The mortality rate was about 24 per cent. For the purpose of positively identifying the bladder when opened by accident Skeel advocates the routine use of methylene blue for a day or so prior to the herniotomy.

The Menopause.—D. H. Craig (*Jour. Amer. Med. Assoc.*, 1908, li, 1507) believes there are three normal types of the menopause, sometimes seen more or less in combination with one another, and departures from these types are easily recognized. The symptomatology alone will very often point to the organ or region at fault, but such deduction should always be given physical confirmation. Lesions at this time are as deserving of and amenable to treatment as at any other time, and neglect of such treatment engenders inestimable, unnecessary suffering, and is one of the greatest factors in causing so many malignant

uterine growths to attain an inoperable state of advancement. Failure properly to diagnosticate and treat such cases will inevitably bring opprobrium on the profession as a whole.

Experimental Contributions on the Operative Treatment of Acute Puerperal Peritonitis.—Zangemeister (*Ztschr. f. Geb. u. Gyn.*, 1908, v, lxii, 510) reports the results of several series of experiments with streptococcic infection of mice. He injected a diluted quantity of streptococcus culture into the tails of these animals and then amputated them. He found that the removal of the infected area delayed death. Antistreptococcic serum given alone prolonged life also. If, however, amputation and serumtherapy were combined, the effect was augmented to such a degree that none of the animals died, which showed conclusively that the highly curative properties of an effective antistreptococcic serum may be considerably increased by the simultaneous removal of the site of primary infection. The conditions in puerperal peritonitis are very unfavorable, as usually more than half of the period of time between infection and death has passed before symptoms leading to operation appear, nor can the site of primary infection be so readily removed as in animal experiments, and finally, the operation needed is such as greatly to tax the resistance of the organism. In view of these facts, he considers it very doubtful whether the operative removal of the primary seat of infection without simultaneous serumtherapy in acute puerperal streptococcic peritonitis could be successful. The prognosis is much more favorable if the operation is combined with serumtherapy. In such cases the removal of the uterus and adnexa by the abdominal route is advised.

Renal Decapsulation in Eclampsia.—Kehrer (*Zeit. f. Gyn., u. Urol.*, 1908, i, 111) collected and analyzed all cases of eclampsia treated by renal decapsulation of Edebohls, and arrives at the following conclusions: (1) Decapsulation is misapplied when employed in eclampsia during pregnancy. In such cases the detoxinizing therapeutic measures and accouchement forcé are indicated. (2) According to our present knowledge decapsulation is indicated in those severe cases of puerperal eclampsia in which the kidneys are involved to a considerable extent, and it should be recommended in the cases in which, in spite of delivery, venesection, and application of all other measures known to stimulate the excretory organs, the attacks grow worse and the general condition becomes aggravated. In such cases only a few hours should be allowed to elapse before decapsulation is performed. (3) Edebohls' operation excites increased urinary and urea excretion and a simultaneous increase of excretion of the eclamptic toxin in the circulating blood. (4) The success of the decapsulation rests in the increased diuresis which is a physiological and not an anatomical result. The anatomical basis for operation on which Edebohls, O. Franck, and others rely, namely, development of vascular anastomoses between the renal surface and the adipose capsule, does not exist. (5) The improvement in the albuminuria, hematuria, and cylindruria occurs synchronously with increased diuresis, or soon follows. The disappearance of these signs depends directly on the degenerated conditions of the renal parenchyma at the time of operation. This explains why chronic and suddenly increased

inflammatory processes are little or not at all affected. (6) The precise indication for operation and the further study of the influence of the decapsulation on the activity of the kidney and the entire organism depend on most careful functional tests of the kidneys. Microscopic examination of the excised pieces of kidney and comparison with the functional tests before and after operation may also serve to fix accurately the time when decapsulation should be done. It may be possible to ascertain in this way whether in all cases in which indication for decapsulation is recognized bilateral operation is necessary or whether unilateral decapsulation suffices. The effect of massage of the kidney should also be studied experimentally. (7) In Edebohls' renal decapsulation of the kidneys we seem to possess a remedy in severe cases of eclampsia, and there is hope that a portion of the 20 per cent. of cases of eclampsia which do not improve after the uterus is emptied, or those which occur thereafter, may be cured by renal decapsulation at the proper time. The cases of degenerative changes in other organs, particularly of the liver and the heart, cases of pulmonary œdema, and of hemorrhages in internal organs remain hopeless. The future will show whether renal decapsulation is justifiable or whether later renal function is impaired by enclosure of the kidneys in inelastic connective tissue bands.

Inflammatory Proliferation of the Mucous Membrane with Epithelial Hyperplasia in the Fallopian Tube.—M. Neu (*Zeit. f. Geb. u Gyn.*, 1909, lxii, 489) reports the case of a woman, aged fifty-one years, with pulmonary tuberculosis, who developed an irreducible prolapse of the adnexa into the vagina after vaginal total extirpation of the myomatous uterus. At the time of the operation the adnexa appeared to be quite normal. An unusually abundant suppuration followed the operation. The prolapsed adnexa became involved in the inflammatory process, particularly the more deeply situated adnexa on the right side. As a result a suppurative inflammation of the tube and ovary developed which passed into a chronic inflammatory condition. The secretion was finally disposed of by removal of the prolapsed adnexa in the vagina about a year after the operation. The portion of removed tube was cut into a series of 245 sections and examined microscopically. It showed all the characteristic signs of a purulent and hemorrhagic salpingitis and proliferations of adenoma-like character of the partly stratified tubal epithelium.

Malignant Tumor of the Ovary Complicated by Twisted Pedicle.—Guibe (*Ann. de gyn. et d'obst.*, 1909, xxxvi, 110) reported a case of sarcoma of the ovary the pedicle of which organ was many times twisted. The patient, a healthy peasant, aged sixty years, was admitted to the hospital for intestinal obstruction, that had supervened after a severe attack of coughing that immediately gave rise to severe pain in the hypogastrium four days before. The blackened friable tumor mass of the size of two fists was removed. Pulmonary complications began ten days after operation and ended fatally six days later. When malignancy of a growth of an ovary is suspected and its removal decided upon, both appendages and the body of the uterus should be removed, is the advice offered by Guibe, who comments also on the comparative rarity of twisting of the pedicle of solid ovarian tumors as compared to cystic ones.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

UNDER THE CHARGE OF
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Asymmetry of the Hard Palate.—Mosher (*Laryngoscope*, April, 1909) contributes an instructive and well-illustrated paper on "The Form of the Hard Palate," showing the effect of asymmetry upon the teeth, rhinopharynx, nasal fossæ, and contiguous structures. He deems asymmetry of the two sides of the hard palate due to unequal descent of the antrum or to unequal growth of the superior maxillæ of equal importance with faulty eruption of the incisor teeth in causing deviations of the septum.

Thyroid Tumor of the Tongue.—Stuart-Low (*Jour. Laryng., Rhin., and Otol.*, April, 1909) reports a case of a female patient, aged thirty-two years, with a large, firm swelling at the base of the tongue. A similar condition had existed eleven years previously, when an operation was performed. She was again operated upon for recurrence nine months ago. It was now proposed to perform a preliminary laryngotomy and split the tongue in the middle line, so as to remove the tumor radically.

Hereditary Syphilis of the Rhinopharynx.—Trapenard (*Revue hebd. de laryng., d'otol. et de rhin.*, January 30, 1909) reports two cases in children of hereditary syphilis of the rhinopharynx in which the lesions simulated lymphoid hypertrophies at the vault of the pharynx. The cases were recognized, however, and promptly cured under specific treatment.

Intranasal Rhabdomyoma.—Vail (*Laryngoscope*, December, 1908) reports this unique case in a well-nourished woman, aged fifty-eight years, who presented herself with occlusion of the left nasal passage which had gradually followed an attack of grippe six weeks previously. A growth was found completely occluding the nares and presenting post-rhinoscopically a mass of pale, light gray tissue, and feeling like a nodulated mass of tough tallow. It looked suspiciously like a sarcoma despite lack of reddish color and not bleeding when wounded. It was removed piecemeal with considerable difficulty, but without hemorrhage, and inspection seemed to show thorough removal. Microscopic inspection by Dr. Welch of Johns Hopkins University, revealed it as a case of rhabdomyoma sarcomatodes. Recurrence ensued, and an external operation for its removal revealed that it filled the antrum of Highmore completely, and that it was not adherent at its limitations. The removal of the entire mass was easily accomplished by simply lifting it out of its seat. Unfortunately, heart failure took place under the chloroform anesthesia, and in spite of the employment of every

known means of restoring cardiac power, the pulse remained small and thready, and the patient died sixty-eight hours after the operation.

Lymphosarcoma of the Frontal Sinuses.— Abercrombie (*Medical Reports of the Central London Throat and Ear Hospital*, vol. i) reports a case of lymphosarcoma of the frontal sinuses in a woman, aged seventy-five years, who succumbed from erysipelas developed after a third operation for recurrence.

Chondroma of the Left Maxillary Antrum.— Abercrombie (*Ibid*) reports this case in a girl, aged sixteen years. The growth was removed by operation, and in three months a second operation was required for recurrence, after which she did well. Three years after the second operation a similar though much smaller growth was removed from the antrum of the other side. Recovery was uneventful, and she left the hospital after a ten days' residence.

Apparent Primary Laryngeal Tuberculosis.— Atkinson (*Medical Reports of the Central London Throat and Ear Hospital*, vol. i) reports a case of laryngeal tuberculosis in a man, aged sixty years, without any other manifestation. The left vocal cord presented a thickened, slightly fleshy looking appearance limited to its anterior two-thirds, with delayed sluggish movement. On the upper surface one or two enlarged vessels were observed, but nothing else in any way abnormal in any portion of the larynx. Some six weeks later, implication of the lungs became evident. Atkinson deemed this case worthy of being recorded owing to its simulating epithelioma in its early stages, and also because all the evidences, both positive and negative, point to the conclusion that it was one of primary laryngeal tuberculosis.

Subhyoid Pharyngotomy for Epithelioma of the Epiglottis.— Castex *Jour. Laryng., Rhin. and Otol.*, April, 1909) reports a case of a man, aged fifty-four years, from whom a growth on the epiglottis was removed by thermocautery after external access to the parts by subhyoid pharyngotomy. The after-results were very good. A second patient, however, whose case was analogous, sank rapidly thirty-six hours after the operation, with intense pulmonary oppression. Relying on this observation and on that of a laryngectomy in which the trachea had been united to the skin, Castex thinks that some of these rapid deaths are explicable by a sudden and diffuse pulmonary œdema, the cause of which perhaps is a reflex initiated in the vagus and sympathetic filaments irritated at the level of the pharynx.

Laryngotomy and Fulguration in Cancer of the Larynx.— Laurens (*Arch. intern. de laryng., d'otol. et de rhin.*, Janvier-Fevrier, 1909) reports two cases of intrinsic carcinoma of the larynx in which direct access to the growths was secured by thyrotracheotomy, and after removal of the growth, in one case with curette and the other with scissors, electric fulguration was applied to the surface of implantation and a little beyond, for three minutes, at the end of which time the cartilages and surfaces were carbonized. Good recoveries were made in both instances, and the prospects for successful results seemed favorable.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF
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Changes in the Arterial Blood Pressure in Heart Disease during Disturbances in Compensation.—In 1901 Sahli stated that in cases of heart disease the recovery from a break in compensation may be accompanied by a fall in blood pressure. Though the observation has been made casually several times since, there has been no systematic study of this point up to the present time. Lang and Manswetowa (*Deut. Arch. f. klin. Med.*, 1908, xciv, 455) have made observations upon the blood pressure in 28 cases of heart disease and 7 cases of emphysema. In 17 out of 18 cases of uncomplicated mitral disease, both the systolic and diastolic pressure fell with recovery from broken compensation. In some instances the fall in systolic pressure was as much as 30 to 40 mm., and the drop in diastolic pressure even greater. In 7 cases of emphysema there was uniformly a well-marked decrease in pressure with the onset of compensation. Only 5 of the 10 cases of aortic insufficiency and arteriosclerosis presented a fall in pressure. These results at first seem paradoxical, but if one considers the condition of the capillaries during broken compensation the explanation is evident. With the back pressure in the venous system there comes greater peripheral resistance in the capillaries, which may be at least one cause of the high pressure. Again, the cyanosis produced by the poor aëration of the blood in itself brings about a contraction of the peripheral capillaries, thus assisting in the increased pressure. It is noteworthy that in those types of heart disease in which the right side bears the brunt of the lesion and in emphysema, in which, again, the right side of the heart is principally affected, there is the most constant and marked fall in blood pressure during recovery from broken compensation. This is probably due to the fact that the left ventricle is capable of the attempt to overcome the increased peripheral resistance. In 20 of the 35 cases the pulse pressure was higher during disturbances in compensation than after compensation had been restored. No definite relationship could be determined between the appearance and disappearance of œdema and the alterations in blood pressure.

The Hematogenous Origin of Purulent Nephritis.—J. Koch (*Zeit. f. Hyp. u. Infekt.*, 1909, lxi, Heft iii) considers the most important factor in descending purulent nephritis caused by the staphylococcus to be that staphylococci in the blood stream are excreted through the kidneys in the urine. Staphylococci, which metastasize only by the blood, are found in the blood in many local, as well as general, infections, and, as is shown by Lenhartz, are much more apt to metastasize than

streptococci (in 95 per cent. of 22 cases, while streptococci showed 35 per cent. in 160 cases). This elimination through the kidneys, however, is a pathological, not a physiological, condition; that is, some injury to the eliminating organs—a glomerulonephritis—must exist, otherwise the staphylococci in the blood stream will pass on. This injury, according to Koch, can be dealt by the toxin elaborated by the staphylococci, which are thus enabled to pass through the vessel walls of the glomeruli and the renal epithelium. Whether or not it is possible for the staphylococci secondarily to make their way through the blood-vessel walls and epithelium of the convoluted tubules is not yet determined. He distinguishes two forms of abscesses: Those arranged in groups in the cortex with streak-like prolongations into the medulla; and secondly, those in the medulla, with little or no involvement of the cortex. In experimental nephritis he finds that the former are produced by injections of virulent cultures; the latter, if the strains of staphylococci are weakened. The streak-like abscesses in the medulla arise from the tubules being blocked by casts, which then not only offer a favorable medium for the growth of the staphylococci, but allow the elaboration of toxins which damage the surrounding renal tissue and make it susceptible to extension of the abscess. Koch has found in his experiments occasional cases of chronic nephritis, perirenal inflammation, or lesions in one kidney only. He considers the demonstration of staphylococci in the urine to be of importance in the diagnosis of such staphylomycoses as pyemia, osteomyelitis, endocarditis, phlegmon, furunculosis, and carbuncles.

The So-called Abnormal Tendon Fibers of the Heart.—In 1905 Tawara, in an exhaustive monograph on the system that conducts the contracting impulse in the heart of mammals, proved that the so-called Purkinje fibers were in reality the broadened out and modified terminations of the atrioventricular bundle of His. He later (*Beitr. zur. path. Anat.*, 1906, xxxix) asserted that the small bundles attached by one extremity to the septum of the heart and by the other to the base of a papillary muscle (the so-called "abnormal tendon fibers") are in reality nothing but Purkinje fibers which are reaching their final destination (the papillary muscles) by an abnormal route. Instead of being subendocardial, as is normally the case, Tawara claims that the fibers in these cases have become isolated from the myocardium and invested with a separate endocardial sheath. He bases his argument on the fact that the fibers arise from the interventricular septum, where the left branch of the bundle of His terminates, and end in a papillary muscle, where the Purkinje fibers also terminate. Mönckeberg, however, takes exception to this claim (*Verhandl. d. deut. path. Gesell.*, 1908, S. 160), and proves that in many cases, at least, the bundle of His or the Purkinje fibers are not contained in these fibers. By studying them microscopically and in serial sections at their origin and insertion, he was able to demonstrate four distinct types: one in which there were no muscular elements of any kind; a second consisting of normal heart muscle (*i. e.*, a connecting bridge between septum and papillary muscle); a third consisting wholly of a continuation of the left branch of the bundle of His (*i. e.*, the same as those described by Tawara); and a fourth made up of both normal heart muscle and the continuation of the left branch (*i. e.*, combination of second and third).

In this connection it is interesting to note the observations of Saigo (*Verhandl. de deut. path. Gesell.*, 1908, S. 165) on the part played by Purkinje fibers in diseases of the myocardium. In such regressive changes of the myocardium as fatty degeneration, necrosis, connective tissue increase, etc., he finds that these fibers are always more or less involved. The perivascular nodules found in the myocardium by Aschoff and Geibel in cases of acute rheumatic fever were also found to invade the Purkinje fibers. In brown atrophy or hypertrophy of the heart, however, the Purkinje fibers were found to be distinctly less involved than the rest of the heart muscle.

In the same journal (p. 150) there is a short communication by Aschoff, with two illustrations showing the presence of glycogen in the Purkinje fibers. This, in connection with the morphology of the impulse-conducting system (that is, narrow, incompletely striated fibers, with numerous nuclei) is taken as another proof of their intermediate position between adult and embryonal heart muscle.

The Disappearance of Trypsin in the Circulation.—Since von Bergmann and Guleke have shown that with the destruction of a large portion of the pancreas in the body, substances are formed which when absorbed lead to fatal intoxication, it seemed possible to Bamberg (*Ztschr. f. exp. Path. und Therap.*, 1909, xv, 743) that in less marked disturbances of the pancreas trypsin might be absorbed and ultimately eliminated by the kidneys in the urine. This led him to repeat some of the experiments upon the appearance of trypsin in the urine under normal and pathological conditions. By a method which enabled Bamberg to detect a quantity of trypsin equal to 5 decimilligrams of the dried trypsin powder he was unable to obtain tryptic digestion in the urine from fourteen normal men and eight dogs. Injection of trypsin into dogs demonstrated that only after very large amounts had been given could trypsin be found in the urine. Transplantation of the pancreas of one dog into the abdomen of another led to extensive fat necrosis, toxemia, and death, but only once in six experiments was trypsin eliminated in the urine. After considering the various possibilities which might account for the disappearance of the trypsin in the body, Bamberg finally concludes that it is in some way made ineffective by the antitryptic property of the blood serum. In none of his experiments was the antitryptic property of the blood serum overneutralized. The blood serum in no instance showed tryptic properties, but rather an increase over the normal of antitryptic property.

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ORIGINAL ARTICLES.

A STUDY OF ACHYLIA GASTRICA.

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What is achylia gastrica? It was defined by Einhorn as comprising 'a class of cases in which there is absence of gastric secretion," and, "in which clinically the diagnosis of atrophy of the gastric mucosa seems to be justifiable." This writer would not include cases depending upon pernicious anemia. Apparently this definition describes a result following a variety of causes, hence we are led to conceive of the condition not as a disease, but as the result of diseases. If this be true, I fail to understand why the condition when occurring in pernicious anemia should not be included.

I have attempted to state here some conclusions reached in the study of 132 cases. Some of these did not fall strictly within the definition given. If, however, we are to form an opinion as to the causes leading to achylia gastrica, and of cases in the stage of development, then these cases should be included. I believe that it is wise to include them and to attempt the explanation of a pathological change that is not very uncommon.

The Incidence of Achylia Gastrica. These 132 cases are all private cases, not including hospital cases, and they bear a ratio to other stomach diseases examined as 1 is to 20. They may be divided into groups:

1. Cases in which there is persistent absence of secretion of ferments and of hydrochloric acid.

2. Cases in which there is a very low, but steadily decreasing secretion, which finally disappears entirely and remains permanently absent.

3. A group of cases which behave like one or the other of the preceding, save that at long intervals there may occasionally be found traces of combined chlorides or peptone.

4. There is rarely found a case in which all secretion is absent for a long period, after which there is a slow and gradual return (this group does not include the more frequently seen cases in which there is temporary absence of secretion, lasting for days or weeks, to be followed by normal digestion).

Causes. The various causes that result in achylia gastrica probably differ in nature: (*a*) Those that accompany pernicious anemia apparently result from a definite atrophy of glandular parenchyma of the stomach. (*b*) Others seem to follow gastritis. (*c*) Others appear to be secondary to general infection, possibly from gastritis, as is seen after typhoid fever, syphilis, etc. (*d*) There remains a large group in which, from unknown causes, the secretion becomes more and more depressed (as mentioned in the second heading) until complete achylia is established. It has been suggested that the trouble in the beginning is functional, and that subsequently gland structure disappears, similar to atrophy from lack of use in other regions. In attempting to follow the course of cases apparently about to become complete achylia gastrica, there is confessedly a source of possible error through misinterpretation. A case which shows a trace of combined chlorides or a faint biuret reaction may go on to complete loss of secretion; but, on the other hand, secretion may be found restored if the case is studied long enough. Nevertheless, these cases should be studied in relation to achylia gastrica, because in them only are we able to discover achylia gastrica in its process of development. To limit the discussion to cases in which no secretion has been found after prolonged observation is to limit the study not to the process itself, but to its result. Of course, there is profit in studying the effects upon the economy of the permanent loss of gastric secretion. This is one question, and it has value; but I feel that there is greater value in another question, that of analyzing the processes that lead to the permanent suppression of gastric secretion.

Sex. Of these 132 cases, 62 were males and 70 females.

Age. Ages were from twenty-one to seventy-two years; but only five cases were under thirty and only one beyond seventy. There were 29 cases between the ages of thirty and forty; 37 cases between forty and fifty; 28 between fifty and sixty; and 25 between sixty and sixty-nine. A pretty even distribution between males and females occurred in these several decades.

Occupation. In occupation the greatest variety occurred. There were 44 different occupations listed out of 130 cases. Appar-

ently occupation has no bearing. Perhaps it should be noted that 13 per cent. were farmers.

Previous Health. The previous condition of health is not as completely recorded in the earlier cases as I could wish. In studying the footings I am convinced that this is a matter of importance. In those in which the previous history was carefully recorded about 25 per cent. complained of long-continued stomach trouble; about 14 per cent. of prolonged mental strain or worry; 14 per cent. of influenza; and what seems to be of greatest significance, 20 per cent. had typhoid fever. In one case there is a definite statement that the stomach trouble began immediately after typhoid fever, that it never disappeared, and that complete achylia is now present. 10 per cent. had had pneumonia, and the history of hard drinking was found in only one case. There were only three cases in which syphilis was certain. One patient had a tape-worm and one had diabetes.

Of the accompanying diseases or morbid conditions, some stand out prominently. I am unable to state the proportion of patients subjected to a critical eye examination; but there were 30 recorded as having eyestrain. I believe that this condition was present in 50 per cent. of the cases. Pernicious anemia was present in 23 cases; marked arteriosclerosis in 8 cases; catarrhal gastritis in 6; hysteria in 4; persistent headache in 5; advanced nephritis in 3; marked tobacco intoxication in 2; marked alcoholic intoxication in 1; morphine habit in 2. Anemia was very commonly complained of.

Condition of the Intestine. Of 115 cases, the bowels were reported regular in 23; constipated or sluggish in 52; loose (diarrhœa) in 31; irregular in 9. The diarrhœa often followed a period of constipation. In connection with the state of the bowels, it seems to me important to consider specially the question of gastric motility.

In the majority of cases of achylia gastrica the stomach is found to empty itself more quickly than is normal. Frequently the test meal has to be withdrawn before the lapse of the usual time in order to find contents present. Occasionally the stomach empties itself almost immediately. This is found true in cases in which diarrhœa is present, and also when there is constipation. The diarrhœa was sometimes increased by nervous excitement, sometimes by unusual exercise. From long familiarity with these patients I have the fixed impression that overgastric motility is the rule, and that for a time there is present what might be called a compensatory constipation. This may remain or it may terminate in a diarrhœa, often postprandial in character, the act of eating seeming to excite an unusual motor activity which, beginning in the stomach, continues to the colon. Several times I have predicted the diagnosis merely from the hurried emptying of the bowel after eating. Especially is this true as regards breakfast. Lesions of the intestines are comparatively unimportant.

Liver. In 33 cases the liver is recorded as diseased; in 23, large; in 6, small; induration in the region of the gall-bladder, in 2; tenderness around the gall-bladder, in 1.

Kidneys. The state of the urine was recorded in 80 cases, among which albumin was found in 33, tube casts in 16, overacidity in 19, indican in large amount in 13, alkalinity in 3, specific gravity relatively low in 21, amount decreased in 29, urea decreased in 40. This emphatically disposes of the statement once made that indican in the urine is the rule in low secretion of hydrochloric acid. The frequency in which albumin appears is somewhat remarkable. The lowered secretion both in the quantity of urine and in solids is interesting, but was to have been expected.

Gastro-intestinal Symptoms. Vomiting was complained of in 29 cases, sometimes at rare intervals, often at periods of a few days only; sometimes excited by a hearty meal or by very cold or very stimulating food. Nausea was present in 10 cases, regurgitation in 11, sour regurgitation in 6, eructation in 24, pain in 19, sense of soreness in 9, anorexia in 20, hyperorexia a number of times, gastric distress in 30, excessive salivation in 2. I have been unable to find any relation between achylia and ptosis or gastrectasis.

Gastric symptoms are sometimes very conspicuous. One patient had excessive vomiting for two years, bringing up large quantities of watery mucus. She said she felt sure there were two places where food went down. In one place it passed on, in the other it stopped; this she vomited. She suffered immediately after eating, especially meat, but she liked and, indeed, craved it. One of the most conspicuous stomach symptoms is appetite, which the patient is afraid to satisfy because of distress thus induced.

Mentality. There is a field for a perverted mental attitude in patients who know that they suffer from this condition, but that is true of all dyspeptics. One patient regularly wakes between twelve and three with distress and nausea. Then she regurgitates once or twice a tablespoonful of fluid, feels better, and sleeps until morning.

General Symptoms. Generally patients with achylia gastrica suffer from vague general symptoms, and, sometimes, at most, merely suspect the stomach. Complaint is made of unaccountable fatigue, occipital headache, myalgia, disturbed sleep, mental depression, arthralgia, loss of weight. Usually there is complaint about the intestines or liver. One interesting patient suffered from the stomach for years; five years ago he practised gastric lavage for three months. Following this he had severe fever for five weeks, probably typhoid. Two months later he began desquamating, and within a year had lost the nails of his hands and feet. Afterward his stomach symptoms were better until within the last seven months.

General weakness or depression was complained of in 33 cases, nervous excitement in 4 cases, nervous depression in 14, loss of weight in 24, arteriosclerosis in 24.

A biuret reaction was discovered sooner or later in 20 per cent. of the cases, although only in faint traces, except in two or three rare instances when real improvement occurred; rennin or rennet zymogen ultimately appeared in ten cases when formerly absent; free hydrochloric acid appeared ultimately in three cases, together with general improvement in secretion. Lactic acid was rarely present in large amount, but appeared at least in traces in 44 cases. More than a normal amount of mucus was present in 62 cases.

I have observed in several cases of complete achylia in women that the symptoms were all ameliorated and the general health improved during pregnancy and lactation. In one case there was persistent diarrhœa occurring after meals, accompanied with great distress, hunger which was cut short before the repast was completed, and a sense of general depression and fatigue. Objectively there was the appearance of poor health. All these disappeared during pregnancy, and the patient gained rapidly in weight and a good condition continued until she was delivered. This was repeated during three consecutive gestations, and at no time was there gastric secretion. In another case there was marked improvement during pregnancy which terminated by premature delivery. With this event the symptoms recurred and the gastric secretion remained absent.

It has been stated that gastric secretion recurred in some cases after a long period of treatment. The first case seemed typical except that it was accompanied by marked excess of mucus. In about a month's treatment the secretion returned sufficiently to show traces of free hydrochloric acid with biuret reaction. The case was probably secondary to gastritis.[1] A second case under observation for four years had complete absence of all evidence of secretion for one year, after which there was present a biuret reaction during about half of the time. Traces of combined chlorides were found. After three years free hydrochloric acid appeared and the digestion was in every way improved. This was a case of "lues in the innocent." The third case had been under treatment at intervals during two years and three months, at the end of which time he had a biuret reaction and free hydrochloric acid. The man subsequently gained very much in weight and general vigor. In this case also there was usually present too much mucus. I think these cases should all pass for instances of achylia gastrica. The general picture was quite unlike that of cases of subacidity which go on showing presence of emzymes and, from time to time, a faint trace of hydrochloric acid. I am not able to say that arteriosclerosis occurred in a greater proportion of cases than would be expected in the same number of other chronic diseases. It is noted as having been found very marked in 8 cases.

[1] Faber and Lange (Ztschr. für klin. Med., 1908, lxvi), reporting 56 cases, hold that in all cases of achylia gastrica we have to do with a gastritis or a degeneration of the secreting structure of the mucosa.

Stomach symptoms are not uniformly present in achylia gastrica. The proportion of cases in which this is true, I am unable to state. The symptoms are often inconspicuous, complaint being made merely of a sense of "goneness" or hunger appearing too soon after eating.

In the examination of stomach contents of supposedly healthy individuals, the gastric secretion has occasionally been found absent. Such cannot be counted as instances of achylia except after repeated examinations have been made. Anyone who has repeatedly examined many stomachs will be struck by the fact that those that promise to be achylia will show in a few days a normal or even an excessive secretion.

In some instances of achylia the stomach is resentful of all acids. It is at times difficult to administer hydrochloric acid even in small quantities to these patients; at other times this remedy gives distinct relief and seems to benefit the patient generally. It has seemed to me that the administration of pepsin has added to the comfort and well-being of these patients.

I am indebted to Dr. Myrtle L. Massey for the intelligent analysis of the cases upon which this paper is founded.

DUODENAL ULCER AND ITS TREATMENT.[1]

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Formerly duodenal ulcer was looked upon as a rare disease, which could hardly be recognized during life. When chronic ulcers of the stomach began to be treated surgically, duodenal ulcers were often met with at the operation. It was shown that, in many cases in which the ulcer was believed to be in the stomach, it was found in the duodenum. Pagenstecher[2] justly laid stress upon the importance of surgical treatment of duodenal ulcer. In 1899 he reported several cases of ulcer of the duodenum, which were treated successfully by him by gastro-enterostomy. Moynihan[3] points out the difficulty of the diagnosis of duodenal ulcer, and says: "The symptoms of duodenal ulcer are chiefly characterized by their lack of ostentation. In more than half of the cases in which ulceration is found at the necropsy, symptoms have never been present. In 151 cases collected by Perry and Shaw, in which duodenal ulceration was found post-

[1] Read at a meeting of the American Gastro-enterological Association, Atlantic City, N. J., June 5, 1909.

[2] Die chirurgische Behandlung des Duodenalgeschwürs. Deutsche Zeitsch. f. Chir., 1899, p. 541.

[3] On Duodenal Ulcer and its Surgical Treatment, Lancet, 1901, p. 1656.

mortem, there were 91 in which there were no antecedent symptoms." Treatment should be medical. Surgical treatment is in order: first in perforation; second, when abscesses develop; third, when pain and hemorrhages occur persistently; and fourth, when stenosis associated with dilatation of the stomach develops. The same author, in conjunction with Robson,[4] expresses himself in a similar manner: "Duodenal ulcer very frequently fails to yield to general treatment, and from our experience of these cases we believe it is much more frequently the cause of fatal symptoms than is generally recognized. In hemorrhage from the stomach or duodenum, as shown by hematemesis or melena, where general treatment has been tried and failed, and where the bleeding is persisting, or recurring after brief intervals, operation is advisable with a view to find and to secure, if possible, the bleeding point or points, or of performing gastroenterostomy in order to obtain physiological rest, and thus to favor natural hemostasis."

Mayo[5] calls especial attention to the great frequency of duodenal ulcers. In his 193 cases operated on for ulceration, he found stomach ulcer in 60 patients, duodenal in 119, and both combined in 14. As contrasted with ulcer of the stomach, duodenal ulcer is found more frequently in men than in women; according to Mayo, men furnish 73 per cent., women 27 per cent.; according to Robson, 86 per cent. and 14 per cent. respectively. Mayo tries to explain this as follows: "The curve of the duodenum in men is usually higher than in women; that is, the first portion of the duodenum in men is nearly always ascending, while in women it is often transverse. It seems probable that, for mechanical reasons, the alkaline secretions of the liver and pancreas more readily neutralize the acid chyme in the upper duodenum in women than in men."

Robson[6] agrees with the great frequency of duodenal ulcers. He says: "Operative experience shows that in a large proportion of cases duodenal and gastric ulcers co-exist, and that the real proportion of duodenal to gastric ulcers is 1 to 2 or thereabout." Regarding treatment, Robson says: "While gastric ulcer is distinctly amenable to medical treatment in the earlier stages, it is questionable if, when once peptic duodenal ulcer has passed to the stage of being recognized by definite symptoms, it is ever really permanently cured by medical treatment alone." Referring to a material of 66 cases of duodenal ulcer operated on by himself without a single death, Robson tries to define its symptomatology. There is usually pain two to three hours after meals. It is due to the irritating effect of the acid on the open ulcer, and partly to spasm of the pylorus and to dragging on adhesions which are often present.

[4] Diseases of the Stomach and their Surgical Treatment, New York, 1904.

[5] The Contributions of Surgery to a Better Understanding of Gastric and Duodenal Ulcer, Annals of Surgery, 1907, p. 810.

[6] Duodenal Ulcer and its Treatment, Brit. Med. Jour., 1907, p. 248.

The following symptoms are more or less characteristic: Intermittency of the attacks; dilatation of the stomach during the exacerbations due to pyloric spasm; hyperchlorhydria; vomiting not frequent; hematemesis or melena; and perforation in 10 per cent. of the cases. Surgical treatment consists in excision of the ulcer or an infolding of the duodenal wall at the site of the disease, or gastroenterostomy. The latter forms the rule.

Deaver and Ashhurst[7] agree with Robson with reference to the symptomatology of duodenal ulcer; they place the frequency of perforation at 25 per cent. M. Jaffe[8] mentions two cases of duodenal ulcer with perforation. Both were operated upon, one recovering and the other dying. In neither of them were there any symptoms of ulceration; perforation was the first sign of the disease. There was rigidity of the right rectus muscle, very intense pain, and tympanites in the region of the liver. The ulcers are usually small, about the size of a quarter dollar.

Whereas all of the mentioned papers were written by surgeons, a few important contributions have been published lately by clinicians. Kuttner[9] describes two cases of duodenal ulcer in children in the first decennium, the one being four weeks old and the other four years. Both died of gastric and intestinal hemorrhages. Autopsy showed ulcers in the horizontal part of the duodenum. Kuttner advises in all cases, when medicinal treatment is inefficient, to think of surgical intervention. Pewsner[10] reports a case of duodenal ulcer with autopsy. During the disease there existed at first a digestive and later a continuous hypersecretion of gastric juice. There was no pain on pressure in the epigastrium, nor was there any connection between the taking of food and the pains. Before death a severe hemorrhage occurred. At the autopsy an ulcer, the size of a dollar, with thickened margins was found on the posterior wall of the duodenum. No signs were found microscopically of a neoplasm.

Until lately a positive diagnosis of duodenal ulcers could be made only on the operating or postmortem table, as the clinical symptoms were not sufficiently marked to indicate the condition with certainty. Since I[11] have used the thread test I believe the diagnosis of duodenal ulcer can be made clinically with some certainty. I therefore consider it advisable to describe all the cases of duodenal ulcer which I had occasion to observe during the last year and in which the thread test was employed.

Case I.—Nathan B., aged thirty-three years, has been suffering for about eight years from frequently recurring attacks of gastralgia

[7] Surgery of the Upper Abdomen, 1909, p. 200.

[8] Durchbruch von Magen- und Duodenalgeschwüren, Berl. klin. Woch., 1908, p. 346.

[9] Ueber das Vorkommen von Ulcus duodeni im ersten Decennium, Berl. klin. Woch., 1908, p. 2099.

[10] Ueber einen Falle von Ulcus duodeni, Arch. f. Verdauungskr., 1908, xiv, 645.

[11] A New Method of Recognizing Ulcers of the Upper Digestive Tract, Med. Record, 1909, p. 549.

about two to three hours after meals, and severe constipation. The attacks last one to two months and alternate with intervals of freedom from pain. In October, 1908, the patient vomited a large quantity of blood (half a quart). On November 25, 1908, he was examined by me. The thoracic organs were intact, the stomach was not dilated, palpation revealing no painful area. The stomach bucket showed one hour after test breakfast: HCl + diluted twelve times; in other words a marked hyperchlorhydria. On November 26, 1908, the duodenal bucket was allowed to remain in the digestive tract over night, and was removed early on the twenty-seventh in the fasting condition. The bucket was filled with a yellowish bile-like fluid; reaction alkaline. The thread was colored golden yellow from 53 to 73 cm. and reddish brown from 61 to 62 cm. A similar result was found with the duodenal bucket on December 1, 1908—a reddish brown coloration of the thread between 61 to 62 cm. The patient was given alkalies, and his condition improved.

Case II.—H. L., aged thirty-eight years, has suffered for ten years from attacks of pain occurring once or twice yearly in the region of the stomach, associated with nausea, sometimes with real vomiting, and lasting from several days to two weeks. The attack usually begins suddenly at midnight; the patient awakes and complains of nausea. During the period of attack considerable stomach symptoms are present, particularly two to three hours after meals, together with heart burn, frequent eructations, and the raising of water brash. The patient usually feels rather weak during the attack, and must generally remain abed the first few days. In the intervals complete euphoria exists. He never had jaundice. The stool is generally regular, rarely constipated. Physical examination showed cardioptosis, hepatoptosis, atony, and ptosis of the stomach. The stomach contents one hour after test breakfast showed: HCl +: total acidity = 85; no occult blood. Palpation of the abdomen showed only slight sensitiveness to pressure toward the right of the linea alba, in the region of the liver and below. The duodenal bucket was twice used, the patient retaining it over night. The first test on February 11, 1909, showed, on withdrawing the bucket, slight resistance for about 15 cm., then no resistance until the entrance of the œsophagus was reached. The bucket was filled with bile; reaction alkaline; steapsin present; at 66 cm. from the knot a trace of brownish coloration for about 1 mm. was visible. The second test was made February 21, 1909, with the following results: thread colored yellow at the bucket; thread distinctly reddish brown, from 59 to 62 cm. Contents: HCl trace; after neutralizing steapsin and trypsin were found. The patient was told to wear an abdominal supporting bandage, and was given alkalies and olive oil twice daily. He felt better and gained somewhat in weight.

Case III.—J. H. S., aged forty years, traveller, has had no syphilis; is a moderate user of alcohol and tobacco. He has always been

well until five years ago. He then began to suffer from attacks of indigestion, distress in the stomach, constipation, and wind, all of which would be relieved by a movement of the bowels or by carbonated sodium. About two years ago he had a hemorrhage from the bowel (the blood was tarry). This had been preceded about two weeks by weakness, distress, and malaise. He did not observe any special diet, and felt well for about one year, when he had a second hemorrhage (melena), which was accompanied by pain two inches above the navel rather than in the pit of the stomach, as in the former attack. The quantity of blood lost on several successive occasions at that time was not very large. He was then very careful as to his diet (raw eggs, milk, and bread and butter, no meat). He had pains about two to four hours after eating, never immediately after meals. He finally got well. About November 1, 1908, he had another attack, which lasted until I saw him (February 2, 1909.) He then had the pain again in the epigastrium and not over the navel. His pain was never constant, and was relieved temporarily by eating, but returns again in one to two hours. Bowels have always been constipated. The physical examination of the chest and abdominal organs did not reveal anything abnormal. Palpation of the gastric and epigastric regions was not painful. One hour after test breakfast the gastric contents showed: HCl +; acidity = 115; no occult blood. On February 4 the patient was given the duodenal bucket for the night; on February 5 the bucket was removed in the fasting condition of the patient. The thread length was 65 cm. It was brownish at the bucket and between 58 to 59 cm. The contents showed free HCl +; no bile. Rectal alimentation and then liquid diet were advised.

Case IV.—B. F. M., aged about fifty-three years, was seen on February 1, 1909; had ten years ago a very severe attack of indigestion, causing spasms of pain which lasted several days. After about a week or ten days he suddenly developed jaundice, which, however, disappeared in twenty-four hours. About two days after that he had a slight paralysis, which left him speechless for twelve hours, but he rallied, and after a few months was as well as ever. Two years later he had a very similar attack, without jaundice and with no other after-effect. Four years later he again had an attack of indigestion just as severe, but with none of the other ailments. For the past two years he has had trouble with his stomach, which seems to increase. He has colicky pains, which generally leave him with a feeling of exhaustion. They come and go at all times, but seem to occur mainly about two hours after meals and to get less when patient eats something. They start about four inches above the navel. They generally go all the way around to the kidney, and while never very severe and nothing like as severe as eight to ten years ago, still they are very annoying and disturb patient considerably. They interfere with his appetite, and patient has lost about ten

pounds in weight in the last six months, weighing now one hundred and eighty pounds. He has not smoked for ten years, although he was formerly an excessive smoker, from fifteen to twenty cigars a day. He is a very moderate drinker, however, sometimes not taking alcohol in any form for two or three months. He also gave up coffee and tea on account of the tannin contained in them, which he thought was injurious to him. Nothing abnormal was found by the physical examination. One hour after the test breakfast the gastric contents show HCl +; acidity = 105; no occult blood; no stagnation of food. The duodenal bucket was twice applied on February 3 and 5, 1909. The thread showed a reddish brown discoloration between 66 to 59 cm. on February 3, and similar discoloration at 55 to 61 cm. on February 5.

Case V.—Moses O. S. aged about thirty-four years; salesman; used to be a moderate drinker; never smoked; never had syphilis. In 1900 he was taken sick with diarrhœa, and had to go to the toilet six to seven times daily, usually after eating. He had a pressing down sensation in the rectum and intestines, as though he wanted to go to stool. He was treated off and on until 1902, when he came to me. After some treatment he improved and went out West. In 1905, however, the diarrhœa returned; about two to three times weekly he would have loose bowels. He has a beating sensation in the bowels, as though his heart was in his bowels. Then he has a dull aching pain in the hypogastrium, as though his food had not properly digested. He has an anguished feeling in the stomach as if a lot of hydrochloric acid were rolling around in it. This seems to gather there about two hours after he has eaten, and gives him distress. It lasts until he eats again. He frequently has a passage that looks like goat or sheep manure, little hard round lumps. He sleeps well and has a good appetite. The examination showed that the stomach extends to about a hand's width below the navel. The right kidney is slightly movable. One hour after the test breakfast the gastric contents show: HCl +; acidity = 80. The duodenal bucket was twice applied (January 26 and March 30, 1909). The first test showed a distinctly reddish brown discoloration of the thread between 61 to 66 cm. The second test, which was taken two months later, revealed a trace of brownish discoloration of the thread at 65 cm. The patient was treated with alkalies and was advised to wear an abdominal supporter. He has gained in weight ten pounds in the last two and one half months, and feels considerably better.

Case VI.—J. F. H.[12]

Epicrisis. In all six cases the diagnosis of duodenal ulcer was made by the thread test. In four the symptoms pointed to duodenal ulcer with some probability; in two, however, the symptoms were in no way characteristic. In one patient who was operated

[12] See Medical Record, April 3, 1909.

upon the ulcer was found in the duodenum, one inch beyond the pylorus, as diagnosticated. Regarding the frequency of duodenal ulcer, the surgeons (Mayo-Robson and others) maintain that the proportion of gastric to duodenal ulcer is as 3 to 2, and according to Mayo even as 2 to 3, whereas pathologists find at autopsies a proportion of 10 to 1. This seeming contradiction can be easily explained by the fact that the surgeons see only those cases in which an operation is necessary. Among these the ulcers at the pylorus or in its immediate vicinity, as they lead to disturbance of motility of the stomach, occupy the first place. Therefore, the proportion of duodenal ulcers must be high. Most gastric ulcers not situated at the pylorus are cured (cicatrization) without surgical intervention. These ulcers or cicatrices are then found only at autopsy.

According to my own experience gained with the thread test, the proportion of gastric to duodenal ulcers is about as 4 to 1; of course, the proportion must be still higher in favor of stomach ulcers, as those situated at the fundus cannot be demonstrated by means of this test.

Symptomatology. In all six patients a marked hyperchlorhydria was present. In Cases III and IV there also was a slight continuous hypersecretion of gastric juice. Slight peristaltic restlessness of the stomach was found in only one (Case VI). Pains are generally present two to three hours after meals, and are alleviated by the ingestion of food. Moynihan calls this symptom "hunger pain." In nearly all the patients, periods of suffering alternate with periods of perfect euphoria. Hemorrhages (hematemesis or melena) were found in two of the cases. Usually constipation is present, but a tendency to diarrhea was found in two. Pain on pressure was absent in nearly all the cases.

Diagnosis. The symptoms just enumerated (except hemorrhages) are all caused by hyperchlorhydria and frequently occur without the presence of duodenal ulcer. If, however, in association with the above symptoms hemorrhages in the digestive tract occur, the diagnosis of duodenal ulcer is justified—for the real gastric ulcers generally act differently: pains immediately after meals, pressure pains in the epigastrium, etc. The diagnosis appears to become more positive if the thread test shows a distinct blood spot at a distance of 58 to 66 cm. from the teeth.

Treatment.—The treatment should at first be medical. In the mild cases regulation of the diet (frequent meals, abstention from highly seasoned substances, acids, and too fatty foods), improving the general condition by means of iron, arsenic, cold sponging, good air, avoidance of bodily exercise, and the use of alkalies are sufficient to effect a considerable amelioration, if not a cure. In several cases olive oil (two tablespoonsful morning and evening) seemed to be of service.

In graver cases of duodenal ulcer (hemorrhages, severe pain, etc.) a strict ulcer cure with rest in bed and rectal alimentation, and afterward fluid diet, must be instituted. In these cases large doses of magnesia and bismuth are of benefit: calcined magnesia, 0.5 (8 grains) bismuth subnitrate, 2 (30 grains), in powders—one powder three times a day, a half hour before meals. If a strict rest cure has been unsuccessful, or if we have to deal with severe hemorrhages endangering life, and returning frequently, or if obstinate spasm of the pylorus occurs, associated with severe pains in the pyloric region and slight peristalic restlessness of the stomach, an operation (usually gastro-enterostomy) is indicated.

In duodenal ulcer the clinician must advise surgical treatment sooner than in gastric ulcer, as the former, through its complications (hemorrhages, perforations, stenosis of the pylorus), endangers life much more readily than the latter. Gastro-enterostomy in these cases is fortunately attended with good results. The ulcer will then soon heal, as the gastric juice does not longer flow over the ulcerated surface in the duodenum or irritate it. At all events, the dangers of hemorrhage, perforation, and pyloric stenosis are thereby prevented.

CLINICAL EXPERIMENTS WITH HOMOLOGOUS VACCINES IN THE TREATMENT OF SEPTIC ENDOCARDITIS AND PYEMIA.[1]

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Following the method of Sir Almroth E. Wright[2] of the injection of homologous or autochthonous vaccines, I have treated a series of cases of septic (streptococcal) endocarditis and pyemia with results which have again demonstrated its effectiveness, and afford strong hope of greater success when the technique shall have become better understood. Of the series herein reported, three cases of septic endocarditis were cured, with one of serious pyemia, and in several other cases of septic endocarditis there was clinical evidence that the septic process had been completely controlled, although subsequently death ensued from such complications as tuberculosis or pneumonia, which as yet are beyond the influence of vaccine treatment.

In several of the cases polyvalent vaccines were employed, but without benefit, before homologous vaccines could be obtained, which latter proved effective. In fact, in no case in which the homol-

[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 11, 1909.

[2] Trans. Harvey Society of New York, 1907.

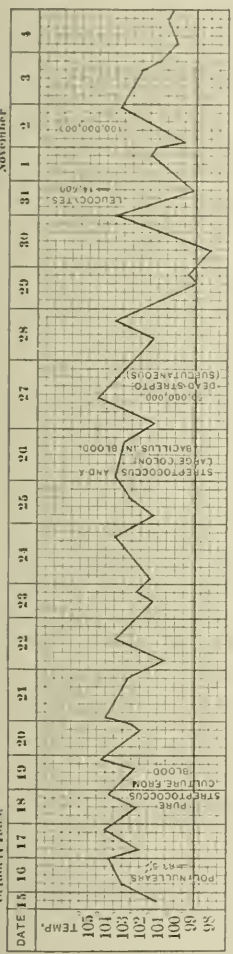
ogous vaccines were used was there failure to produce some degree of reaction, such as a downward temperature movement and other indications of at least temporary betterment, and several patients who came under treatment after months of illness, in a condition in which a fatal issue seemed imminent, gave evidence of an arrested progress of the disease for many weeks.

Case I.—*Septic Endocarditis.* The patient, an Italian laborer, aged thirty-four years, had been ill for a month before he entered the Presbyterian Hospital. He became suddenly ill with rigors, prostration, and fever having a daily periodicity. On admission the heart was found enlarged an inch to the left, with a loud, rough, booming apical systolic murmur. A similar murmur appeared subsequently over the aortic valve. Numerous petechiæ were present, and there were subcrepitant rales at the base of both lungs. The murmurs increased in intensity, and three weeks before death the lower lobe of the left lung became consolidated and fluid was aspirated from the pleura. A pure streptococcus pyogenes aureus culture was twice obtained from the blood, being once associated with a large colon bacillus. The percentage of polynuclear cells in the blood was 83.5, and the leukocytes numbered 14,600. Although the patient died forty-four days after admission, his temperature (Chart I) illustrated strikingly the influence of increasing doses of the vaccine derived from the first blood culture, which caused a total fall of 11° (from 104.5° to 93.5° F.) Recovery was impossible from the fact that, in addition to the vegetative endocarditis observed at autopsy, there were present acute nephritis, chronic pulmonary tuberculosis, pleurisy, and infarct of the spleen. Examination of the heart showed fine vegetations on the free border of the left anterior aortic cusp, on the ventricular surface; the posterior cusp was retracted and thickened along the free border, and presented a large mulberry vegetation, 1 x 2 cm.; on the corresponding aortic surface of this cusp were two cul-de-sacs which extended into the vegetation. The other valves and the coronary arteries were normal.

The patient received four subcutaneous vaccinations at intervals of six days, containing in series, 50,000,000, 100,000,000, and two of 200,000,000 dead streptococci. For the ten days preceding the first vaccination the temperature had ranged steadily between 101.5° and 104.5° F. The first vaccination of 50,000,000 reduced the temperature in thirty-six hours from 104.5° to normal, and kept it normal and slightly subnormal for eighteen hours. It rose again to 103.5°, when the second vaccination brought it gradually down in two days to normal and subnormal. It rose again until it reached 105° F., when the third vaccination of double the previous strength again reduced it in thirty-six hours to normal, and finally subnormal, where it remained until the fourth vaccination.

Up to this time there appeared to be improvement in the patient's condition, commensurate with the decline in temperature. It was

determined, however, to give the fourth vaccination, but for the fourteen succeeding days before death the temperature remained markedly subnormal, and on one occasion fell as low as 93.5° F., as stated above. During this time the patient's pulmonary and pleural symptoms made serious advance, and he became rapidly weaker. It may have been an error to have given so large a final



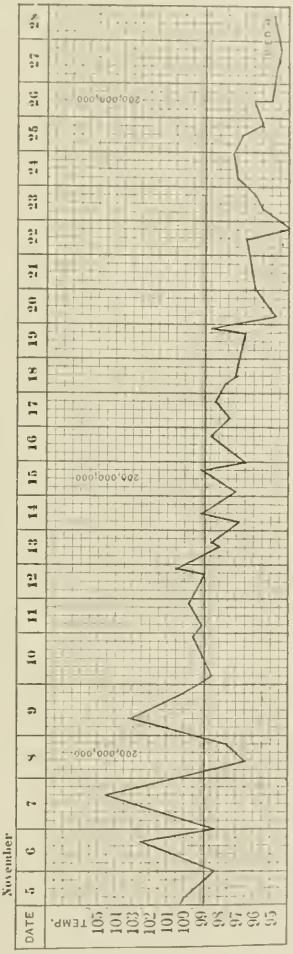


CHART I.—Streptococcic endocarditis, showing the effect of increasing doses of vaccine (Case I). Death from tuberculosis and nephritis.

vaccination, but if so it was due to lack of experience with an entirely new method, although the probability remains that death was more directly caused by the severe nephritis, tuberculosis, and the extreme damage to the aortic cusp.

CASE II.—*Septic Endocarditis.* This was likewise an unfair test of any treatment, owing to the great number of complicating

maladies, yet it illustrates, in less striking manner, the influence of the vaccines on temperature. The patient, an Italian, aged forty years, had been at work in apparent health, until three weeks before admission to the Presbyterian Hospital, when he was attacked with paroxysmal rigors and fever. He felt somewhat better on alternate days, but was always feverish. One week before admission the calves of both legs became swollen and painful, presumably from neuritis, and palpitation was complained of. On admission there was no cardiac murmur, but later a loud aortic systolic murmur developed, and the apex was found in the fifth intercostal space 11.5 cm. to the left of the midsternal line. The leukocytes numbered 19,300, and the polynuclear percentage was 86.7. It is worth noting that as the case progressed and before treatment began, the leukocytes spontaneously dropped to 10,000 and the polynuclear count to 81 per cent. Two blood cultures gave pure streptococci. For some days there was a daily severe chill that accompanied great fluctuations in temperature, which on one occasion amounted to 9.5° F. (from 96° to 105.5° F.). Four weeks after admission a subcutaneous vaccination of 50,000,000 dead streptococci was given, followed in six days by another of twice that amount. The first vaccination kept the temperature at or below the normal for two days, and the second was followed by three days of normal temperature, when death ensued. There were so many lesions observed at autopsy that this case, like the preceding one, must have been doomed from the commencement. These lesions were: Senile emphysema with pulmonary gangrene; chronic interstitial nephritis and myocarditis; cholelithiasis; chronic pancreatitis; acute perisplenitis; early cirrhosis of the liver; abscesses of abdominal lymph nodes; white infarcts of the spleen, and red infarcts of the kidney. The aortic valves presented acute and subacute vegetative endocarditis, and smears from the vegetations gave streptococci. In this case the vaccines unquestionably controlled the temperature, but, owing doubtless to the numerous complications present, produced no other effect.

Case III.—*Septic Endocarditis.* The patient was a railway guard, aged thirty years. Two weeks before admission to the Bellevue Hospital he became acutely ill with a sore throat, chills, sweating, headache, slight cough, and mucous expectoration. On admission no cardiac murmur was heard, but a fortnight later a loud, harsh, systolic aortic murmur developed and persisted. During the first three weeks that the patient was under observation he exhibited an exceedingly irregular temperature (Chart II), which several times rose to 106° F., and on one occasion fluctuated 9° F. in one day (from 97° to 106° F.). During this period, also, five blood cultures were made, three of which gave pure stains of Streptococcus pyogenes aureus, but it is interesting to remark that the first and fourth were sterile. The leukocytes numbered 16,000, and the polynuclear

percentage was 84. During the succeeding four weeks ten subcutaneous vaccinations were given at varying intervals, some of

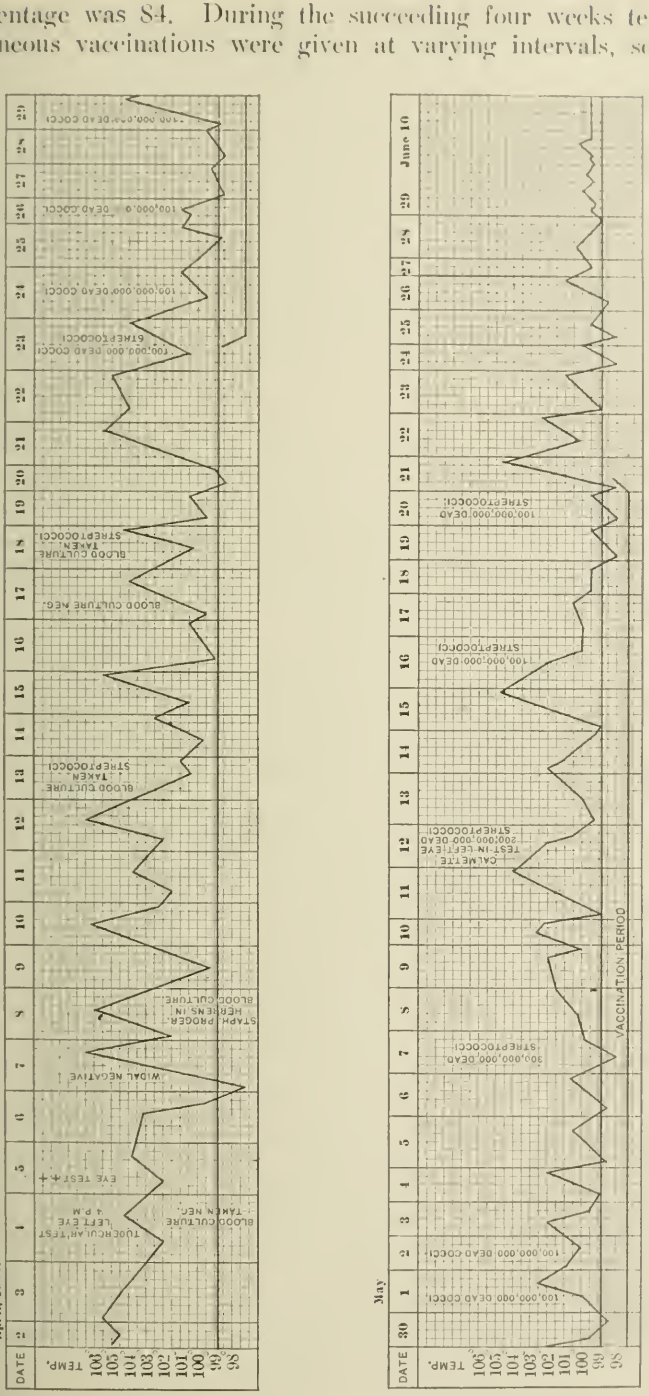


CHART II.—Streptococcic endocarditis cured by vaccination (Case III.)

them on succeeding days, others at intervals of four or five days. In 8 of the vaccinations 100,000,000 dead streptococci were given, derived from the patient's own blood cultures. In one instance 300,000,000 and in another 200,000,000 were given. Thus, in all, the patient received 1,300,000,000.. The injections had remarkable effect upon the temperature, reducing it within two days from 105° F. to normal, and repeatedly reducing it when it tended to rise again. It never became so markedly subnormal as in the two preceding cases. There was coincident improvement in all the symptoms. Having been apparently dying, the patient gained in strength and weight, the leukocyte count dropped to 8000, and the polynuclear percentage to 53; the temperature finally remained normal and recovery became complete, with a gain in weight of thirteen pounds.

Case IV.—*Septic Endocarditis.* The patient, a girl, aged seventeen years, had had a septic temperature for more than five months, with occasional chills and sweating, when first seen by me in consultation. A loud, harsh, mitral systolic murmur had developed after a few weeks of illness, and petechiæ were present. Despite the apparent good digestion of ample food, emaciation was extreme, and reached a degree rarely equalled in the young excepting in protracted cerebrospinal meningitis. At my suggestion a blood culture was made by Dr. Hastings, and it proved to contain Streptococcus tenuans. This germ, injected in a mouse, caused death in three days, with extraordinary emaciation, so that the animal appeared to be merely skin and bones. A vaccine made from the blood culture was given repeatedly in doses of 13,000,000 to 20,000,000. The dosage was small because of the feebleness of the patient and the difficulty of giving hypodermic injections to one so emaciated. About two dozen vaccinations were given, at first one every other day, and later one every day. The effect on the temperature, doubtless owing to the small dosage, was less spectacular than in the three previous cases, but the vaccinations steadied the temperature, that is, converted it from a markedly septic type with wide daily excursions to a continued type with a gradual favorable downward trend. The heart action also became steadier and less tumultuous, the systole being better sustained. On one occasion the temperature fell to normal for the first time in a month after vaccination, and there was a coincident fall in the pulse rate from an average of 120 to 94. A blood culture made after several vaccinations had been given, showed a marked reduction in the number of germs present. It is conceivable that in this case the refractory temperature was in part due to tissue starvation, for I have rarely seen greater emaciation. Normal heat regulation in such a case naturally would be disturbed. The tendency to emaciation and asthenia, however, did not yield to the vaccination treatment, and after two months the patient succumbed.

Case V.—*Septic Endocarditis.* The patient was a young woman, aged nineteen years, pregnant on admission to the Presbyterian Hospital. On admission she was very weak and emaciated, and she exhibited a septic temperature which ranged from 97° to 103.5° F., progressive anemia, petechiæ, and a loud, harsh, but variable systolic murmur over the base of the heart. Polynucleosis reached 89 per cent., but the leukocyte count was never high, the maximum being 10,300, with subsequent spontaneous fall to 6000. Two blood cultures were negative, and 5 injections of 10 c.c. each of Parke Davis' antistreptococcus polyvalent serum produced no effect of any kind. A third culture gave Streptococcus pyogenes aureus, from which a vaccine was made by Dr. Meakins. Two vaccinations of 100,000,000 each and one of 200,000,000 were given at intervals of four days. The effect on the temperature resembled that in the previous case, that is, the maximum was lowered a degree and the course became steadier and distinctly less septic. Shortly after the second vaccination the patient was delivered of a seven months' feeble infant, which lived about twenty-four hours. I was interested to learn whether this infant's blood was infected, but a culture proved sterile. Soon afterward the mother died, the vaccinations having proved of merely slight temporary benefit.

Case VI.—*Septic Endocarditis.* The patient was a young married woman, aged twenty years, who entered Bellevue Hospital with a septic vaginal discharge two weeks after an abortion. She had a septic temperature ranging from 101° to 103° F., and was very feeble. There was a general neuritis involving both arms and legs. She was curetted and a vaccine was made from the uterine streptococci. Subsequently large abscesses were opened in the left ankle, left hand, and both elbows. A second vaccine was made from one of the abscesses. The patient developed a faint pulmonic systolic and a presystolic mitral murmur while under observation. A few petechiæ appeared. After several vaccinations were given, the patient showed decided improvement and streptococci disappeared from the pus in the last abscess. In all, eight vaccinations were given, in quantities varying from 45,000,000 to 150,000,000 dead streptococci. Four blood cultures proved negative. After five weeks of fever the temperature began to decline, and a week later it became normal and so remained, while the patient completely recovered excepting the persistence of the heart murmur.

This case may fairly rank as a septic endocarditis, for, despite the negative blood cultures, streptococci were obtained from the uterus and abscesses, and the heart murmurs and petechiæ developed under observation.

Case VII.—*Septic Endocarditis. Recovery from Sepsis; Subsequent Death from Pneumonia.* For the following abstract of an additional case observed at the Lincoln Hospital I am indebted to

Dr. Walter L. Niles, who followed the case in the service of Dr. N. R. Norton:

The patient was a Greek boy, aged sixteen years, who, two weeks before admission to the hospital, was taken ill acutely with palpitation, dyspnœa, œdema of the feet, and lumbar pain. Under observation he developed a septic temperature which lasted three weeks until controlled by vaccine. There was a diastolic and systolic aortic murmur and a systolic mitral murmur. Pericarditis with effusion required aspiration. Blood culture revealed Staphylococcus aureus. There was a leukocytosis of 18,000 and a polymucleosis of 91 per cent. In all, 10 vaccinations with homologous vaccine were given in periods varying from one to three days, and doses varying from 100,000,000 to 725,000,000. The vaccinations produced remarkable response in lowering the temperature, the largest dose causing a fall in two days of 8° F. (from 103° to 95° F.).

After sixteen days of vaccination treatment, the temperature remained normal and subnormal for a month, and the notes of the case record "clinical picture entirely changed; patient eats well, sleeps well, feels well; wants to get up." The blood cultures became negative and the patient was evidently cured of the septicemia. Unfortunately, at the end of the month he acquired pneumonia and died.

Case VIII.—*Pyemia.* The patient was a railway employee, aged thirty years, with pyemia. Ten days before admission to the Presbyterian Hospital the patient burned off a "wart" on his right hand with caustic. The wound became infected with staphylococci and the hand greatly swollen. Bronchopneumonia ensued, for which the patient sought hospital relief. The bronchopneumonia ran the usual course, but defervescence was interrupted by a septic temperature which lasted three weeks, with a maximum rise to 105° F. and daily fluctuations of 3° F. (Chart III). The right hand and wrist had to be incised twice. Shortly after admission the left hand became the seat of purulent infection, and likewise was incised. On the fifteenth day septic arthritis of the right knee-joint developed, with extensive fluctuation. On the nineteenth day pyonephrosis was discovered, and on the twenty-seventh day a rapidly spreading diffuse abscess formed over the upper left side of the chest wall and shoulder, which soon contained a pint of pus. I invited one of the visiting surgeons to open it, but the patient was so extremely weak that the surgeon said death would probably occur within a few hours, and thought it useless to operate. I therefore directed my house physician to insert an aspirating needle and remove what pus he could. The next day vaccine treatment was begun, and such prompt improvement followed that the surgeon consented to open the shoulder abscess, and evacuated a dozen ounces of pus. The knee-joint had been previously aspirated. I was embarrassed in obtaining a vaccine for this patient, because the first blood culture was sterile. In all,

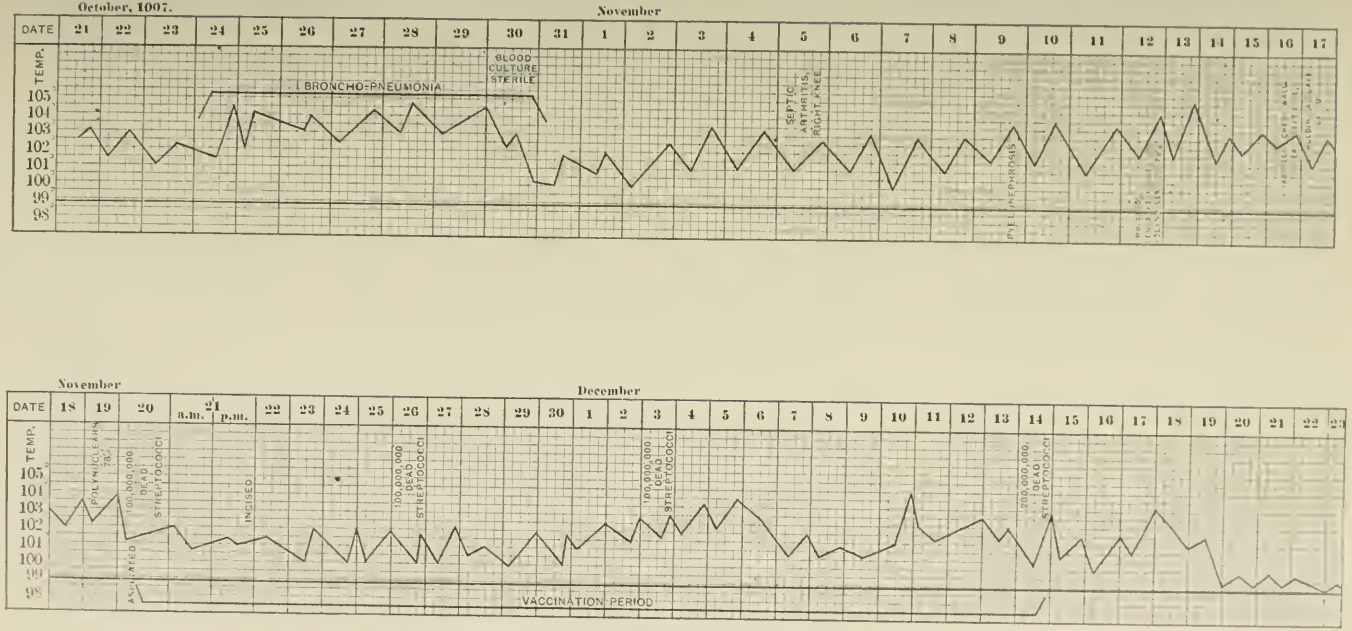


CHART III.—Pyemia; recovery after vaccinations (Case VIII).

six cultures were attempted. The third and fourth only showed Streptococcus pyogenes aureus, the four others being sterile. Four subcutaneous vaccinations were given at weekly intervals, three of 100,000,000 each and one of 200,000,000. Immediately after the first vaccination the temperature, which had averaged 103° to 103.5° F., fell 2°, and remained below 101.5° for three days, while the patient showed great improvement in every way. Subsequent slight elevations were controlled by the other injections until, after a total of two months of fever from the commencement of the illness, and one month from the first vaccination, the temperature remained normal, and recovery was complete excepting the damaged knee-joint. The patient finally left the institution with only moderate stiffness of the joint. The *x*-rays showed slight destruction of bone. Six months later I saw the patient digging a trench in the street.

In this patient the maximum leukocytosis was 18,800, with a polynucleosis of only 78 per cent., which latter subsequently rose to 87 per cent.

In another case of malignant endocarditis I was unable to obtain any vaccine, for four successive blood cultures were sterile, and there was no localization of pus. I therefore again employed in 10 c.c. doses the Parke-Davis polyvalent serum, made from virulent cultures of Streptococcus erysipelatus, but without benefit, and, in fact, without reaction of any kind. This patient was a youth, aged twenty years, who had a septic temperature ranging from subnormal to 105° F., anemia, emaciation, petechiæ, leukocytosis, varying between 45,000 and 20,000, and polynucleosis of 88.6 per cent. He developed a loud mitral systolic murmur while under observation, which autopsy showed was due to a circumscribed ulcer surrounded by a ring of vegetations and situated upon the lesser cusp of the mitral valve. The aortic valve was normal.

J. Barr[3] reports a case of malignant endocarditis with streptococcic blood infection, cured by vaccine treatment. Antistreptococcic serum was previously employed without effect, as in two of the cases above described.

There are certain diagnostic features observed in malignant endocarditis, illustrated by the cases previously cited, to which I would direct attention:

1. The observation of petechiæ is most important. While usually present first upon the legs, they may occur where they are easily overlooked. In three of my cases they were first seen on the lower eyelid, close to the sclerotic, and on the hard palate.

2. The ordinary blood count should not be too much relied upon in diagnosis, for it may prove misleading or variable, and may apparently improve as the patient grows worse. In one case of this series the leukocyte count fell spontaneously from 12,000 to 6000.

[3] Lancet, February 23, 1908.

I have records of several fatal cases in which the polynuclear count was below 60 per cent., and in one it was 53 per cent.

3. A negative blood culture should not prejudice against the diagnosis of septic endocarditis. In Case III herewith reported, two out of five cultures were sterile. In Case V, two out of three cultures were sterile, and in Case VIII, four out of six were sterile. In a fatal case, in which streptococci were obtained from vegetations upon the mitral valve, four blood cultures had previously proved sterile.

4. The character of the heart murmur is often distinctive. Usually most intense over the aortic region, it may be transmitted to the mitral area, or may be heard over the latter area first. It grows rapidly louder, and is of a harsh booming character when fully developed. It is usually accompanied by dilatation, and the pulse may be of the Corrigan type.

5. Anemia is progressive and extreme, and emaciation may become very pronounced, although digestion is seemingly unimpaired and the patient may take abundant food.

6. Peripheral neuritis with pain, tenderness, and sometimes swelling is not uncommon in the extremities, independent of abscess formation in the joints.

7. The temperature is characterized by extreme irregularity, and there are few conditions in which such wide daily fluctuations are encountered. In two of the cases above detailed, this variation amounted to more than 9° F. within half a day, and in another it was 8° F. On the other hand, the entire lack of septic temperature or petechiæ may lead to failure of diagnosis, as illustrated by the following case: The patient, a man, aged thirty years, entered the hospital nearly moribund from intense dyspnœa, cyanosis, and general anasarca. His heart was greatly hypertrophied and dilated, and there was a loud, harsh, mitral systolic murmur, with a fainter mitral presystolic, and an aortic stenotic murmur. The leukocytosis was 22,000. The case appeared to be primarily one of mitral regurgitation with intense chronic cyanosis, which had lasted several months. During the patient's life of three weeks in the hospital, the temperature never rose above 100°, nor did it vary in all more than a degree and a half. He died suddenly when he seemed to be improving, and the autopsy showed on the aortic cusp vegetations as large as a raspberry, with thickening and ulceration, and there were a few discrete pediculated vegetations on the larger cusp of the mitral valve, which yielded a pure streptococcus culture. Unfortunately no blood culture was made during life.

The safety and usefulness of the homologous vaccine treatment has been demonstrated by Wright, in whose original report[4] two cases of septic endocarditis were cited in which the temperature was markedly

[4] Loc. cit.

influenced, although the patients died. Much depends upon technique, the proper dosage, and intervals of dosage, as well as upon bringing the patient promptly under treatment before destructive lesions have developed in organs other than the heart. The difficulty of not always obtaining a positive blood culture from which to prepare an homologous vaccine may be overcome sometimes in other ways. In one of the cases of this series vaccines were made from streptococci derived from the patient's septic uterus, and subsequently from an abscess in the arm. In another case the vaccine was derived from pus in the knee-joint. The cultures and vaccines employed in the described cases were prepared for me by Prof. T. W. Hastings, of the Cornell University Medical School, and Dr. J. C. Meakins, of the pathological department of the Presbyterian Hospital, the vaccines being derived in each case strictly in accordance with the method originally devised by Wright.

As the subject is a new and important one, I have reported the fatal as well as the cured cases, with some degree of detail, for in several of the fatal cases the vaccines produced marked temporary beneficial effect, and the patient apparently was cured of sepsis, only to die of some other disease. In summary: Of the seven cases of septic endocarditis, three were cured by homologous vaccines; in two fatal cases the effect upon the septic phenomena was striking and temporarily beneficial, and in two fatal cases it was slight, but clearly demonstrable. In addition, one advanced case of pyemia was cured.

ACUTE GANGRENOUS PANCREATITIS.

By Louis Jurist, M.D.,
of Philadelphia.

The number of cases of gangrenous pancreatitis, especially of those eventuating in recovery, that have been reported, is so small that an additional case may be of value in bringing forward diagnostic and therapeutic points.

H. M. N., a male, aged thirty-nine years, a merchant, is an only child. His father died of some liver condition associated with ascites. His mother is living, but is a diabetic. The patient has had the ordinary diseases of childhood, typhoid fever when a boy, and pneumonia at the age of twenty-five years. He is married and has two children, living and well. He is temperate, fond of exercise, and eats moderately of plain food, but is occasionally costive.

Some nine years ago the patient first noticed a severe epigastric pain, lasting over night. After that, at long intervals, he had similar

pains, but of less marked severity. In the summer of 1907 he had a sudden attack of pain which had to be relieved by morphine, and since then the attacks have been somewhat more frequent. The last one was about six weeks before the present illness, and again morphine was used. In all of these attacks the paroxysm, while severe, has been of comparatively short duration and unaccompanied by jaundice.

On December 15, 1907, the patient again had severe abdominal pain during the night. He obtained relief from a small hypodermic injection of morphine, but for the next two days had more or less epigastric uneasiness and did not feel well. On December 16, when seen by me, he had some tenderness in the epigastrium, to the left and below the ensiform cartilage. On the following day the pain increased, but no morphine was required. Lavage of the stomach with about a quart of warm water appeared to give him relief. On the next day, December 18, 1907, the patient went to business, but still felt uncomfortable. About seven o'clock on the evening of this day he was seized with severe, agonizing pain in the epigastrium, and soon after this vomited.

Physical examination at this time showed the patient to be suffering severe pain, which was localized in the epigastrium to the left of the median line. A hypodermic injection of one-fourth grain of morphine, followed soon after by half that amount, gave only partial relief. The temperature was 95°, the pulse ranged from 80 to 90 per minute. The patient's skin had a sallow hue, he was perspiring freely, and his extremities were cold. He complained of slight epigastric tenderness and some nausea, and at times regurgitated a small amount of greenish fluid. Olive oil and calomel given during the night and next morning were also vomited. In the next two days the patient developed slight jaundice, which disappeared at the end of forty-eight hours. His bowels did not move until the second day. Elevation of temperature up to 100.2° was at times noted, and the pulse became rapid, weak, and irregular. The vomiting became less, but did not cease, and in the intervals the patient was troubled by persistant belching, which caused him great pain. Epigastric distention developed about December 20, and gradually became more marked. Associated with this was some slight rigidity of the left rectus muscle with epigastric tenderness. The gastric tympany extended to the navel, and the heart apex was pushed upward an interspace. About four days after the acute onset there was noted limited expansion of the left chest, some tenderness in the left loin, and a suggestion of a friction sound in the left axilla. The bowels were moved by enemas, but flatulence continued to distress the patient. The patient's general condition was one of marked toxemia, greatest at about December 21. His pulse was rapid and weak, the respirations were from 34 to 50 per minute, and the temperature irregularly febrile, but never exceeding

101°. The hemoglobin was low, at one time 56 per cent., while there was a continued and increasing leukocystosis, reaching a maximum of 23,400 on December 25. At no time, however, was there an excess of polymorphonuclear leukocytes.

The patient gradually improved as to his general condition. He was seen in consultation by Dr. John B. Deaver and Dr. John H. Musser, of Philadelphia, and on December 26, by Dr. Reginald Fitz, of Boston. At this time there was noted a deep-seated, not tender mass in the epigastric and upper umbilical regions, which was apparently continued into the left loin. There was marked tenderness in the left flank. The epigastrium was slightly distended, but there was no rigidity of the recti muscles. The edge of the spleen was palpable and there was an extended area of dulness in the left lower axillary region. The hepatic dulness extended from nipple to navel.

Up to this time there had been no laboratory findings of importance, except the leukocyte count at various times. The urine, at first scanty and high colored, later became more free. It showed albumin and a few casts, but no sugar. The Cammidge test was performed twice, once with positive and once with negative result.

The diagnosis of acute pancreatitis was first made December 21, and on December 26 all the signs pointed to an accompanying omental bursitis, without, however, an accompanying peritonitis. Operation was considered as being indicated, the only cause of delay being the hope of still further improvement in the patient's condition. This occurred in the next few days, and justified our postponement of operation.

The treatment medically up to the time of operation was comparatively simple. The patient was kept on very light liquid diet, and, as occasion required, was given small doses of strychnine. His bowels were kept open by enemas at first, but later these were not needed. At the time of greatest depression continuous saline enteroclysis by the Murphy method was given, and seemed to cause marked improvement both in the patient's general condition and in the kidney function.

On December 27, 1908, the patient was removed to the German hospital. On December 28, 1908, he was operated upon by Dr. John B. Deaver, under ether narcosis. The pancreatic collection was approached by the extraperitoneal route through the left loin, the incision being exactly as for a kidney operation. When the location of the pancreas was reached, thick dark necrotic material was evacuated. There was some fat necrosis of the fatty tissues along the line of entrance to the omental bursa. No attempt was made to do anything but drain the peripancreatic space and the omental bursa. This was done by means of two rubber tubes of large caliber; several gauze tampons were also introduced.

The cultures from the necrotic material removed at operation were

negative, as were also those from a slight amount of clear fluid aspirated into a hypodermic needle at operation, which seemed to come from just beneath the muscles and from a point above the necrotic and purulent material.

The patient left the operating room in good condition. For a week after the operation his pulse continued rapid, his respirations were above the normal, and the temperature showed marked fluctuations. It was often subnormal in the morning, and reached a maximum in the evening of 103.4°. The patient complained at times of chilly sensations and nausea.

On December 31 the leukocytes rose to 31,800, but with only 76.5 per cent. of polymorphonuclears and 15.5 per cent. of transitional cells. The lymphocytes, large and small, were but 6.5 per cent. On January 9 the left chest was aspirated because of the still persisting dulness there, but no fluid was found. On January 8 a large slough was removed from the wound 4 x 2 x 2 cm. in size; microscopic section showed it to be necrotic pancreas. The wound after this seemed to discharge less, but the discharge again became very free. Twelve days later a portion of gangrenous pancreas (confirmed microscopically) 6 x 3½ x 3 cm. was removed, and on the following day, perhaps because of trauma by the instruments, there was a profuse hemorrhage from the wound. The wound was tightly packed, and after that improved steadily, although still existing in the form of a small discharging sinus at the time of the patient's discharge from the hospital on March 12. The discharge from the wound was always very irritating to the skin, and analysis showed it to consist of pancreatic secretion and pus cells. Trypsin was present in it.

After the operation the patient's temperature was irregular for a time, but gradually dropped to the normal level. The treatment was such as would be given after any grave operation. Some difficulty was experienced in regulating his diet at first, but no digestants except a little pepsin were given. The urine during his stay in the hospital was at no time scanty, and showed no sugar, but always contained a few casts and a trace of albumin.

At the present time the patient is in perfect health. The sinus has permanently healed, after several temporary closures just after he left the hospital. He has had no glycosuria, has gained flesh, and digests all foods, although he has had several slight attacks of indigestion.

An examination of his metabolism by Dr. D. L. Edsall, of Philadelphia, shows it to be practically normal and very satisfactory, considering his recent disturbance. While taking a diet liberally supplied with fat, an average of 103.5 grams a day, he absorbed 93.8 per cent., good even for a normal person. He absorbed 90.1 per cent. of nitrogen, a little below the normal, and the stools contained 1.8 grams a day, which also showed rather poor absorption. He

excreted an average of 12.3 grams of nitrogen in the urine, hence with the 1.8 grams in the feces he lost 14.1 grams daily while taking 17.9 grams. Hence he retained a daily average of 3.8 grams, which he was evidently using in building tissue.

A case such as this furnishes many points of interest from a purely surgical point of view, but to the practitioner or clinician the etiology and diagnosis are of paramount importance. I shall, therefore, scarcely refer to the surgical aspects of the case, and confine my remarks to the clinical features of this particular case.

As to the etiology we must be somewhat uncertain. The patient was not of the obese and alcoholic type usually said to be associated with acute pancreatitis, although a review of the later literature shows this to be less often so than was formerly supposed. The repeated attacks of transient colicky pain, with slight evidences of indigestion and constipation at times present, render a diagnosis of biliary calculus probable, even in the absence of jaundice at any time. The attacks of pain might be considered as being due to pancreatic lithiasis, but in the absence of marked signs of disturbance in the pancreatic function this is untenable. Indeed, this diagnosis has so rarely been correctly made that we may well leave it entirely out of consideration. It is possible that cholelithiasis was the underlying cause of the acute pancreatic lesion. Its direct causation is open to question. It is undoubtedly an "acute" case, yet at operation there was no evidence that a large amount of blood had been lost, although some degenerated blood pigment, etc., was present. It seems likely, in view of the patient's previous good health, and of a few days of indefinite illness before the actual onset of ultra acute manifestations, that pancreatic inflammation preceded the hemorrhage. The two factors mentioned are the only ones upon which we can base such a supposition, because clinically such a distinction at the time of onset is, of course, impossible. On this assumption, then, the course was that of some biliary infection producing a mild inflammatory condition of the pancreas, and this in turn producing a hemorrhage. Following the hemorrhage a typical gangrenous pancreatitis occurred, such as would be expected after interference with the blood supply of a large portion of the organ. A patient in whom this is the sequence of events would seem to stand a somewhat better chance of recovery than one in whom the hemorrhage is primary, due to a diseased bloodvessel wall. In such case we have to deal not only with the grave local lesion, but also with the general arteriosclerosis, of which the pancreatic apoplexy is only a terminal manifestation.

The further course of the case as a gangrenous pancreatitis is to be distinguished from those in which a localized pancreatic infection causes a purulent condition of parts of the organ and its surrounding tissues. The former affection is undoubtedly the more grave.

The diagnosis in any case of acute pancreatitis is usually not easy. In this instance the rule holds good. At various stages of the disease during the first few days a number of conditions were simulated, at least to such a degree that a definite diagnosis was impossible. Before the acute paroxysmal onset of the graver symptoms the case presented no evidence of being anything more than perhaps an atypical form of gastritis or gastroduodenitis. The patient seemed slightly more ill than he should be under those conditions, but nothing definite supervened. At the time of the acute attack of pain, that is, the actual beginning of the gangrenous pancreatitis, there was nothing pathognomonic, yet the symptoms were suggestive. At that time they were: agonizing epigastric pain, subnormal temperature, slow full pulse, and absence of a localized point of tenderness or local abdominal signs.

The pain might be taken to resemble that of either a perforating peritonitis of the upper abdomen or an attack of gallstone colic. It was referred to the epigastrium and at times to the left thereof, but not with any great degree of definiteness. As against possible gallstone colic we have the absence of any radiation to the shoulders, back, etc. But it may safely be stated that at its very onset it was impossible to differentiate this pain from either of those mentioned. The subnormal temperature would occur in any severe sudden abdominal trauma, and depends upon the associated shock. Of more importance is the pulse. This in the attack was slow, regular, and of good volume. The association of a good slow pulse with subnormal temperature and shock is infrequent. Especially does it serve to eliminate from the diagnosis a perforative peritonitis, for in this the pulse practically always is rapid and often weak.

The association of slow pulse with acute pancreatitis has been dwelt upon by the later German investigators, notably Noetzel and Doberaner, both of whom have noted but have not attempted to explain the condition. Mayo Robson does not speak of it, however. John B. Deaver states that in his experience the presence of a slow pulse at the very onset of the attack is common. This must, however, not be confused with the pulse condition some hours later. In practically every instance of a true acute hemorrhagic or gangrenous pancreatitis the pulse in a very short time becomes rapid, weak, and often irregular, as in the present instance. Cyanosis, often included in the syndrome of an acute pancreatitis, was absent in this instance, as were also any definite localizing signs in the abdomen at the onset of the attack.

The diagnosis then at the onset was impossible, but within twenty-four hours certain added features tended to make a definite diagnosis possible. Obstinate constipation supervened, which could not be overcome by ordinary means. Vomiting, which had occurred once before the acute attack of pain, now took place several times in the twenty-four hours. Both of these conditions are practically

always present in acute pancreatitis. More important, however, as a subjective sign was the obstinate belching. Probably due to a diaphragmatic inflammatory involvement from the inflamed retroperitoneal space, it has been noted almost without fail in every case of acute pancreatitis. Not only this, but it has been to the patient a far more distressing thing than even severe vomiting, as each eructation seems to cause great pain.

Slight icterus came on within twenty-four hours, and remained forty-eight hours. This also would point to a pancreatic or biliary condition as opposed to a lesion of one of the upper abdominal viscera. In addition to these general symptoms occurring within the first forty-eight hours, certain other local signs, together with the change in the patient's general condition, tended to make a diagnosis of pancreatic trouble stand out alone as by far the most probable.

The general condition of the patient was one of profound toxemia rather than one of infection. With a rising leukocyte count and some fever, there were no marked exacerbations in the fever, chills, etc. At the same time the depression was so profound that there could be no mistake as to the toxic condition. The differential leukocyte count also showed a persistently low percentage of polynuclears, another point in favor of a toxic agent not purely bacterial in origin. And, indeed, recent investigations tend to show that in these cases there is a specific form of trypsin poisoning, which accounts for the constitutional condition that is so grave a feature of the disease. The local symptoms as described in the history of the case indicate, when considered in sequence, the gradual formation of an exudate deep in the upper abdomen and gravitating to the left loin.

Taken collectively, and regarded in the view of subsequent occurrences, the symptom syndrome in this case is fairly typical of acute pancreatitis. In the differential diagnosis of the case, however, we had to deal at the onset with a very acute condition, in which it was imperative to decide whether or not surgical interference was called for, and each day changed the aspect of affairs.

The differential diagnosis of acute pancreatitis under any circumstances is one of considerable difficulty. Cases have been reported in which intestinal obstruction, acute pelvic disease, acute appendicitis, etc., were simulated to a greater or less degree. I shall not attempt to enter into the general aspects of the matter, but merely to discuss it as applying to this partiticular instance. At the beginning of the illness in this case, or rather at the onset of the ultra-acute symptoms, three possibilities were to be considered. The pain was epigastric, everything pointed to an upper abdominal lesion. Therefore, acute pancreatitis, biliary colic, and the actual or impending perforation of a gastric or duodenal ulcer presented themselves. The pain resembled closely that of biliary colic, and

with the history of a previous attack of pain, a preceding digestive disturbance and the patient's sallow appearance made this the first thought. Against it we had the absence of any palpable condition about the gall-bladder and of any tenderness in this region or over Mayo Robson's point. The shock with slow pulse would have been a possibility in this condition also. The subsequent course of the illness soon showed a purely biliary condition to be out of the question. The toxemia and depression, beginning epigastric swelling, tympany, and tenderness, especially to the left of the median line, proved that we had to deal with a condition more grave than simple biliary colic, even one associated with marked inflammation of the bile passages. Nothing at any time pointed to a perforative lesion of the gall-bladder or ducts. Perforated gastric or duodenal ulcer would give the pain and shock. Yet in this we should have a rapidly spreading peritonitis of the greater cavity, with its typical symptoms, with rapid running pulse, etc., unless the perforation were partly walled off or had taken place upon the posterior wall of the stomach. This is unlikely, and in any case the symptoms would more probably have been those of a septic purulent condition in the greater or lesser peritoneal cavity. The possibility was borne in mind, however, until the toxemia with belching and icterus and the localizing signs deep in the left side left no doubt as to the true condition.

A word or two as to the laboratory findings may not be out of place. They were carefully considered, but, as in most cases they should be, they were counted entirely secondary to the clinical signs. The persistently high leukocyte count, with low polynuclear percentage, pointed to an intoxication rather than a pus infection. The comparatively high count, also, pointed to better powers of resistance than a low one would have done. The absence of glycosuria, except in one doubtful test, did not weigh against the diagnosis of acute pancreatitis, for, as Körte and others have pointed out, glycosuria is a rare accompaniment of the acute pancreatic lesions. The Cammidge test was, as stated, once positive and once negative, before operation. Until it is further developed, this must still be considered more as an interesting test than an important one, at least in the acute forms of pancreatic disease.

The treatment of acute pancreatitis is, of course, surgical in all instances. To the medical man such a case as this presents but one problem, surgically speaking—that is, when to advise operation. Recent German operators who have had opportunity of seeing a number of these cases advise early operation, to avoid the toxemia, to establish drainage, etc. Doubtless this may be the correct course to pursue in many instances, yet we must be guided by the condition present. In the present case, I have no doubt that immediate operation, say within forty-eight hours, when the depression and toxemia were at a maximum, would have resulted fatally, and that

by giving the patient a chance to react therefrom we gave him his one chance of recovery.

In conclusion, I wish to thank Dr. John B. Deaver and Dr. Reginald H. Fitz, of Boston, for their interest, and the use of their notes to supplement my own.

THE TYPHOID CUTANEOUS REACTION.

By Cleaveland Floyd, M.D.,
and
Williston W. Barker, M.D.,
of Boston.

In any disease, such as typhoid fever, in which the type of infection is that of a septicemia, every portion of the body is affected. This is true not only of tissue directly invaded by Bacillus typhosus, but on account of its diffusible toxins, of all the cells of the body. The dissemination of the endotoxins of the organism, through its destruction in the course of the disease, makes this possible, and when large numbers of bacilli are broken down by the action of bactericidal substances, the tissue cells become thoroughly sensitized to the poison. The rapidity of this sensitization is often marked, but it is frequently of short duration. This is accounted for partly by the relatively short period of the infection and possibly by inherent properties of the toxin itself. The union of the toxic molecule of the typhoid bacillus with the cell proteid is apparently unstable and easily broken up. In tuberculosis the converse is true. Infection is generally of long duration, and therefore the continued action of the tubercle toxin on tissue is much more lasting. Furthermore, its union with the cellular elements of the body is much closer and more stable. The old inactive lesions of tuberculosis respond to the stimulation of tuberculin for years after activity has apparently ceased. The addition of tuberculin to the conjunctiva or skin liberates locally toxic bodies, and by the unity with previously formed antibodies a reaction is produced. The reactive powers of the body to the toxin of tubercle slowly decrease with the subsidence of the lesion, but reactivity is quickly renewed when antibodies have previously been formed by the inoculation of tuberculin. In such instances the response to this stimulation is often delayed for several hours over the usual time required for a reaction.

In typhoid fever in which the infection is of relatively short duration, except occasionally in the gall-bladder, the result is different. In a previous paper it was shown that cellular sensitiveness as shown

by the ophthalmic test lasted only as long as the active stage of the disease. To determine if possible, if this sensitiveness affected the skin as well as the conjunctiva, and if so, whether it could be used diagnostically, has been the object of this work.

Preparation of the Solution. Typhoid bacilli of high virulence were grown in pure culture on agar-agar for twenty-four hours, at 37° C. The growth was then washed off with normal saline solution and the emulsion incubated for four days. At the end of this period it was sterilized in a hot water bath at 60° C. for one-half hour, and then standardized after the method of Wright. The sterile emulsion was then centrifuged and the clear supernatant fluid used for inoculation. The solution used was considerably stronger than that used in the ophthalmic test, being the extract of one billion bacilli. In some instances centrifugalization was omitted and the whole emulsion used for inoculation, with the result of more marked reactions. A standard solution was used in all instances, and not an emulsion of the homologous bacillus in the individual case.

Method of Inoculation. In making the test, exactly the same technique was followed as in obtaining the cutaneous reaction in tuberculosis. After cleansing the arm with alcohol a drop of the solution was transferred to it by means of a sterile platinum loop, and this was then worked into the superficial layers of the skin by means of a platinum lancet. The inoculated material was then allowed to dry and a second puncture made below as a control. On the day following the inoculation there appeared about the point of scarification a moderate red areola ranging from 0.5 to 1 cm. in diameter. This reached its maximum in about twenty-four hours, and gradually diminished in intensity, disappearing at the end of forty-eight hours. The presence of vesicles and œdema, so often seen in the cutaneous tuberculin reaction, was noticeable by its absence. Constitutional symptoms were lacking in all cases. In a series of cases of typhoid fever treated by Richardson with bacterial residues well marked local reactions occurred in a number of cases, and persisted for some time. There were, however, general as well as local reactions in the cases treated.

Clinical Results. In all there were 48 inoculations on 47 patients, of whom 30 were ill with typhoid fever, 2 with β-paratyphoid infection, 2 with unclassified fever (clinically not different from typhoid fever), 8 with tuberculosis, 1 with pneumonia, 1 with acute articular rheumatism, 1 with pernicious anemia, and 2 were apparently normal individuals. Of the typhoid fever cases, the reaction was positive 19 times, negative 9 times, and rendered unsatisfactory by the patient twice. One of the cases of unclassified fever responded to the test, although giving no other evidence of typhoid infection. Neither of the cases of paratyphoid fever gave any reaction whatever. With the exception of a slight reaction in the patient having pneumonia, all of the definite non-typhoid cases gave no re-

action. All of the positive reactions occurred during the active period of the disease, the single case which was inoculated twice giving a much more striking result at the first inoculation than the second. This result is of interest when we note that in tuberculosis the repetition of the cutaneous test when it has previously been positive gives a more active response at the second inoculation. The failure of the cases of β-paratyphoid infection to respond to the test may be due to the different character of the organism causing the infection.

TABLE.

Case.	Age.	Disease. Typhoid fever.	Days entrance.	Widal Positive Day of disease.	Blood culture Positive. Day of disease.	Cutaneous. Day of disease.	Character.	Time in hours.
1	7	Relapse	14	16	neg. (2)	20	Positive	48
2	21	Typhoid fever	5	5	..	17	Positive	48
3	7	Typhoid fever	14	16	..	25	Positive	48
4	13	Typhoid fever	14	17	neg. (10)	39	Positive	48
5	50	Typhoid fever	10	12	23	22	Positive	24
6	38	Relapse	7	7	7	10		
7	25	Typhoid fever	14	20	..	18		
8	31	Phlebitis—typhoid fever	28	31	..	90	Positive	24
9	27	Relapse	7	7	neg. (2)	60	Positive	24
10	17	Typhoid fever	8	8	..	17		
11	30	Typhoid fever	14	14	..	21		
12*a*	31	Typhoid fever	6	6	..	7	Positive	24
12*b*	31	Typhoid fever	6	6	..	35	Positive	
13	4	Typhoid fever	8	8	..	35		
14	17	Typhoid fever	14	16	..	30	Positive	24
15	47	Typhoid fever	10	17	..	30	Sl. positive	24
16	3	Typhoid fever	28	29	29	32		
17	19	Typhoid fever	14	20	..	34		
18	10	Relapse	8	8	..	13	Positive	24
19	27	Typhoid fever	4	6	..	10	Positive	24
20	6	Typhoid fever	6	6	..	9		
21	7	Typhoid fever	14	15	..	17	Positive	24
22	7	Typhoid fever	15	15	..	17	Unsatisfactory	
23	24	Typhoid fever	7	7	7	9	Positive	24
24	27	Typhoid fever	9	13	10	10		
25	38	Typhoid fever	14	16	..	24	Unsatisfactory	
26	16	Typhoid fever	15	15	..	16	Positive	24
27	14	Typhoid fever	9	11	..	22	Positive	24
28	37	Typhoid fever	9	13	9	13	Positive	24
29	6	Typhoid fever	14	14	..	25	Sl. positive	24
30	40	Typhoid fever	35	35	..	43		
31	25	Beta-paratyphoid	8	13	12	16		
32	21	Beta-paratyphoid	21	30	24	45		
33	34	Fever, cause (?)	8	4	2	9		
34	19	Fever, cause (?)	14	2	neg. (2)	14	Positive	24
35	39	Pneumonia	7	..	..	8	Sl. positive	24
36	28	Rheumatic fever						
37	51	Pernicious anemia						
38 to 45, tuberculosis								
46 and 47, normal individuals								

No attempt has been made to discover how early this reaction appears in the course of the disease, and the figures as to relative incidence of positive Widal reaction and blood culture are purely incidental to the description of the case and in no instance competitive.

The value of the reaction in diagnosis is problematical. The proportion of negative reactions in this small series is so great that little can be inferred from a positive or negative result in a given case.

The use of bacterial residues of greater toxic power than the bacillary extract might increase the percentage of positive reactions very materially. If this be true, the cutaneous reaction might be made of real value. In so far as the reaction has been obtained in typhoid fever, it is in harmony with similar observations made in cases of tuberculosis, and substantiates the theory of general tissue susceptibility to bacterial toxins in certain general infections.

We wish to express our indebtedness to the physicians of the Massachusetts General Hospital and the Children's Hospital for allowing these studies to be carried on in their wards.

SARCOMA OF THE STOMACH.

By Logan Clendening, M.D.,

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The following are the notes of two cases of sarcoma of the stomach that have come to autopsy in the pathological laboratory of the University of Kansas.

Case I.—Case of Dr. James F. Trexler and Dr. Jacob Block. H. H., male, aged thirty-three years. His father is living, aged seventy-two years; his mother is living, aged sixty-one years; one brother is living, aged twenty-seven years; and one brother died, aged eighteen years, of "inflammation of the brain." The patient is a bartender; is married, and has one child. He had no serious illness until he was eighteen years of age, when he contracted specific urethritis, from which he never fully recovered.

In 1900 Dr. Trexler was called to see the patient, because of a very large gastric hemorrhage. The day following the hemorrhage the patient passed several tarry stools. Under treatment he recovered symptomatically, and was not seen until October, 1903, when he presented the following picture: He was a tall, thin man, strong and wiry. He complained of pain in the thorax. The heart showed an enlarged area of dulness with distinct heart sounds, but no murmurs (fluid in the pericardium). The pulse was 120. There was an area of complete dulness and absence of vocal fremitus and breath sounds over the lower lobe of the left lung. There was no pleural friction. The right lung was negative. A stomach analysis was never made. Urine (single specimen) was yellow, acid; specific gravity, 1025; contained no albumin or sugar. Microscopic examination was negative.

The patient died ten days later. The clinical diagnosis was pericarditis with effusion; left-sided pleurisy with effusion.

Autopsy Report. The body is that of a well-developed male, weighing 140 pounds. The body surface shows no evidence of disease.

The pupils are equal and dilated. The tissues of the chest are œdmatous, particularly on the left side and below the clavicle. The abdomen is tympanitic and contains a small amount of fluid. Both pleural cavities are full of greenish fluid. The pericardium measures six inches along the diaphragm. The sac is full of brownish fluid. After the lighter fluid is removed a quantity of free blood remains. The heart apex is displaced to the fourth interspace. The heart is small and is covered with a ragged, fibrinous exudate in the form of a membrane, which can be easily torn off. The auricles and ventricles are negative. Nothing of present interest is found in the lungs, the pancreas, the kidneys, the liver, or the spleen. The lymph nodes at the lesser curvature of the stomach are distinctly enlarged. The stomach contains a quantity of light chocolate-colored fluid. On the posterior wall near the lesser curvature, two inches from the pyloric ring, is an area three inches in diameter, whitish in color, presenting a slightly rugous surface, the entire area

FIG. 1.—Sarcoma of the stomach, showing as a round flat disk on the posterior wall. Two small pieces have been removed for microscopic examination (Case I).

somewhat elevated above the surface of the mucosa (Fig. 1). The growth extends outward in places, apparently following the rugæ. Over the stomach the veins are much dilated. The mucosa also shows a number of whitish follicles, one to two millimeters in diameter.

Microscopic Appearance of the Neoplasm in the Stomach. The tumor is a mixed-cell sarcoma, composed of round and spindle cells held in a loose, wide, network of fibrillary connective tissue. There are also numerous capillaries. The cells are small round, large round, and spindle-shaped, hematoxophile, with a small amount of cytoplasm and large granular nucleus. The nuclei at the edge of the growth next to the lumen of the stomach are large and vacuolated, and here the cells appear to be degenerating. The growth was evidently primary in the submucosa, invading the muscularis mucosæ and mucosa, leaving the muscle bands and serosa more or less intact. The mucosa is almost entirely obliterated in the circumscribed area of the growth, only a few tubules remaining, and these

at the periphery of the growth. The mucosa presents the thickest point of infiltration; the muscularis maintains its identity, but with considerable accumulation of cells between the muscle bundles. The submucosa is extensively infiltrated, but exhibits numerous strands of normal connective tissue intact. The visceral wall of the pericardium presents a metastasis of the growth. The blood-vessels in the vicinity are filled with sarcoma cells and the fibrous tissues are considerably infiltrated. The cells present in general the same appearances as in the primary tumor.

This case presents some points of considerable interest. Clinically, the great resemblance in symptoms to gastric ulcer is notable. In this instance, due to the fact that there was no tumor large enough to be palpated through the abdominal walls, the differential diagnosis must be considered absolutely impossible. Pathologically, the sharply circumscribed limitation of the tumor, the destruction of the mucosa, and the metastasis in the pericardium (the only record of such metastasis) are the interesting features. In the light of the autopsy findings we may reasonably believe that the first hemorrhage occurred from the sarcoma in the stomach, and that later metastasis formed in the pericardium, giving rise to an effusion three years later. The pleural effusion remains unexplained so far as this tumor is concerned.

Case II.—Dr. J. W. Perkins' case, St. Margaret's Hospital. W. H., aged fifty-eight years; male; December 7, 1905.

The patient had had no serious sickness up to six months ago, when he noticed loss of appetite and distress after eating. Later, he began vomiting once or twice daily, about two hours after eating. Sometimes he vomited immediately, "if he ate anything which did not agree with him." The pain after eating was relieved by frequent belching. He has lost about thirty pounds in four months. He is constipated; feels as though there were some stoppage below the stomach. He has had a cough for two or three months.

The patient is anemic, sallow, and cachectic. There is no jaundice. The tongue is large, moist, and flabby. A large mass, about the size of two fists, is felt in the upper abdomen in the region of the stomach, extending over the splenic region. The mass is movable and can be brought over to the right side. The heart and lungs are negative. The urine is negative. The gastric contents are not recorded. The clinical diagnosis was gastric tumor.

Operation disclosed a tumor of the stomach, involving almost the entire organ. The inoperability of the growth was apparent, and no effort was made to remove it. When the patient went home, twelve days after the operation, the tumor could be plainly seen through the abdominal wall. After death the stomach was sent to the laboratory for examination.

Pathological Report. The stomach is enormously enlarged; the walls greatly thickened, measuring at the pylorus two and one-

quarter inches. The pylorus, though surrounded by the growth, is patent. The neoplasm extends along the greater curvature, lesser curvature and posterior walls, as a lobulated tumor mass growing into the cavity of the stomach, especially at the pylorus (Fig. 2). The mucous membrane is generally smooth, but in some places in ulcerated. The serosa is smooth.

Microscopic Appearances. Mixed-cell sarcoma, with large and small round cells. The cells take the stain very unevenly, so that the tumor presents a varied picture to the eye: some cells have large some small dark staining nuclei; some large and some small light staining nuclei; some vacuolated or granular nuclei. There are a few spindle-shaped cells. The connective tissue groundwork is

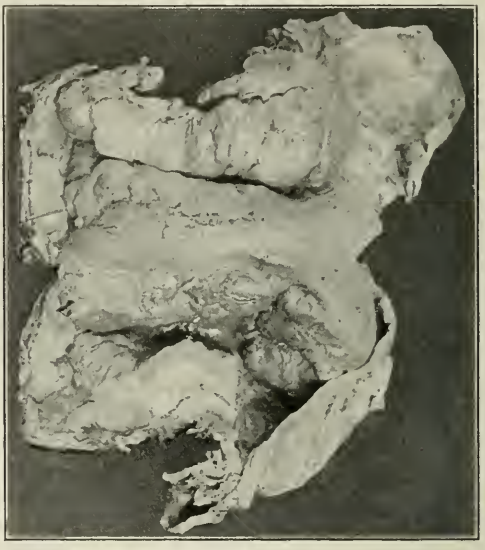

FIG. 2.—The stomach opened, showing the sarcoma projecting into the lumen as a nodular mass (Case II).

composed of slender fibrils, and is quite abundant. There are numerous capillaries in the tumor substance. The growth is more strictly confined to the submucosa than in Case I, although the muscularis and mucosa are also involved. The tubules are not so widely destroyed as in the previous case, but the superficial epithelium of the stomach in almost entirely gone.

According to Howard,[1] the earliest report of a case of sarcoma of the stomach is by Bruch in 1847. Schlesinger,[2] in 1897, however, made the first comprehensive report, collecting 33 cases and report-

[1] Jour. Amer. Med. Assoc., 1902, xxxviii, 392. [2] Ztschr. f. klin. Med., 1897, xxxii.

ing three. Riegel's[3] article in Nothnagel's *System* is a review of Schlesinger's article. Brooks[4] in 1898, reported a case and analyzed 15 from the literature. Dock[5] reported a case in 1900 and collected 13 more. Howard, in 1902, in the most comprehensive article in the literature, adds 7 more cases from the literature and reports 4 of his own. The total number of cases reported is 61, which are carefully analyzed by Howard, whose digest of the literature is as follows:

Frequency. After carcinoma, the most frequent tumor of the stomach; 61 cases are reported.

Etiology. Sex: 30 cases in females; 25 in males. In 6 the sex is not mentioned.

Age. Youngest patient, three and one-half years (Finlayson[6]). Fifteen to twenty years, 6 cases; twenty to thirty years, 9 cases; thirty to forty years, 7 cases; forty to fifty years, 10 cases; fifty to sixty years, 10 cases; sixty to seventy years, 5 cases; seventy-eight years, 2 cases; over eighty years, 2 cases. Incidence in second half of life distinct.

Seat of Tumors. Diffuse, 13 cases; pylorus, 8 cases; pylorus and lower half of stomach, 6 cases; pylorus and greater curvature, 1 case; total, involving pylorus, 16 cases, producing stenosis in 5; greater curvature, 7 cases; greater curvature and posterior wall, 1 case; lesser curvature, 2 cases; anterior wall, 1 case; lesser curvature alone, 5 cases; cardia and lesser curvature, 3 cases; posterior wall and lesser curvature, 1 case; greater curvature and posterior wall, 1 case; posterior wall alone, 6 cases; seat not mentioned, 5 cases.

Size of Tumors. Pigeon's egg to man's head. (Baldy's[7] must have been larger, as he reports this remarkable tumor as filling the abdomen from the symphysis to the ensiform.)

General Appearance. Nodular in 27 cases; ulcerated in 10 cases. Tumors usually in the submucosa. Cystic, hyaline, calcareous, and myxomatous degeneration occurred. Metastasis occurred in 40 per cent; not so widespread as in carcinoma. Mesenteric glands, omentum, liver, spleen, and kidneys were seats of metastasis. (In my first case, the pericardium.)

Histology of the Tumors. Round cell, 16; spindle cell, 8; mixed cell, 4; lymphosarcoma, 15; myxosarcoma, 4; fibrosarcoma, 3; angiosarcoma, 3; endothelioma, 1.

Since Howard's article there have been eight articles describing this condition and ten cases have been reported. Oberst,[8] Dobromyslow,[9] Weinberg,[10] McCormick and Welsh,[11] each describe 1 case; Yates[12] reports 2 cases, Manges,[13] 3, and Dalton,[14] 1. Weinberg's case was a tumor arising from the lesser curvature and cardia with

[3] Nothnagel's Spec. Path., Band xvi.
[4] Med. News, 1898, i, 617.
[5] Jour. Amer. Med. Assoc., 1900, xxxv, 156.
[6] Brit. Med. Jour., 1899, ii, 1535.
[7] Jour. Amer. Med. Assoc., 1898, i, 617.
[8] Beitr. z. klin. Chir., xlv, No 2.
[9] Russki. Chir. Archiv, 1902, Heft 5.
[10] Inaug. Dis. München., 1901.
[11] Scottish Med. Surg. Jour., 1906.
[12] Ann. Surg., 1906, xliv, 599.
[13] Mt. Sinai Hosp. Reports, 1907, 68.
[14] Trans. Lond. Path. Soc., 1906; Brit. Med. Jour., 1906, 1638.

metastases in the peritoneum and abdominal organs. Dobromylow's case, in a patient aged fifty-three years, was a spindle-cell sarcoma on the lesser curvature of the stomach near the pylorus. It involved the muscularis and left the mucosa intact. In the centre was a focus of softening.

Oberst's case was in a man, aged twenty-five years, who became sick suddenly, with hematemesis, pain in the epigastrium, blood in the stools, and great tenderness in the epigastrium, followed by a high, remittent fever and right-sided pleurisy. With expectant treatment, the bleeding subsided, but the infiltration in the epigastrium remained. The autopsy revealed a tumor, including the pylorus, sharply marked off from the duodenum, spreading out over the stomach wall and extending some centimeters into the liver substance. The tumor was fixed behind by strong adhesions. The stomach wall was 6 cm. thick. The atrophic mucosa covered the whole tumor, except in one place, where there was a deep depression the size of a five-mark piece, the bottom of which was formed by degenerated liver tissue. There was no lymphatic enlargement nearby. Microscopically, the tumor proved to be a round-cell sarcoma, originating in the muscularis, involving the mucosa, but not obliterating the glands and not involving the serosa. Oberst discusses the differential diagnosis between sarcoma and carcinoma of the stomach, and says that bleeding is less frequent in sarcoma. Metastasis, he states, is not always conclusive, as von Leube found a carcinoma of the stomach with sarcomas of the skin.

Norman Dalton's case occurred in a male, aged fifteen years. The symptoms were abdominal swelling and pain, covering a period of three or four months. At autopsy there was found a small round-cell sarcoma of the stomach and lymph glands.

Manges reports 3 cases: One was in a man, aged forty-eight years, in whom an epigastric tumor had developed between two examinations, four months apart. At autopsy a lymphosarcoma was found growing at the pylorus, with metastasis in the pleura. One was in a woman, aged forty-eight years, in which the autopsy revealed a scar of an old ulcer at the pylorus and a soft sessile tumor, a myosarcoma, on the outer wall of the stomach near the pylorus. One was in a girl, aged nineteen years, who had bloody vomiting, pain after eating, and tenderness and pain in the left hypochondrium. At operation an inoperable diffuse lymphosarcoma was found involving almost the entire stomach.

Manges comments on the difficulty of diagnosis, due to lack of gastric symptoms. He calls attention to these points: (1) Gastric sarcomas arise from the muscularis and submucosa. (2) The growth may be diffuse (lymphosarcoma) or polypoid (spindle-cell). (3) The pylorus is rarely stenosed. (4) Degenerations frequently occur. (5) The tumors are often large. (6) The tumors may be primary or secondary. (He states that they may be local

manifestations of Hodgkin's disease, a statement which, in view of the recent studies of Longcope and Reed on the histology of Hodgkin's disease, seems unwarranted.) (7) The great range in the age of the patient.

Of Yates' two cases (operated upon by Dr. A. J. Ochsner), the first was a large round-cell sarcoma, which involved the greater curvature of the stomach, 3 cm. from the pylorus. It occurred in a woman, aged thirty-seven years. The second case is remarkable as being one of the few cases of cure on record. The patient was a man, aged forty-four years, who had had an attack of hematemesis four months before operation; later, a tumor could be felt beneath the costal margin. It proved to be a large spindle-cell sarcoma, which arose by a pedicle from the posterior wall of the stomach. It was removed, and the patient, when examined seventeen months after the operation, was well.

Sarcoma of the stomach is of considerable interest pathologically, as in frequency it is the second among the tumors of that organ. From my study of the 2 cases and the descriptions of various authors, the usual type seems a mixed-cell sarcoma, composed of round and spindle cells lying in a connective tissue matrix of fine interlacing fibrils. From this group must be separated 3 cases, described as angiosarcoma, and 1 case of endothelioma. The tumors are generally reported as not involving the mucosa, although my cases are decidedly at variance with this. The tumors grow to considerable size, but seem not so disposed to metastasis as carcinoma (metastasis in about 40 per cent. of the cases). Sarcoma is of interest, too, as occurring in the majority of cases in individuals over forty years of age.

Clinically, it is of somewhat less interest. In three cases (Oberst's, Manges' third case, and my second case) the vomiting of blood in a young individual made the consideration of a differential diagnosis between the affection and ulcer important. The chemical analysis of the gastric contents in these cases gives us about as little help in diagnosis as such analysis ordinarily does. The total acidity runs about 50; the HCl is sometimes, though not uniformly, absent, and may be high. Sediment is modified by bleeding and presence of stenosis. The differential diagnosis between sarcoma and carcinoma is very difficult, and is unimportant as regards treatment. Pyloric stenosis occurs in about 7 per cent. of the cases. Fifteen cases have been operated upon. Dock's patient was well at the end of five months, and in 1 case reported by Yates the patient was well at the end of seventeen months. Yates also quotes Corner and Bairbank[15] as reporting 4 cases well at the end of four, five, twelve, and twenty-four months after operation. The tumor is usually fatal within one year.

[15] Practitioner, 1904, lxii, 810.

ENDEMIC AMŒBIC DYSENTERY IN NEW YORK, WITH A REVIEW OF ITS DISTRIBUTION IN NORTH AMERICA.

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The infrequency with which endemic amœbic dysentery is met in New York, and the prevailing belief that it is a disease of the tropics justify recording the following cases. Only the essential facts of the histories are included:

Case I.—S. W. McG., admitted to the Vanderbilt Clinic July 7, 1908; history 39,054; aged nineteen years; U. S.; single; electrician.

In September, 1907, the patient was suddenly taken with a cutting pain about the navel, intermittent, and lasting four days, at the end of which he began to pass "brown slime" which "looked like tobacco juice." At first he had as many as seven passages at night, accompanied by pain in the abdomen and rectal tenesmus. The diarrhœa kept up with variable intensity until a week before admission, when the symptoms became so much worse that he had to come to the clinic for relief. He had lost twenty pounds. At one time during his illness he was admitted to a hospital, but a stool was never examined. *He had not been off Manhattan Island since one day in the first two weeks of June,* 1907. On admission, the temperature was 98.2°; pulse, 92; respirations, 24; and the weight 112 pounds. He was poorly nourished and wasted. The tongue was moist and slightly coated. There was no lymphatic enlargement.

The heart was negative. The pulse was of good size and force. Physical examination otherwise was negative.

On July 8 a fresh stool was examined, and found to contain bodies like amœbæ, but no motility was seen. The proctoscope was passed and some bloody mucus obtained, which contained many actively motile amœbæ.

Case II.—W. K., admitted January 20, 1909; history 61,986; aged twenty-eight years; married; longshoreman.

Four years ago he began to have bloody diarrhœa, with about two stools at night and three in the day. For the last two years the diarrhœa has been more severe, with four to five stools in the day time, and an equal number at night. He has lost considerable strength, but no weight. No other symptoms. On admission he was well nourished, but pale. No abnormal physical signs were found. By the proctoscope bloody mucus containing a few motile amœbæ and many similar inactive bodies, was obtained.

On January 21, mucus obtained through the proctoscope and examined on a warm stage showed a fair number of motile amœbæ.

The patient states that he was born, brought up, and always

lived in Fishkill up to two years ago. (Fishkill is on the east bank of the Hudson, about sixty-six miles from New York.) He has never been outside of New York State except to unload bricks in New Jersey opposite New York City. For about seven years previous to his illness he had been working on a brick boat, plying between New York and Fishkill. The drinking water was at times taken from the river near Fishkill, when the water, after a rain, was not brackish, and allowed to stand in a barrel.

Case III.—Clinic number 76,899; male, aged thirty-eight years; Russian; married; calciminer; admitted to the Vanderbilt Clinic May 19, 1909.

The family history and the past history are unimportant.

The patient came to America six years ago, and since his arrival has lived continuously in Brooklyn. In August, 1908, he developed dysentery, and was in a Brooklyn hospital for four months. He now has six or seven watery and bloody stools a day. Physical examination was negative.

On May 21, 1909, sigmoidoscopic examination showed small, superficial ulcerations; one just inside the sphincter was sloughing. Mucus examined microscopically on a warm stage showed fairly numerous motile amœbæ coli.

In the first case the patient was undoubtedly infected in New York City. Musgrave and Clegg,[1] whose admirable work of growing the amœba in symbiosis with bacteria harmless to animals and man marks an epoch in our knowledge of amœbiasis, found that in animals fed on cultures the period of incubation was from twenty to seventy days. Injections produced focal lesions containing amœbæ as early as the second day. To a healthy man, who had been observed ten days, three gelatin capsules were fed of three week's old cultures of amœba in symbiosis with a bacterium proved harmless to man and animals. On the twelfth day there was diarrhœa, and amœbæ for the first time were present in the stools. Consequently all the experimental evidence that Musgrave and Clegg have furnished would indicate that our first case was infected some time subsequent to his outing from the city.

Among New York physicians the belief is current that intestinal amœbiasis is to be expected only in persons recently returned from the tropics. Although an ill-defined impression exists in the minds of a few practitioners that occasional endemic cases of amœbic dysentery occur in New York, the existence of this condition is not as generally known as it should be, because the cases have been few and because systematic collection of them has never been presented to the profession. The important features of amœbic dysentery are not sufficiently sharply defined from those of other forms of colitis to enable one unacquainted with it to recognize the condition.

[1] Bull. 18, Bureau of Gov. Lab., Manila, p. 1.

Consequently the only way the diagnosis can be made in the majority of endemic cases is to suspect that the condition exists.

The following collection of endemic cases in New York City is offered in the hope that it will stimulate efforts to recognize the disease and determine its exact frequency in this vicinity.

In 1893 Brannon[2] reported the case of an inhabitant of New York who had been a sailor in his youth, but had given up that occupation years before his illness began, to become a grocer. There is nothing in the history to indicate that he was infected outside of New York. In 1886 he had a liver abscess, and in the following six years he had a succession of five other such abscesses, to the last of which he succumbed in March, 1892. Amœbæ were found in the liver and colon postmortem.

At a meeting of the New York Academy of Medicine, in 1900, Graser[3] reported two endemic cases. One patient was a hotel keeper, aged thirty years, who had never been out of New York. The other patient had never been farther than Staten Island. At the same meeting, in commenting on a series of twelve cases in his own experience, Brooks[4] stated that two of them were certainly infected in New York. In the discussion of a paper by Dock,[5] in 1902, Libman said that within the last year at the Mount Sinai Hospital specimens from at least ten cases of amœbic abscess of the liver had been examined, from which no history of any residence outside of New York City could be obtained.

Distribution Throughout North America. *Canada.* The late Dr. A. Brayton Ball[6] reported a case in 1892 that originated in Winnipeg. This was the first case to be reported from New York City.

Maine. Lockwood's[7] case in all probability contracted the disease in Old Orchard Beach.

New Hampshire. One of the earliest American cases on record was that of Gerry and Fitz,[8] who, in 1891, contributed the history of a young woman, who was supposed to have contracted the disease in the White Mountains.

New York. Stockton[9] reported, in 1894, an endemic case from Buffalo. He mentions a case occurring in his practice the preceding year. Nydegger[10] mentions a case occurring in the practice of a medical friend in the northern part of the State.

Pennsylvania. Musser[11] was among the first to report endemic cases. In December, 1890, he reported from Philadelphia two

[2] New York Medical Journal, 1893, lvii, 317.
[3] Medical News, 1900, lxxvi, 433.
[4] Medical News, 1900, lxxvi, 433
[5] Jour. Amer. Med. Assoc., 1902, xxxix, 617.
[6] Therap. Gazette, 1892, 3 S. viii, 523.
[7] Medical Record, 1897, li, 475.
[8] Boston Medical and Surgical Journal, 1891, cxxv, 592.
[9] Internat. Clin., 4 S. i, 69.
[10] West Virigina Medical Journal, 1907, ii, 11.
[11] University Medical Magazine, 1890, iii, 116.

hospital cases, and mentioned two occurring in private practice. About the same time Stengel[12] reported three, one of which was a private case alluded to by Musser. A few years later Musser and Willard[13] reported another case from Philadelphia.

Maryland. In 1890 Lafleur[14] reported the first endemic case in this country. Since then Howard,[15] Street,[16] Preston and Rurah,[17] Lewis,[18] Amberg,[19] and Boggs,[20] have reported a large number of cases.

District of Columbia. In a series of cases treated at the Johns Hopkins Hospital, Boggs reported three from the District.

Virginia. In the series just alluded to, Boggs reported fourteen from this state. Slaughter[21] has reported a case probably infected in Richmond.

West Virginia. In the series reported by Boggs, eight acquired the disease in this State.

North Carolina contributed five cases to Bogg's series.

South Carolina. Wasdin[22] has reported three cases.

Georgia. Boggs has contributed four cases, Wasdin one, Harris[23] thirty-two, and Diamond[24] three.

Florida. Boggs has contributed two cases. I myself have seen two cases which were infected in this State.

Tennessee. Boggs' report includes one case from this State, while Harris has reported two.

Alabama. Boggs has reported four cases, Harris and Withington[25] one each, while Taylor[26] has reported a fatal case from the centre of the State.

Mississippi. McElroy[27] reports a case from Stovall.

Ohio. Eichberg,[28] in 1891, reported from Cincinnati a case of amœbic liver abscess, no statement being made that the case was imported.

Illinois. Kurtz[29] has contributed a case from Neoga, while Preble[30] from Chicago, reports two cases; the first patient had lived for nine years in the city, and had been in the country a few weeks, six months previous to the beginning of the illness. The second patient had not been out of Chicago for thirty years.

[12] Medical News, 1890, lvii, 500.
[13] University Medical Magazine, 1892, v, 525.
[14] Johns Hopkins Hospital Bulletin, 1890, i, 91.
[15] Medical News, 1892, lxi, 705.
[16] Maryland Medical Journal, 1893-94, xxx, 92.
[17] New York Medical Journal, 1894, lx, 593.
[18] Maryland Medical Journal, 1896, xxxv, 145.
[19] Johns Hopkins Hospital Bulletin, 1901, xii, 355.
[20] Virginia Medical Semimonthly, 1908, xiii, 9.
[21] Virginia Medical Monthly, 1895–96, xxii, 722.
[22] Medical News, 1891, lix, 656.
[23] Amer. Jour. Med. Sci., 1898, N. S., cxv, 384.
[24] Phila. Med. Jour., 1900, v, 817.
[25] Boston Medical and Surgical Journal, 1894, cxxx, 516.
[26] Alabama Medical Journal, 1903–04, xvi, 10.
[27] Memphis Medical Monthly, 1902, xxii, 169.
[28] Medical Mews, 1891, lix, 201.
[29] Medicine, 1903, ix, 595.
[30] Chicago Medical Recorder, 1899, xvi, 33.

Missouri. Nietert[31] has reported seven cases which developed in or near St. Louis. Meyer[32] observed a case in St. Louis and mentioned one of Fischel's, while Funkhouser[33] has seen one originating in the same city. Murphy[34] has contributed a case from Kansas City.

Michigan. Dock, in 1902, reported one case infected in Michigan. The patient had not been out of the State since 1893. There is no history of any contamination by persons returning from the tropics.

Minnesota. Head,[35] in 1904, reported a case originating in Maple Lake, Itasca County, and in 1905 Aurand[36] a case in a millworker who had lived in Minneapolis many years and had never been South.

Montana. Spelman and Wherry,[37] in 1906, reported a case which was unquestionably infected in Montana.

Arkansas. One of West's[38] cases was probably infected in this state.

Indian Territory. West contribted one case from here.

Texas. Boggs has contributed to the literature two cases. Hektoen[39] has reported one, Dock,[40] in one report, one case, in a second,[41] twelve, Diamond[42] two cases, and West three.

The review of the literature on this subject shows, therefore, that the prevailing idea concerning the source of amœbic infection is hardly justifiable. Probably this erroneous belief has been responsible for failure to recognize the disease. A realization that this type of disease is endemic in New York led to a search for it, with a result that these three cases were found within a year. It is to be hoped that a realization of its existence elsewhere will give rise to the recognition of cases and to the establishment of proper prophylactic and therapeutic measures.

BLOOD CULTURES IN HUMAN GLANDERS.

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The invasion of the blood current in cases of glanders in human subjects is of considerable scientific interest from three points of view: (1) By reason of the light which it throws on the pathological processes of the disease and the modes of dissemination of the infection; (2) on account of the valuable theoretical suggestions in

[31] St. Louis Medical Review, 1902, xlvi, 55.
[32] Ibid., 19.
[33] Ibid., 1903, xlviii, 343.
[34] Journal of the American Medical Association, 1894, xxii, 149.
[35] Northwest Lancet, 1904, xxiv, 249.
[36] Ibid., 1905, xxv, 321.
[37] Ibid., 1906, iv, 277.
[38] Trans. First Pan Amer. Med. Cong., 1893, i, 301.
[39] North. Amer. Pract., 1892, iv, 554.
[40] Medical Record, 1891, xl, 7.
[41] Quoted by Harris.
[42] Philadelphia Medical Journal, 1900, v, 817.

relation to infections in general, their mode of establishment, and means of propagation; and (3) by reason of the diagnostic aid given in cases of infection with indefinite or unrecognizable origin. The marked progress recently made in the study of blood invasions by bacteria has assisted materially toward an understanding of the sequence of events and rationale of diseases in which the circulatory medium is invaded by pathogenic organisms, either primarily or secondarily. Certainly, blood cultures in such diseases as typhoid fever, acute bacterial endocarditis, infective venous thrombosis, etc. have thrown new light upon the clinical course of the diseased processes, and have helped materially in advancing a more logical mode of studying and treating these cases.

Human glanders may safely be regarded as another of the bacterial diseases in which the blood current, sooner or later, is invaded by disease producing bacteria. It is still more probable that this fact plays not a secondary, but an important and essential part in the dissemination of the infection.

Soon after the first conception of the bacterial nature of certain diseases was established, and coincident with the discovery by Loeffler,[1] in 1882, of Bacillus mallei, glanders was regarded as an infection of pyemic nature. This conclusion was readily drawn by clinicians and was founded upon the simple observations that the disease began in a primary focus of infection, and that it pursued its course with the early establishment of foci of suppuration far distant from the orginal point of inoculation and apparently not transmitted by the lymphatics or by cutaneous auto-inoculation. As in these metastatic deposits, the original offending organism, as seen in the primary focus, was also readily demonstrable, the natural conclusion was drawn that the blood current was the medium of transmission. In support of this, the older pathologists in the era before the introduction of blood cultures, demonstrated the fact that in almost all the autopsies on cases of this nature, Bacillus mallei could be seen in smears of the blood after death (Koch,[2] Wassilief,[3] Kiemann,[4] Babes[5]).

The earliest attempts to recover the bacilli from the circulating medium were unreliable because imperfect methods were used. The first success was that of Frese, Ross, and Wilbert,[6] who, in 1891, succeeded in recovering the living organism from a patient suffering from the acute form of the disease. Since that date, and with the advent of improved methods, there have been sixteen cases in which the blood from the patient has been cultured (see Table). The results are fairly uniform in character, and the cases group themselves into two classes: (1) Acute glanders with positive or negative blood cultures, and (2) chronic glanders.

[1] Arbeit a. d. Kais. Ges., 1886, Band i, b. 141.
[2] Arch. klin. Chir., lxv, 37.
[3] Deut. med. Woch., 1883, p. 155.
[4] Wien. med. Woch., 1888, i, 515.
[5] Arch. de méd. exp., 1891, iii, 619.
[6] Medical News, 1891, p. 679.

Case No.	Author	Clinical Data			Cultural Data			Result
		Onset	Symptoms	Course	Day of culture	Symptoms at time of culture	Technique of culture	
I	Frese, Ross, and Wilbert: Med. News, 1891, p. 679	Pain in right shoulder. (Occupation, driver)	Rapid pyemia; pustules; nasal mucosa and respiratory mucous membrane involved	17 days	14th	Before general pyemic demonstration; pain in shoulder only symptom at time of culture	Blood taken in small glass cell, examined microscopically, and cultured	+
II	Duval, Gasne, and Guillemont: Arch. de méd. exp., viii, 361	Onychia on hand. (Occupation, hostler)	Temperature, 102.2° F.; pulse, 100; eruption on tenth day after admission; pustular eruption on eleventh day after admission	36 days	33d	Temperature, 100.2° F. to 104° F.; pustular eruption on previous day	Tubes of gelatin and bouillon inoculated; on fifth day one colony; 10 c.c. of blood (not all used)	+
III	Brault and Rouget: Gaz. hebd. de méd. et chir., 1896, p. 1212	Malaise for several days	High temperature; vomiting; lungs negative; abscesses on thirteenth day of disease; rales in chest; fever; chills; pustular rash on nineteenth day of disease	20 days	17th	No pustular rash until two days after culture; chills; fever; metastatic foci	?	+
IV	Coleman and Ewing: Jour. Med. Res., 1903, iv, 223	Bitten on hand by horse three weeks before; headache; malaise	Abscess of leg on eleventh day; pustular rash on twelfth day; pyemic foci (thirteenth day); autopsy, glanders and farcy	17 days	16th	One day before death, pyemia	Five c.c. of blood put into bouillon and kept on ice for two days; late flocculent growth	+
V	Denis: Lyon Méd., 1903, i, 664	Pain in right hypochondrium; fever; chills	Muscular abscesses; pyemia; pustular rash three days antemortem	27 days	24th	Pustular rash appeared same day as culture was taken	Bouillon flasks	+
VI	Gabrielides and Remlinger: Compt.-rend. hebd. de soc. de biol., 1902, p. 1147	High fever; typhoid state (Occupation, gardener)	High fever; pectoral abscess; typhoid state	26 days	23d	Complete pyemia	?	+
VII	Van Ingen: Med and Surg. Reports, Presbyterian Hosp., 1904, p. 320	Malaise for three weeks; delirium	Bronchopneumonia; swollen knee; patches in mouth; pyarthrosis; pustular eruption one day ante-mortem	32 days	23d 24th 30th	Broncho-pneumonia; pyemia; pustular rash on twenty-ninth day; positive blood culture on thirtieth day	?	— — +

VIII	Lewis and Duval: Jour. Med. Research, 1904-05, viii, 535	Pain in chest; chills; fever. (Occupation, hostler	Bronchopneumonia; general pyemia; pustular rash	12 days	10th	Complete pyemia; pustular rash	Agar plates; colonies on third day, 150 to 200 to every plate	+
IX	Pilcher: Annals of Surgery, March, 1907, p. 444	Bitten six weeks previous by glandered horse; pain in chest; cough; dyspnœa	Pneumonia; knee involvement on seventh day; general pyemia on ninth day; autopsy; broncho-pneumonia; pyemia; ulcer of larynx	13 days	10th	Pyemia; pneumonia	Agar plates; 48 hours, 35 colonies per plate; raised, white, opaque, viscid; flasks show flocculent sediment	+
X	Meyer and Crohn: Jour. Amer. Med. Assoc., May 16, 1908, p. 593	Patient lived over stable	General pyemia; rales heard in chest; deep muscular abscesses; pustular rash	11 days	7th 9th	Pyemia; pustular rash before cultures	See text (Technique)	+ +
XI	Unpublished case. (See text)	Sudden onset; fever	Deep muscular abscess over shoulder; pustular rash	13 days	13th	Deep prostration; one metastatic focus; pustular rash same day	See text (Technique)	+
XII	Gutowski: Arch. gen. med., 1903, ii, 2186	Pain in right side; pleurisy; malaise	Pleurisy; Bacillus mallei found in pleural fluid; superficial pustules; deep muscular abscesses	27 days	13th	First pustules and first abscess on eleventh day; delirium; profuse rash on thirteenth day	?	—
XIII	Potter: Proc. N. Y. Path. Soc., i, 120	Pleurisy; abdominal pain; fever	Pleurisy; first metastatic focus on fourteenth day of illness; autopsy, glanders and farcy of nasal mucosa, etc.	35 days	21st	(Day of admission)	?	—
XIV	Woodcock: Lancet, 1906, i, 288	Acute pyemia	Rapid acute pyemia beginning in lung; pustular rash	...	...	Day before death	Unsatisfactory bacteriological study	—
XV	Post: Boston Med. and Surg. Jour., 1905, cliii, 580	Pneumonia; small multiple abscesses	Multiple abscesses; no rash; pneumonia; case has lasted nine months; still living at time of publication of paper	9 mos.	...	?	?	—
XVI	Sieur: Arch. de méd. et phar. militaire, 1905, xlvi, 313	Inoculation of finger	Chronic pyemia	2½ yrs.	...	?	?	—

Acute Glanders. This series includes 14 cases, of which, 11 showed positive and 3 negative blood findings. All of these cases, those with positive and those with negative results, clinically follow rather closely one type of the disease. There is almost invariably a history of exposure to equine sources of infection; in 4 cases there was an actual point of inoculation on the hand. The onset is acute, usually with sharp pain in the lower thorax, rapid development of chills, fever, malaise, general prostration, etc. Initial symptoms referable to the lungs are found in over 80 per cent. of the cases. The picture of a general pyemia is gradually superimposed, or presents itself early in the course of the infection. Numerous subcutaneous and intramuscular and arthritic foci of suppuration appear. There is increasing prostration and rapidly supervening delirium, until the breaking out of a generalized pustular rash heralds the closely following fatal issue. The duration of the disease varies from eleven to thirty-six days. The picture is invariably one of a profound pyemia. Pulmonary involvement, either early and probably primarily, or later and secondary to a distal point of inoculation somewhere on the body, is invariably encountered. The localization of the infection, however, in the upper respiratory passages, so characteristic of glanders in the horse, is rarely seen in man. In the latter the picture of farcy, that is, accentuation of the localized point of inoculation with rapidly developing blood invasion and metastatic deposits, supervenes.

As has been noted, of the 14 cases of acute glanders with blood cultures, 11 were positive and 3 negative. A more detailed scrutiny of these findings reveals the following: Of the positive cases, the blood culture was obtained in almost all, when the disease was well advanced, that is, in from one to three days antemortem; in one case (Case X), it was taken four days before death. Considering that the total duration of the symptoms varied from eleven to thirty-six days, it is clear that the positive culture occurred late. In all but one case (Case I) there were already established numerous metastatic abscesses and active pulmonary phenomena; in 7 of the 11 the pustular rash had already made its appearance. In all the cases at the time of culture, there was profound prostration, high continuous fever, rapid pulse, and signs of intense generalized infection.

Of the three negatives cases, that reported by Woodcock (Case XIV) can be disregarded, as the subsequent bacteriology of the whole case was not satisfactorily nor conclusively worked out, nor was the organism accurately identified. Regarding the other cases with negative results, it is to be noted that they correspond in their clinical aspects fairly closely with the above series of cases with positive cultures. The only apparent difference is in the somewhat protracted course (twenty-seven and thirty-five days) and the less rapid appearance of the disseminated metastatic foci. In these two cases the attempt to isolate the organisms from the blood was made

relatively earlier in the course of the symptoms as compared with the previous series; in Case XII on the thirteenth day (total duration twenty-seven days), in Case XIII on the twenty-first day (total course thirty-five days). In neither case was the generalized pustular rash evident at the time of the culture.

The case reported by Van Ingen (Case VII) is of unusual interest for the reason that the blood cultures taken respectively on the twenty-third and twenty-fifth day of the disease were negative, though lung involvement and numerous metastatic abscesses were present. The culture taken on the thirteenth day (forty-eight hours antemortem), and the day just preceding the outbreak of the pustular rash, was positive.

Chronic Cases. Of two cases, both had negative blood cultures. That of Post (Case XV) had run a course of nine months with pulmonary and metastatic foci, but no pustular rash. The patient was still living at time of publication of his paper. That of Sieur (Case XVI) ran a course of two and one-half years, following a local infection of the finger, and presented the symptoms of chronic pyemia without pulmonary or cutaneous signs except toward the very end.

Technique and Technical Results. These have varied considerably in the past, and show numerous wide discrepancies according to the dates and degree of development of the bacteriological methods in vogue at the time. In general, the blood was drawn from the median or saphenous vein and inoculated immediately into either bouillon or agar or gelatin. (In Case IV the blood after being mixed with bouillon, was kept on ice two days before being incubated.) The results seemed good with any of the media used. Colonies appeared rather late (on the third to the fifth day) on the plates. No type colony has been described. In several of the cases the findings were complicated by the simultaneous growth in the culture of streptococci or Staphylococcus aureus, this being in all probability a secondary and late invasion of the blood from some of the numerous superficial and deep foci of suppuration.

It may be of some interest to recount the methods and results used in obtaining cultures in our own cases (Cases X and XI). In Case X (there were two blood cultures). In the first culture 15 c.c of blood was evenly distributed into a flask of plain and 2 per cent. glucose bouillon and into a plate each of agar, serum agar, glucose agar, and serum-glucose agar. Growth appeared at the end of forty-eight hours in both flasks and in all the plates; there seemed to be no decided advantage in any one medium for the cultivation of the bacilli. The appearance of the flasks was as follows: Plain bouillon, after forty-eight hours, slight cloudiness; after seventy-two hours, a heavy murky sediment, the supernatant fluid being unchanged in color. The 2 per cent. glucose bouillon flask showed at forty-eight hours a slight reddish hue, which increased in depth until, after seventy-two to ninety-six hours, the whole presented a deep Bur-

gundy-red color with a heavy sediment at the bottom. The average number of bacilli per cubic centimeter (as estimated from the plates) was 1.6 colonies.

The second blood culture on the same case was performed with identical methods, except that glycerin agar was added to the mentioned media, and the flasks were omitted. Growth again appeared relatively early, in forty-eight hours, the serum-agar plate showing the earliest and most profuse growth. Some blood that had been inoculated into ammonium oxalate[7] after incubation for twenty-two hours was plated out with agar, and showed after forty-eight hours from two to four times as many colonies as were found in the plate that was poured in agar at the bedside.

The colony, as appearing on glucose agar, with or without serum, may be described as a minute punctate (surface) or whetstone shape (deep) colony, of the size of a pin-point, or very slightly larger, brownish by direct and grayish by reflected light, and surrounded by a relatively large greenish area of discoloration in the medium about the colony. This discolored area, when first seen, is 2 to 5 mm. in diameter and increases in circumference rapidly. The morphology of the colony is noteworthy on account of the disproportion between the minute, almost invisible point of growth, and the large spreading greenish area of discoloration. When typical, it resembles very closely that of the typhoid-type colony described by Epstein[8] on glucose-agar plates. In the second culture in this case the average number of organisms per cubic centimeter of the blood was 16.8 (previous culture, 1.6), showing a rapid increase in the number of organisms in the circulating blood in the interval between the two cultures (two days).

The blood culture on the second of our two cases (Case XI) was made similarly to the above-described culture but taken on the operating table a few hours before death. The type of colony on the media was similar to the above; the time of appearance was somewhat earlier, the first colony being observed seven hours after inoculation. The average number of colonies was 300 per cubic centimeter.

From our experience (limited as it is) it would seem that the best results are obtained with the flasks and glucose-agar media; the ammonium oxalate method may apparently be used with safety.

Remarks. The conclusions that may be drawn from the study of the cases of human glanders in which blood cultures have been taken are as follows: In the acute type, cultivations from the blood early in the course of the disease are likely to be negative; those taken at a time later in the development of the symptoms, particularly at or after the time of the appearance of the pustular rash, are invariably positive. Chronic cases have thus far given negative results.

[7] Epstein, Amer. Jour. Med. Sci., September, 1907.

[8] Ibid., August, 1908.

These conclusions are singularly in contrast with the results obtained by investigators of equine glanders. Thus, MacFadyean,[9] an eminent authority on the subject, says: "The glanders bacillus is a tissue parasite, and in all cases of glanders in the horse it is almost entirely confined to the lesions and the discharges from them. It is, no doubt, transported by the blood as well as the lymph channels, but the disease in the horse is never septicemic. Even in the acute cases the bacilli are so sparingly present in the blood that their discovery by microscopic and cultural methods is nearly always impossible, and even considerable quantities of blood fail to transmit the disease by inoculation." The explanation of this difference lies in the fact that the disease in the horse, rarely, if ever, runs so acute a course as it does in man, manifesting itself in the former mainly as a subacute or chronic affection of the respiratory apparatus or as a localized farcy of the skin and related lymph organs. The corresponding type in man, that of the so-called chronic glanders, also gives negative blood cultures. The usual picture of the infection in the human is, however, that of a rapidly developing and grave pyemia with a less considerable and more slowly developing involvement of the related lymphatic channels, but with the rapid evolution of metastatic muscle, bone, and joint foci which have, in all probability, been transmitted through the circulation. It is quite probable that bacteria are present in the blood in human cases early in the disease; in fact, as early as the first metastatic deposit has made its appearance, but they are in such few numbers that with our present methods we are incapable of demonstrating them. It is in the later stages of the disease, when the bacteria become much more numerous in the circulation (as evidenced by the outbreak of a general pustular rash, the pustules containing bacilli), that we are successful in cultivating them. In the chronic cases, those which present the picture of generalized glanders of the respiratory tract, the blood is probably sterile. In those cases with the establishment of farcy buds it would perhaps be possible to cultivate the organisms from the circulation, if the attempt were made just at the time of the new outbreak or at the time of a new deposit of the infection. Between the eruptions of the peripheral foci, attempts at isolating the specific organism from the blood would be, in the greatest probability, unsuccessful.

As to the nature of the bacillemia, the question of chief importance is: "In what manner, do the bacteria enter the blood and circulate therein?" Is it an overflow from the lymphopoietic system? Is it a condition of thrombus formation with multiple minute emboli? Is it a true bacillemia? These are the three views which would seem most tenable. In discussing the bacillemia of typhoid fever, Schottmueller[10] and, later, Coleman and Buxton[11] have suggested that the

[9] Jour. Comp. Path. and Ther., 1904, p 295.
[10] Münch. med. Woch., 1902, No. 38.
[11] Amer. Jour. Med. Sci., June, 1907, p. 869.

bacillemia is secondary to the infection of the lymphoid apparatus of the gastro-intestinal tract, and is an overflow from the lymphopoietic system. Whether the infection of the blood stream in glanders may be the result of an overflow from the lymph system, which is invariably involved with the local lesion, is seriously to be considered. Here we must understand that the lymph gland has no longer become capable of maintaining its function as a filter for the regional lymphatics, and the organisms passing this barrier are swept into the general blood current and so enabled to establish the metastatic foci. If we accept the hypothesis of the possibility of a primary infection of the lungs and respiratory tract by inhalation, we must suppose the bronchial glands to be the source of overflow in most of our cases.

On the other hand, the recent work of Duval,[12] on "Vascular Lesions in Experimental Glanders," has demonstrated that there is a marked reaction of the vessel wall to the specific toxin, and that thrombosis of the venous radicals in the neighborhood of the point of inoculation does occur. In the light of this fact, a priori, it may be supposed that the metastatic foci and free bacteria have a common origin in these thrombi and that the distal deposits result from minute thrombotic emboli. In opposition it must be maintained, that it would be an unusual and rare variety of embolus which chooses the deeper sets of muscles and joints as its site of preference and omits those organs usually the seat of infarcts (spleen, kidney, lungs). Emboli arising from foci of inoculation on the extremities or upper respiratory mucous membrane (nose, pharynx, larynx) should cause infarction in the pulmonary artery system and the lungs, and emboli arising from pathological processes in the lungs would be expected to cause the more usual infarction of the larger abdominal organs.

The third view, that it is a true bacteriemia, should also be taken into consideration. According to this view, we should understand the disease as one in which the bacteria, by injuring with its toxins and so depressing the vitality of the vessel wall, escape into the blood current and establish metastatic foci in those places specific to the glanders bacillus. Probably early in the disease, with a beginning local lesion and a mild degeneration of the vessel wall, there is only a scant, if any, escape of bacteria into the circulating medium, those few cases in literature in which the culture was taken early, neglecting to show the organisms. Further, with the advancement of the lesions and probably inevitable involvement of the respiratory tract there is a greatly increased escape of bacteria through pervious vessel walls, and possibly an active multiplication by reproduction of those organisms free in the current.

On account of the paucity of records and the few cases in the literature, no absolute deduction can be drawn as to the origin and

[12] Jour. Exp. Med., 1907, ix, 241.

course of the bacteriemia. With our improving methods for early recognition of the condition, and with more frequent and earlier cultures, the future will probably contribute much toward the solution of the problems connected with this rare but interesting disease.

The following is the history of the case referred to as Case XI:

The patient, a female, Russian, aged forty-three years, was admitted to the Mt. Sinai Hospital (service of Dr. Gerster), September 22, 1908.

The family history was negative and the previous history uneventful. The patient was married at twenty-seven years of age; had seven children; no miscarriage; she had fever lasting three or four days after the last labor. The present illness began suddenly nine days before admission with chills, fever, and vomiting. She had headache and profuse night sweats accompanied by chills. She vomited clear fluid; not bloody. Synchronously with the onset of febrile phenomena she developed severe pain in the left shoulder with loss of function of the upper extremity on this side. The pain was localized to the shoulder-joint and scapular region, was very intense, boring in character, and severe enough to prevent sleep. Since the onset the fever had slightly subsided, though the local focus continued very tender and painful. She had no abdominal symptoms, and no jaundice.

Upon physical examination on admission the general condition was fair; she was poorly nourished. The glands were not enlarged. The skin was negative, except over the scapular focus, where the swelling was surrounded for a small area by a few raised pustules, each pustule being circumscribed by a small red ring of inflammation. Active motion in the left shoulder was impossible; passive motion unimpaired, but painful. Over the left scapula was a smooth, rounded, firm swelling; no fluctuation was obtained. The skin over the swelling was reddened and elevated. There were cup marks over the swelling and signs of an aspiration wound. The lungs and heart were negative. The pulse was rapid, of good volume, diminished tension, 90 to 96 in rate. The abdomen was negative. There was a profuse, blood-stained vaginal discharge.

September 24. Aspiration of the tumor over scapula by Dr. A. A. Berg. Only a little blood was obtained, although the needle touched bare bone. Blood count: White blood cells, 22,000; polynuclears, 90 per cent.; lymphocytes, 9 per cent.; eosinophiles, 1 per cent. The pulse rate varied between 118 and 128. The temperature was remittent in character, reaching its highest point at about four in the afternoon (104.6° F.), and its lowest point at about four in the morning, when it was 100.2° F. Respirations varied between 24 and 28; regular.

September 25. Blood count: White blood cells, 9000; polynuclears, 91 per cent.; large lymphocytes, 5 per cent.; small lymphocytes, 4 per cent. The pustular eruption has spread from the

scapular region so that it now is more general in character and involves the neck, back of the extremities, and the entire dorsal surface of the trunk.

Operation, September 26: Incision and drainage of acute suppurative myositis. Dr. Berg. A four-inch incision over infraspinous fossa down to bone. Intraspinous muscle is found to be the seat of innumerable miliary abscesses varying in size from pinhead to the size of a grape seed. Multiple incisions into the muscle and excision of a piece for examination. Wound packed with gauze; wet dressing.

The patient's condition was rather poor on leaving the operating room. Soon after, respirations became more regular and strong, the pulse good at the temples, but absent at the wrist. Shortly the patient had a severe chill. The condition became quite poor. She re-acted poorly to vigorous stimulation. Murphy infusions proved ineffectual. She vomited several ounces of brownish fluid. Stomach tube was passed and stomach washed; six ounces of bloody fluid was withdrawn, but the patient immediately collapsed and ceased to breathe after five minutes.

Urine, September 23: Amber; turbid; acid; 1020; very heavy cloud of albumin; no sugar; loaded with hyaline and finely granular casts. September 26: Amber; acid; 1022; heavy trace albumin; many granular and hyaline casts; few epithelial and pus cells.

This patient was in the hospital four days. No evidence of exposure to glanders in any form was obtained. The local focus in the left shoulder was apparently present from the onset. The infection was severe, the course rapid. There was general prostration and the toxemic reaction was intense. Even as early as nine days after the onset the beginnings of the pustular rash were seen. It is noteworthy that in this case there was no other metastatic focus established as far as could be ascertained. The blood culture was taken on the operating table. It will be noted again that the blood culture was taken late in the course of the disease after the appearance and spread of the pustular rash. There was no involvement, as far as could be discovered, of the lungs or any part of the respiratory tract. Permission for an autopsy was refused.

Bacteriological Data. Smears from pus removed at operation showed the morphology of Bacillus mallei. Emulsion of the same material inoculated intra-abdominally into male guinea-pigs caused a characteristic reaction. The organism was further identified by staining methods and cultural means. For notes on the blood findings see under "Technique."

I am indebted to Dr. E. Libman for many valuable suggestions and for his kind guidance throughout the course of the investigation.

A SIMPLE METHOD OF ESTIMATING THE COMMON VARIATIONS AND DEFORMITIES OF THE FOOT.[1]

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Present Methods of Studying the Foot. There is no part of the human body so subject to deformities as the foot, yet there is no part of the body of which the estimation of deformities is so inaccurate and vague. The most common deformity that we see is flat foot, yet the diagnosis of flat foot, so far as its physical signs go, rests on the most ambiguous basis, and in attempting to teach students what we mean by the term, we seldom are able to present a convincing and definite picture. The only method which pretends to any accuracy at all is that of taking a print of the bottom of the foot. The deductions thus made are dependent on the extent with which the sole of the foot touches the underlying surface. Then the shape of the print is supposed to be an index of the lowering of the arch. I see little value in this method. In the first place, the print of the foot only represents the imprint of the skin of the sole. If a foot happens to be fleshy or swollen, it naturally makes a broader impression, and thus seems to connote a lower arch than when a foot is thin and not swollen. Again, a foot which may be very flat may make an almost perfect print, so called, if its arch has not been lowered quite enough to be represented properly in the print. But even if one admits that this method does aid in diagnosis in extreme types of the disorder, it adds nothing in estimating the amount of lowering of the arch in those types which are intermediate. Therefore, in the last few years I have sought for a more definite basis for comprehending certain deviations, with special reference to the estimation of the height of the arch; the aim of this paper is to suggest such a method.

The Estimation of the Deformity in General. In examining the malformations of the body there is no process more valuable than that of studying the relationship of bony landmarks to one another. In fractures of the wrist and of the ankle the relationships of the ends of the long bones are of extreme importance. A splendid example of valuable relationships is Nélaton's line. I may further call attention to such important methods as Bryant's triangle and Meyer's line. These simple geometric methods can hardly be omitted for a clear and proper elucidation of the deformity.

The Selection of Bony Landmarks of the Foot. In the case of the foot no such method has ever been advanced, so far as I know,

[1] This paper represents the substance of a monograph which the author is preparing, in which the work will be presented in greater detail.

and with this in mind I have selected certain landmarks for study. The ones chosen are the lower posterior corner of the internal malleolus, the tubercle of the scaphoid bone, and the lower tubercle on the head of the first metatarsal. The reason for selecting these particular landmarks will be more evident as we proceed. Before advancing, let me briefly examine these landmarks, as it is absolutely essential that the observer know definitely just what part of the skeleton his finger touches in the examination. Fig. 1 is a wash drawing of a disarticulated skeleton which brings into exaggerated relief the landmarks in question.

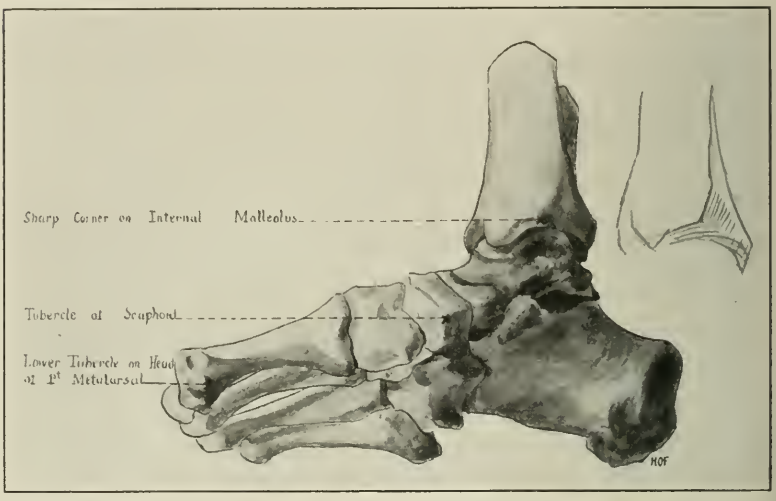

FIG. 1.—The position of the bony landmarks under consideration.

The Lower Tubercle of the Head of the First Metatarsal Bone. In examining the head of the first metatarsal bone in the disarticulated skeleton, we find on its inner aspect two ridges separated by a groove. Each of these ridges ends posteriorly in a prominent tubercle. The upper tubercle does not concern us; the lower one is sharp, and is the one which applies to the present series of observations.

The tubercle of the scaphoid bone is best seen by looking at the scaphoid bone from above. It will then be apparent that the whole internal surface of this bone forms a ridge. The apex of this ridge is the so-called tubercle, being, in fact, the most prominent bone palpable. The tendon of the tibialis posticus ends close to this tubercle.

The Posterior Inferior Corner of the Internal Malleolus. As seen in the sketch, and as will be apparent on examining the skeleton, the internal suface of the tibia is bounded at its lower end (internal malleolus) by three edges—an anterior, an inferior, and a posterior edge. Where the posterior edge strikes the inferior edge a sharp

corner can always be felt. If we run our finger over the part in question we simply determine the most inferior posterior corner of the internal malleolus as thus evidenced.

Changes in the Skeleton of the Foot with Weight-bearing. The investigation of the foot differs from that of the other parts of the body in one respect—that the relationship of the bones becomes altered with weight-bearing. Their alteration is necessarily the first study which most concerns us. As similar studies have been made by other observers, I shall confine myself to the change in the relationship of the landmarks in question.

According to the literature,[2] the view seems pretty well established that when weight is borne the scaphoid travels downward and inward. The downward displacement is, for our purpose, the important thing. In the study of six living feet which I radiographed, with and without weight-bearing, I marked the posterior inferior corners of the tibia, the head of the first metatarsal, and the scaphoid. (The external landmarks selected cannot be accurately identified in the Röntgen pictures.) I then drew a connecting line between the point marked on the internal malleolus and that on the head of the first metatarsal. Figs. 2 and 3 illustrate one of these cases and show that in weight-bearing the scaphoid becomes distinctly depressed with respect to the connecting line. However, as these points on the Röntgen pictures do not exactly correspond to the external landmarks which I selected (earlier described), I marked these landmarks with and without weight-bearing and photographed the feet (Figs. 4 and 5). These pictures point to a similar but more practical conclusion, that in weight-bearing the scaphoid becomes depressed with respect to the line connecting the lower posterior corner of the internal malleolus with the lower tubercle on the head of the first metatarsal.

It is, therefore, evident that in selecting the scaphoid bone, I have taken a part of the foot very susceptible to change in weight-bearing, and on a basis of the above observations and others,[3] not described in this report, I may state that the weight-bearing foot differs from the non-weight-bearing foot according to a definite rule. This rule is that in weight-bearing the scaphoid descends, as measured from the base line as established above.

The Study of the Position of the Scaphoid Tubercle in One Hundred Young Male Adults. After having studied the anatomical data above described, I examined a number of apparently

[2] G. H. V. Meyer, Statik und Mechanik des menschlichen Knockengerustes, 1873, and Statik und Mechanik des menschlichen Fusses, 1886; John Dane, Trans. Amer. Orth. Assoc., 1897; Golobiewski, Ztschr. f. orth. Chir., 1894; Lovett and Cotton, Trans. Amer. Orth. Assoc., vol. xi; Bradford and Lovett, Text-book of Orth. Surg., second and third Editions; Whitman, Orthopedic Surgery; and Quain's Anatomy.

[3] In the monograph another method of measuring the excursion of the scaphoid in weight-bearing is described. This consists in using the angle which it forms with the vertical dropped from the internal malleolus. The results so obtained confirm the main observation.

healthy individuals with reference to these points. The individuals chosen for the observation were young male adults from the evening

Fig. 2.—Röntgen picture of a foot without weight-bearing: *c*, marks the posterior inferior corner of the head of the first metatarsal; *a*, the posterior inferior corner of the internal malleolus, and *b*, the posterior inferior corner of the scaphoid. A vertical line is erected from *b* to *a-c*.

Fig. 3.—Röntgen picture of the same foot as in Fig. 2 with weight-bearing. The same points are marked. Note the increased distance of *b* from the connecting line.

gymnastic class of the Cleveland Young Men's Christian Association. The men were taken as they came along, and no questions were asked. It may be assumed that such a gymnasium would furnish

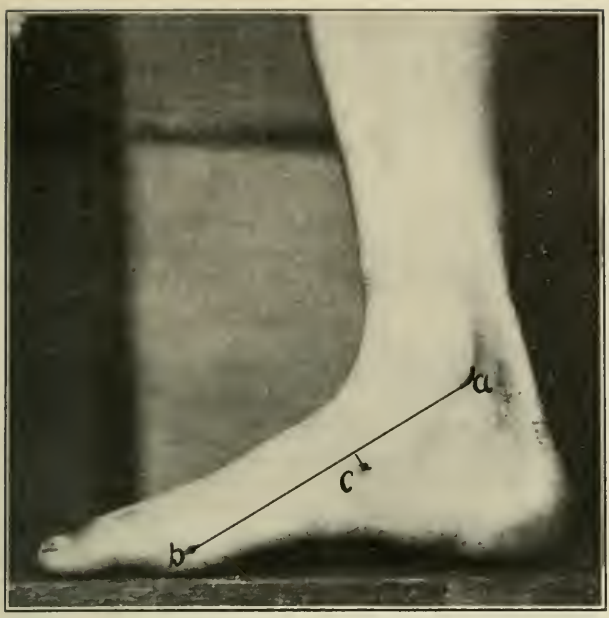

FIG. 4.—Photograph of the same foot as in Figs. 2 and 3, without weight-bearing. The posterior inferior corner of the internal malleolus, *a*, is connected with the lower tubercle on the head of the first metatarsal, *b*, by a line; *c*, represents the scaphoid tubercle.

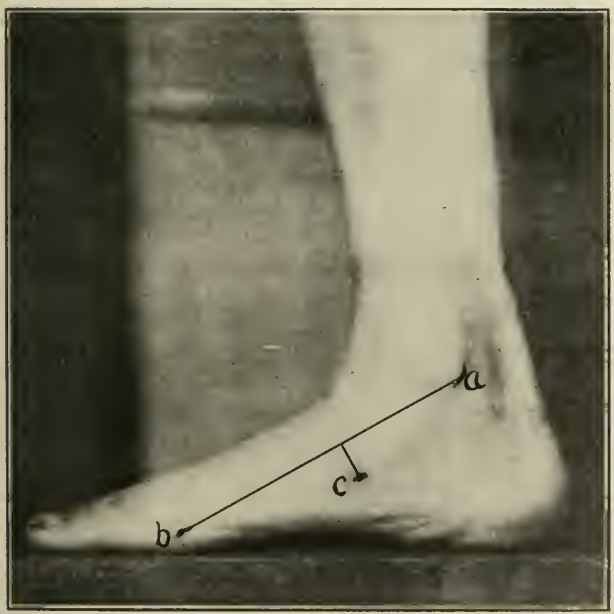

FIG. 5.—Photograph of same foot as in Figs. 2, 3, and 4, with weight-bearing. Note the increased distance of the scaphoid tubercle from the connecting line.

a representative group of healthy young men who conduct the ordinary affairs of life.

In order to make proper observations, a standard method of estimation had to be applied. A wooden box was constructed, consisting of three sides—a base, a posterior side, and a lateral side. Its dimensions were 12 x 12 x 5 inches. The box was placed on the seat of an ordinary chair. The right foot was used. The subject stood on the floor with the left foot straight and the right knee bent. Then the right foot was placed in the box, the calf just touching the upper edge of the box, and the heel resting in the corner (Fig. 6).

Fig. 6.—Position of the foot in the box.

The weight of the body was therefore chiefly transmitted through the left leg. In this way the foot could be examined in a definite and uniform position without weight-bearing. The points marked were those landmarks earlier discussed—the posterior inferior corner of the internal malleolus, the tubercle of the scaphoid, and the lower tubercle of the first metatarsal. The distance of the scaphoid tubercle from the line connecting the internal malleolus with the lower tubercle of the head of the first metatarsal was measured as follows: A thin transparent celluloid rule provided with a scale (Fig. 7) was applied to the foot so that its upper edge coincided with the dots on the head of the first metatarsal and on the corner of the internal malleolus. Then the distance of the tubercle of the

scaphoid from the edge could be read through the transparent rule by means of the scale (the corners of this rule were cut off so that they would not interfere with the sides of the box).

Summary of Important Results. The average age of the one hundred young men was found to be 21.6 years, the youngest being seventeen and the oldest thirty-four years. The average distance

Fig. 7.—The transparent celluloid rule used for determining the distance of the scaphoid tubercle from the connecting line (reduced one-half).

of the tubercle of the scaphoid from the line connecting the internal malleolus with the lower tubercle of the head of the first metatarsal was found to be 0.49 inch.

These results, though interesting, are of less value than the results obtained by arranging the cases in a series graded according to the lowering of the scaphoid tubercle. In the following table the cases were grouped in this fashion.[1]

Distance of the scaphoid tubercle from the line connecting the internal malleolus with head of first metatarsal.	Number of cases.	Distance from scaphoid tubercle to internal malleolus.	Horizontal distance of the tubercle from vertical line dropped from internal malleolus.	Sine of angle formed by line connecting the scaphoid with internal malleolus and the vertical line.	Angle.	
					Deg.	Min.
Inch.						
0	2	2.03125	1.71875	0.8461	57	47
1/16	2	1.875	1.53125	0.8166	54	45
1/8	5	2.0875	1.7	0.1843	54	31
3/16	0					
1/4	11	2.1136	1.6843	0.7956	52	43
5/16	3	2.0625	1.6458	0.7979	52	56
3/8	15	2.1666	1.6833	0.7769	50	58
7/16	2	2.0625	1.5	0.7272	46	39
1/2	19	2.2039	1.6052	0.7283	46	45
9/16	3	2.1666	1.5625	0.7212	46	9
5/8	18	2.125	1.5277	0.7189	45	58
11/16	1	2.0	1.4375	0.7187	45	57
3/4	15	2.3	1.5	0.6521	40	42
13/16	0					
7/8	1	2.375	1.5	0.6315	39	9
15/16	0					
1	3	2.5	1.666	0.6666	41	48
Average	..	2.15	1.59	0.7390	47	40

[1] Only the first two columns apply to the present text, but the other columns may be of interest.

The table shows that there are only nine cases in which the tubercle is less than one-fourth inch below the line, and only four cases in which it is more than three-fourths of an inch below the line. There fore, the great majority of cases show the tubercle depressed from one-fourth of an inch to three-fourths of an inch, which in itself is a large variation (one-half inch).

Conclusions from the Table. In studying the table we must remember that when there is a large variation within normal limits, conclusions based on only one hundred cases are of slight value; but the object of the observations was to determine the nature of the variation rather than its average extent. The important point is that the height of the arch as measured by its index, the tubercle of the scaphoid, does show this marked variation. This variation in a series of non-weight-bearing feet suggests the rule which applies to the individual foot with weight-bearing. In other words, the physiological variation (so far as the scaphoid tubercle is concerned) during weight-bearing function of the individual foot, is a prototype of the anatomical variation of a group without weight-bearing. This conclusion is one which has always been understood by the anatomists. It is simply a perhaps novel method of checking off the older observations; but the main question which is still open is, What is the normal foot? What kind of a foot are we to use on which to base the estimation of a deformity, and in what should a diagnosis of a pathological foot consist? These and allied questions require careful consideration and to this I shall now proceed.

The Average Foot. It was found that in one hundred cases the average depression of the tubercle of the scaphoid from the connecting line was about one-half an inch. As earlier stated, one hundred cases are too few on which to base a fair average. But even if thousands of cases had been used it would be fallacious to reason that all feet in which the scaphoid tubercle is higher than the average are normal, and all feet in which the tubercle is lower are abnormal, because the average is obtained just as much from the figures below as from the figures above. Moreover, the chief point shown in the hundred cases is that there is marked variation in the height of the scaphoid in apparently healthy feet. Consequently, as the average is based on that variation, it has no significance further than what the term indicates, representing simply an average of normal variation. Such an average, therefore, cannot serve as a basis from which to estimate deformity.

The Normal Foot. The problem is not what is the average, but what is the extent of variation which we have a right to expect in the normal physiological foot? In attempting to answer this question we deal with a problem in classification, the same problem which necessarily presents itself in the study of any series of graded objects. Given a series of objects animate or inanimate, which differ from one another only to a slight extent, it is impossible to

state just where we should draw the line dividing one group from another. If we take one hundred shades of color, say yellow, and arrange these shades in a graded series from a light yellow to a brown, the differences between the individual shades which are placed next to each other are so slight as not to be detectable. Yet if we compare the beginning with the end of the series, the difference is very striking. The same holds good in a given structure in the human body in which the individuals are arranged in a series according to the variations in that particular structure. If we take one hundred feet and arrange them in a series with reference to a given point, such as the height of the scaphoid tubercle, and attempt to base a classification on that variation, it is impossible to state where the normal variation ends and where a deformity begins, although the two extremes of the series are strikingly different. This, then, is our problem; the difficulty in dividing the series is apparent; there is no natural dividing line in this arrangement which can be used to define the limits of normality. I did, to be sure, find that the great majority varied between one-fourth and three-fourths of an inch, yet I can lay no stress on these figures, because it is evident that if more than one hundred subjects had been used, the gaps between the figures would have been filled up. Nevertheless, as will be seen, such an arrangement in a graded series is of considerable importance, even if the dividing line between the normal and the pathological does not exist, because this method will help to organize our knowledge on the subject.

The "Type" Foot. It has been shown that the average foot is not a fair basis from which to estimate a deformity, and that it is not possible to make a sharp distinction in the series for purposes of classification. What, then, is a sensible method for systematizing our views? The only course which remains is to make an artificial distinction between the normal and the abnormal, but an artificial distinction which is not only practical, but which has a reasonable theoretical basis. If we study the table of the one hundred cases, we note the two extremes of the series, namely, the one extreme, in which the tubercle is not depressed at all, and the other extreme, in which the tubercle is depressed one inch. There can be no two opinions as to which extreme approximates more closely the physiological foot. There can be no doubt that the foot in which the tubercle is near the connecting line is a better foot than the foot in which the tubercle is one inch below the line. The reasons for this are twofold.

In the first place, as has been shown, the ordinary foot without weight-bearing has its tubercle higher than the same foot with weight-bearing. Now, we certainly know that the foot without weight-bearing has a greater strength and greater efficiency than the foot with weight-bearing, because the power of the former is not yet expended. On the other hand, the foot with weight-bearing is in a static position

in which part of its power is already used; consequently, a weaker foot for further purposes than the foot which has not expended its power. It seems, therefore, that other things being equal, the higher the scaphoid tubercle is, the greater the efficiency of the foot. Consequently, if we see a large number of feet all under the same static conditions, that is, all without weight-bearing, and find this variation of elevation of the scaphoid tubercle, it must mean that those feet in the group with the highest scaphoid (of which the prototype in the individual is the non-weight-bearing foot) must approximate the highest functional extreme.

In the second place, it is apparent from what has come before, and also from a number of healthy feet in which I studied the functional power (besides those used in the present investigation), that all those feet in which the tubercles were high with respect to the connecting line presented high arches and high insteps. It is, I admit, very seldom that the tubercle falls exactly on the line, but quite a few fall very close to it, either just above or below. All feet which showed this property I have found to be extremely well formed. In addition to the elevated arch and high instep, the proportions were extremely attractive and corresponded to what an art critic would deem a beautiful foot. Why is it that the artist considers such a foot beautiful? Simply because, as part of the body, it seems well adapted to carry on its functions. From his point of view, it is a more graceful thing than when it is not so well adapted. It is, therefore, plain that the commonly acknowledged beautiful foot is not acknowledged so because some men happened by chance to call the thing beautiful, but simply because it is the natural kind of foot which seems best fitted for its purpose. In short, both from the point of view of the physiologist and from the point of view of the artist, the foot with the high arch expresses strength and adaptability to function. Therefore, such a foot connoting strength and suggesting beauty, coming naturally at the extreme of a carefully selected series, must have some significance. Why not select this as an arbitrary type, remembering, of course, the method by which the type was chosen? If we do this, and bear in mind that it is only an artificial selection we have a fair starting point on which to base our estimation of deformity. Even if it is asserted that such a method is not completely scientific, it is at least reasonable, certainly more reasonable than having no method at all. A fairly good typical foot is illustrated in Fig. 6.

The Estimation of the Deformity of the Foot. Let me, therefore, suggest the following rule for estimating the deformity of the foot: Other things being equal, the foot is deformed in direct ratio to the deviation of the scaphoid tubercle from the line connecting the lower tubercle on the head of the first metatarsal bone with the lower posterior corner on the internal malleolus. Now, I am careful to state, "other things being equal," because I believe

that no estimation is of the slightest value without considering these other things. In any problem of this sort we have no right to take one physical sign by itself and make our estimation on that sign alone; we must always consider that point in relation to other clinical evidence. In flat foot, for example, we may have a number of considerations before us—the abduction of the foot, the pronation of the ankle, the prominence of the internal contour, etc. All these things may be correlative to the lowering of the scaphoid tubercle; if they are, we are probably dealing with a pathological foot, but if they are not, we must consider the relative value of each piece of evidence, depending in each case upon the individual circumstances of that case. I believe that the lowering of the arch is an important point in considering the deformity and that the best index for estimating the lowering of the arch is the scaphoid tubercle, but that is all the further I am willing to go. A lowered arch does not necessarily mean a deformity, it simply means a variation; but a badly lowered arch means deformity, and what is bad we must determine in the individual case. As will be shown (Case V), a foot may be very abnormal even if the tubercle is above the connecting line. The other physical signs in such a case are more pathognomonic than the height of the tubercle.

The Consideration of the Size of the Foot. With regard to the application of the rule to individuals of different size and age, I believe that its language is sufficiently broad to cover all ordinary cases, provided that one uses reasonably good judgment in the individual. Of course, an equal lowering of the scaphoid tubercle in two feet, one large and the other small, would necessarily be of greater significance in the latter. Thus a depression of the tubercle of three-fourths of an inch in a six-year-old child is of greater consequence than an equal depression in an adult.

The Definition of Flat Foot. It has been shown that the variation within normal limits in a group of feet is similar to the normal change in the individual foot with weight-bearing. From this it was naturally inferred that the more depressed a non-weight-bearing foot is, the less it is able to change with weight-bearing, and consequently that such a foot has lost part of its latent strength. Reasoning from this, I showed that a sensible classification could be made if we regarded such a foot as typical of highest function and beauty which most closely resembled the healthy non-weight-bearing foot, namely, one in which the tubercle is close to the connecting line, providing no other pathological sign co-exists. Flat foot, therefore, may be defined as a position of the bones, resembling that of physiological weight-bearing, which does not disappear when weight is removed, the amount of flattening being in the ratio of the lowering of the scaphoid tubercle.

From the above, it is clear that if we are to take such a foot as typical of beauty and function as has been described, then we can

consider few feet well formed, and that we must regard the great majority of human feet as flat. This is true; but if we regard them in this manner we must speak of the great majority as physiologically flat, or, better, as anthropologically flat. They are only flat as based on the estimation from a type, the type being based on the consideration of the highest function and beauty, and consequently of relatively rare occurrence. It therefore becomes clear that if only a few feet are best adapted to carry on their function from this point of view, and if the great majority of feet are more or less deficient as compared with the high standard that has been set, the diagnosis of flat foot can have little practical weight unless it connotes bad function and pain. If we consider an individual foot, and note that it is moderately flat, it does not mean that the foot is unable to carry on its function; it simply means that it might have been better if it were built more according to the type. If, however, there are signs of disability accompanying the signs of depression of the arch, such as pain and muscular spasm, then our diagnosis is established. We have a similar problem in the consideration of the physical signs of other parts of the body, for example in the consideration of hypertrophy of the heart. Hypertrophy of the heart may occur in apparently healthy individuals, and may not in itself cause symptoms; but once urge that heart to carry on functions beyond the power of compensation, and it loses its resistance to future dilatation much more readily than the heart which has not previously been hypertrophied.

From what has gone before, it is evident that the use of the bony landmarks of the foot as an aid to diagnosis must be made with the greatest caution. When one considers the extreme variation in the normal, when one considers the artificial manner of selecting one kind of foot as typical, one feels some hesitation in advocating the measure as an aid to diagnosis at all. Nevertheless, if the observer is careful, he may in many cases derive some value from such a method. But if he uses it, he can never lose sight of the value of the other points in diagnosis; he simply has one more physical sign to use as evidence. If he goes farther than this and bases his whole diagnosis on that one sign, he is likely to fall into grave error.

Clinical Application of this Method. If one follows such a suggestion as has been made, that of estimating the variation and deformity of a foot from an artificial type, it is necessary that the estimation should rest upon a standard method of examining the patient. Such a method has already been shown, namely, that of using a box of given dimensions, placing it at a given height, and using such a transparent ruler as earlier described. The only measure necessary is that of the distance of the tubercle to the connecting line. Simple as such apparatus is, even these things may be omitted for ordinary work. A method that I suggest is to let the patient stand on his one foot and to have him place the other foot

on an ordinary chair with the inner side of the foot facing the examiner. The weight of the body must be transmitted through the leg which is resting on the floor. Then the landmarks may be marked with a skin pencil (a good skin pencil is a grease paint pencil such as women use for painting their eyebrows), and then a simple ruler or piece of string may be applied to the points marked on the internal malleolus and the head of the first metatarsal. From this the distance of the tubercle can easily be measured. This is all the accuracy that is necessary, and requires no further apparatus. If such a box is used as has been described, it may be used both for the left and for the right foot simply by letting the patient stand on his other foot and by inverting the box. This being the method, the simple rule is: Other things being equal, the foot is deformed according to the deviation of the scaphoid tubercle from the line connecting the lower posterior corner of the internal malleolus with the lower tubercle on the head of the first metatarsal. In order to illustrate the practical applicability thereof, let me cite a few cases:

I. Feet Showing Asymmetry with Reference to the Elevation of the Scaphoid Tubercle. (*a*) *The Tubercle on the Unhealthy Side Lower than on the Healthy Side.*

Case I.—U. B., aged twenty-two years, by occupation a barber, was first seen at my office October 16, 1908. Three months ago the patient began to have trouble in his right foot after previous good health, this consisting in disability, pain, and swelling, and had grown steadily worse. There has been no history of injury. The left foot has not bothered him and has not become swollen. Examination showed the patient limping on his right foot, which was in a position of abduction. There was swelling in the region of the internal malleolus. The skin was somewhat clammy to the touch, and the region of the swelling was bluish white. There was marked stiffness on inversion. Examination of the relation of the tubercle of the scaphoid to the line connecting the internal malleolus with the head of the first metatarsal showed it to be seven-eighths of an inch below. The tubercle of the left foot was found to be five-eighths of an inch below the connecting line. There was no swelling and no signs of spasm in the foot.

It will, therefore, be seen that the lowering of the tubercle in this case, as compared with that on the other side, is correlative to other signs, but the amount of lowering is of considerable help, showing the anatomical difference between the diseased and the other foot. The diagnosis was acute flat foot (perhaps infectious) and irritated by occupation.

Case II.—E. B., business man, aged twenty-four years, was first seen by me September 21, 1908. Several weeks ago he strained his feet playing tennis; before this time he had had little or no trouble. Now there was pain in the right foot, especially on use, and chiefly referred to the arch and the ball of the foot Examination showed

the feet well developed, without spasm, swelling, or calluses. There was no redness and no tenderness. In the right foot, however, the tubercle was found to be seven-eighths of an inch below the connecting line, and in the left foot five-eighths of an inch below. The inner contour of the right foot bulged somewhat more than that of the left.

It is then apparent that if the measurement was accurately made, a slight deformity was present as compared with the other foot, and it is plain that the plantar ligaments must have been weakened and stretched.

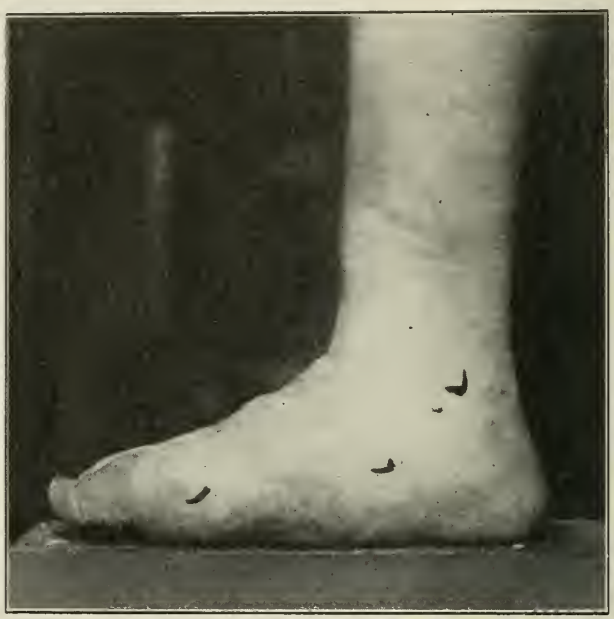

Fig. 8.—Case III. Osteosarcoma. Note the complete obliteration of the arch out of proportion to the amount of lowering of the scaphoid tubercle.

Case III.—F. F., aged thirty-nine years, a laborer, was first seen at the Lakeside Dispensary August 10, 1908. One year ago the right foot began to swell and the patient noticed his arch lowering. The swelling became worse and the arch grew flat. He had some pain and limped. He was treated for flat foot. Examination of the foot (Fig. 8) showed marked swelling, including the whole tarsus from the ankle to the metatarsal region. The foot seemed completely flat. The bony landmarks could not be distinctly felt. What seemed to be the tubercle of the scaphoid was one-half an inch below the connecting line; that on the other foot was about one-fourth of an inch below the connecting line.

Here we had an apparent incongruity of facts. There was bulging

of contour, marked flattening of the arch, and marked swelling, whereas the tubercle, which, if it were a flat foot, should have been markedly depressed, was only one-half an inch below the line. A Röntgen picture was taken and showed plainly that a mass of bone and new tissue had formed and included the whole tarsal region. The diagnosis of osteosarcoma was made. The seemingly contradictory evidence was the reason for taking the Röntgen picture, because, if the arch were as flat as it seemed to be from inspection, the tubercle should have been much lower.

(*b*) *The Tubercle on the Unhealthy Side Higher than on the Healthy Side.*

Case IV.—M. G., a servant, aged about thirty-five years, was first seen at the Lakeside Dispensary October 19, 1908. The preceding April she sustained a fall of eight feet, landing on the sole of the right foot. The foot became swollen, and has disabled her since. She had been treated at another hospital by having the foot strapped. There had been some relief. The pain was chiefly on the outer side of the foot. Examination showed the right foot permitting almost no motion in inversion, but fairly good motion in dorsal and plantar flexion. The restriction of motion seemed to be chiefly due to mechanical bony interference rather than spasm. Some induration was to be felt under the malleoli and on the dorsum near the cuboid. The tubercle of the scaphoid on the affected side, the right, was one-eighth of an inch below the connecting line, and on the good side three-eighths of an inch below. A Röntgen picture was taken and showed a lesion of the calcaneo-astragalar joint, and signs of impaction in the astragalus with fusion of the calcaneo-cuboid joint.

It seemed that in the original injury the astragalus had been crushed into the os calcis, indirectly injuring the cuboid. This anatomical lesion would explain very well the relative elevation of the tubercle of the scaphoid on the affected side, because the malleoli had been lowered by the crushing of the joint beneath, whereas the position of the scaphoid had not become altered.

Case V.—M. K., a school boy, aged about twelve years, had infantile paralysis when a child, causing the present deformities. Examination showed the left foot in the position seen in Fig. 9, with the dorsum very high, the toes markedly contracted, the dorsal flexors of the toes prominent, the arch extremely high, the ball of the big toe prominent, and the plantar fascia very tense. This kind of a foot is immediately recognized as a claw foot or non-deforming club foot. The other foot was in equinus, so that it could not be used as a healthy foot for comparison. Measurement of the scaphoid tubercle on the claw foot showed it to be one-fourth of an inch above the connecting line, indicating the extreme elevation of the arch.

The first three cases hardly require additional comment, except to

call attention to the fact that the measurement of the relative depression of the scaphoid tubercle is of considerable help when making comparison with the other foot. In Case IV the reasoning from these landmarks should have carried with it a diagnosis of fracture, even without a Röntgen picture. In Case V the tubercle is actually elevated above the connecting line. It might be thought that, according to our rule, in which it is stated that the foot is deformed according to the deviation of the scaphoid tubercle from the connecting line, there would be confusion in distinguishing such a foot from the "type" foot, but this case brings out the point on which I laid special stress, namely, that other things have to be equal. Here, to be sure, the scaphoid tubercle is markedly elevated, and

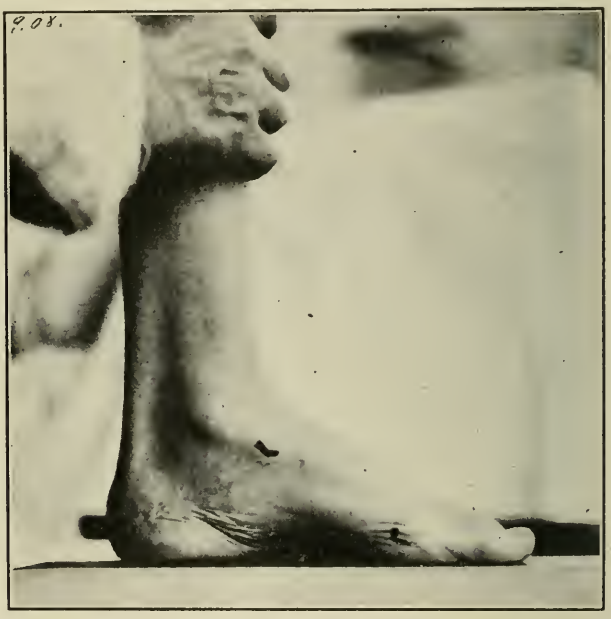

Fig. 9.—Case V. Claw foot (non-weight-bearing) from infantile paralysis. Note the elevation of the scaphoid tubercle.

would by itself suggest beauty and function, but the cause of the high elevation, the paralysis (namely, paralysis of the interossei), has also brought about coincidental deformities, that is, contractures. In other words, other things are not equal. There are pathological signs which make up for the height of the arch, and the co-existence of these other pathological signs and symptoms would prevent us from calling such a foot a "type" foot.

II. Symmetrical Depression of the Scaphoid Tubercle. These are the most common group of cases, and, of course, include the ordinary flat foot.

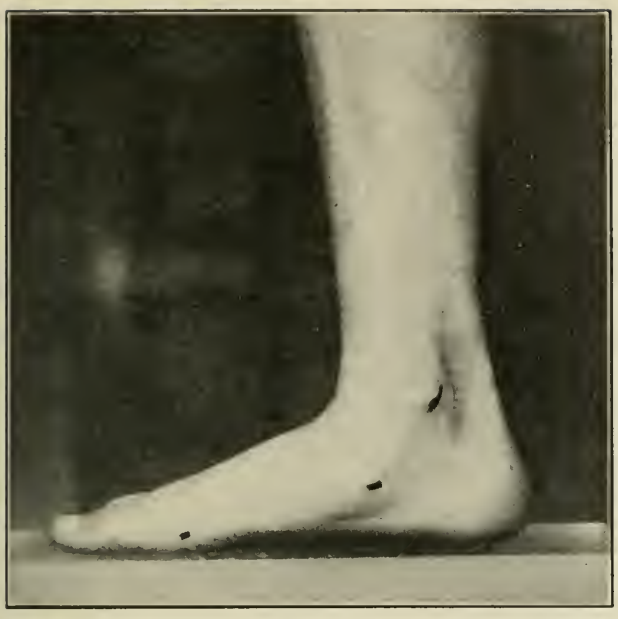

FIG. 10.—Case VI. Flaccid flat foot of moderate degree (non-weight-bearing) in a subject aged ten years. The tubercle is ¾ inch below the connecting line.

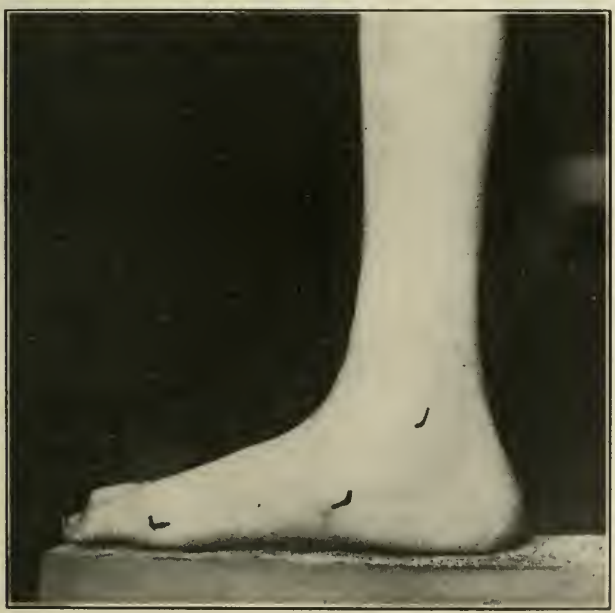

FIG. 11.—Case VII. Rigid flat foot, marked (non-weight-bearing). The tubercle is one inch below the connecting line.

Case VI.—F. M., aged eleven years, was seen at the Lakeside Dispensary, March 15, 1908, complaining of pain in both feet for some time. No swelling was noticed. Examination (Fig. 10)

Fig. 12.—Röntgen picture of Case VII. The metal mark shows the position of the scaphoid tubercle.

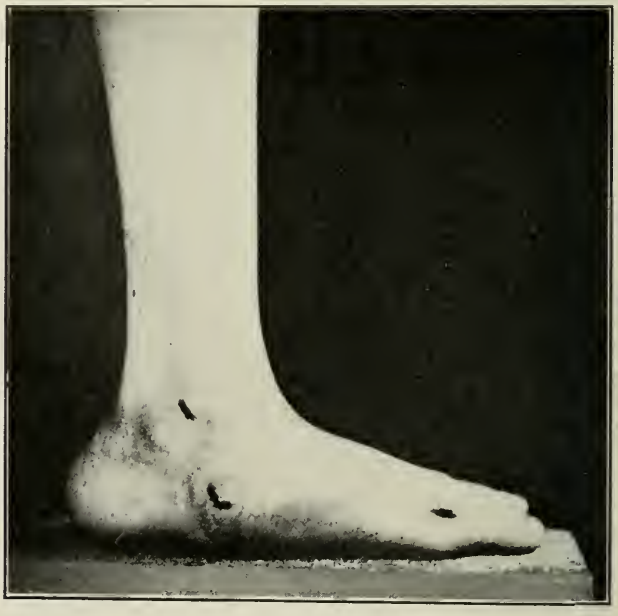

Fig. 13.—Severe valgus following infantile paralysis. The mid-point represents not the tubercle of the scaphoid but the head of the astragalus.

showed the feet long, lanky, and abducted. There was no stiffness, and the arch was apparently low. The tubercle of the scaphoid was about three-fourths of an inch below the connecting line on each foot without weight-bearing. A diagnosis of moderately flaccid flat foot was made.

Case VII.—W. H. Five years ago this boy began to limp. His ankles and wrists became swollen. Since then he has stumped about with his feet in the same condition, and has had much pain and swelling. Examination (Fig. 11) showed marked valgus, abduction, and pronation of the ankles, which were swollen and bluish. On palpation the tubercles of the scaphoid were found to be very low, about an inch below the connecting line. The peroneal tendons were tight. There was stiffness in all directions. The patient toed out when he walked. In this case I placed a metal mark on the tubercle and took a Röntgen picture, which proved that this prominent point on the contour was really the tubercle (Fig. 12). A diagnosis of extreme rigid flat foot was made.

Both these cases illustrate common types of flat foot and the relative significance of the depression of the scaphoid tubercle as suggesting the severity of the deformity.

It is, perhaps, worth while to call attention to a possible source of error. In very severe types of flat foot (as from infantile paralysis) the head of the astragalus twists so far inward as to suggest that its prominence is the tubercle of the scaphoid (Fig. 13). Experience and the study of the skeleton, however, will prevent the observer from being misled.

A FURTHER CONTRIBUTION TO MY "SIMPLE" METHOD FOR THE QUANTITATIVE DETERMINATION OF PEPSIN IN A GIVEN GASTRIC JUICE.

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It is a well-known fact and has been repeatedly demonstrated, by me among others, that the essential factor in the digestion of proteids is pepsin. It is, therefore, of the greatest importance in all cases of chronic gastric disease, especially that form attended with diminished or inhibited secretion of hydrochloric acid, to determine (*a*) whether pepsin is present, and (*b*) if so, whether it is present in normal quantity, in increased or diminished quantity, or (*c*) whether it is altogether wanting.

Various methods have been devised for this purpose, all more or less complicated and not adapted to the needs of the busy practi-

tioner. In 1905 I published[1] my method for the quantitative determination of pepsin found in a given gastric juice. In that article I claimed that my method is the simplest devised, and gives results that are perfectly reliable and in a manner readily and easily interpreted. Since then the application of the method in a much greater number of cases has, to me at least, substantiated all the claims made for it at the time, and has also brought to light some new facts which give it additional value. It is for these reasons that I bring this method once more to the attention of my colleagues.

Since the publication of my first paper two other methods,[2] making the same claims to clinical applicability, have been devised and published. The first is the method of Jacoby and Solms,[3] based upon the fact that a cloudy solution of ricin[4] in dilute hydrochloric acid becomes clear on the addition of a peptic ferment, or a solution thereof.

The technique of the method is as follows: 0.5 gram of the cheap ricin[5] is dissolved in 50 c.c. of a 5 per cent. solution of sodium chloride, and filtered; 2 c.c. of this solution, which is somewhat turbid, is put into each of five test-tubes, and 0.5 c.c. of a decinormal solution of HCl added. The ricin solution becomes at once milky, opaque. The gastric filtrate obtained after an Ewald and Boas test breakfast is variously diluted according to the degree of acidity it is found to possess: The hyperacid, from 1 to 100 to 1 to 10,000; the normal (40 to 60), from 1 to 100 to 1 to 1000. If the gastric juice to be tested is, say, of the normal variety, a 1 per cent. dilution thereof is prepared by adding to 1 c.c of the filtrate 99 c.c. of distilled water. To each of the test-tubes prepared as above, and numbered respectively 1, 2, 3, 4, 5, there is added of the boiled gastric juice (the ferments it contains are destroyed by boiling) to No. 1, 1 c.c; to No. 2, 0.9 c.c.; to No. 3, 0.8 c.c.; to No. 4, 0.5 c.c.; to No. 5, 0 c.c. (none of the boiled filtrate is added). This done, there is further added of the 1 per cent. dilution (unboiled) to No. 1, 0 c.c. (nothing); to No. 2, 0.1 c.c.; to No. 3, 0.2 c.c.; to No. 4, 0.5 c.c.; to No. 5, 1.0 c.c. Each of the test-tubes contains now altogether 3.5 c.c., and we have dilutions of the filtrate, No. 2, 1 to 1000; No. 3, 1 to 500; No. 4, 1 to 200; No. 5, 1 to 100. No. 1, to which no ferment (that is, none of the 1 per cent. dilution) has been added, serves as a control tube. The tubes are now corked and placed in the thermostat and

[1] Einfache Methoden zur quantitativen Bestimmung der vom Magen ausgeschiedenen Enzyme, Archiv f. Verdauungskrankheiten, 1905, xi, 145.

[2] Other methods besides these have been devised since, but they are strictly laboratory methods.

[3] Ztschr. f. klin. Med., vol lxiv.

[4] "Ricin," Kobert, Merck: Exceedingly poisonous agglutinin from the seeds of Ricinus communis L. and closely allied to the bacteriotoxins; yields about 25 to 30 per cent. ash. White Powder.—Sol.: 10 per cent. solution of sodium chloride.—*Caution*: Handle very carefully; small particles in abrasions, eye or nose, may prove fatal; intravenous injections of 3 grains (0.2) are fatal.—Merck's Index, 1907.

[5] An impure preparation made by the Vereinigten Chemischen Farbwerke, Charlottenburg, Berlin.

allowed to remain therein for three hours; then they are taken out and examined to see which dilution has clarified the ricin solution. If a thermostat be not at hand, the procedure may be carried out at room temperature, but then requires a longer time. Having found on repeated examination that, of filtrates of normal acidity, 1 c.c. of a dilution of 1 to 100 (1 per cent.) just cleared the ricin solution in three hours, he called the pepsin content of this 1 c.c. 100 pepsin units, establishing it as his standard for comparison.

Witte[6] has suggested certain modifications in this method. Having found in a series of cases of supposed normal acidity that the pepsin content of 1 c.c. of a dilution of 1 to 100 did not reach 100 pepsin units, he begins his dilutions of gastric juices of normal or slightly lowered acidity with 1 to 50. In cases of marked hypoacidity or anacidity he may use the undiluted filtrate for the first tube and begin his dilutions with 1 to 1. As the HCl element varies greatly in quantity, he would have all gastric juices to be thus examined neutralized with a decinormal solution of potassium hydroxide and then brought to a uniform degree of acidity by the addition of decinormal solution of HCl.

Leaving out of consideration the question of the handling of a substance so fraught with danger, and, furthermore, leaving out of consideration the criticism of the method made by Gross and by Fuld and Levison, namely, that the ricin is full of impurities, and therefore all determinations based upon it are unreliable, a moment's thought will at once demonstrate that it is not a clinical method at all. The solutions and the many dilutions to be prepared, all of which, to give any value to the work, must be made with the greatest accuracy, a minute quantity more or less vitiating the result one way or the other, require more time and more care in their preparation than can be spared by the man in active practice who has a number of gastric juices to examine every day and other duties connected with his work to attend to.

Then, after all that painstaking work, what has the inquirer learned? He has learned that 1 c.c. of a certain dilution of a certain gastric juice is equal to the task of clarifying a certain solution of ricin. Does this reveal to him directly anything as to the digestive capacity of the stomach of the patient from whom the gastric juice was derived? Nothing at all. Only by a long stretch of the imagination and a long mathematical calculation based on figures that are essentially hypothetical (he even does not know the albumin content of the ricin he has used) could he form an idea of the possible digestive capacity of that particular stomach.

The second method is that of Fuld;[7] Levison[8] later published additional details. For his purpose he makes use of a 0.1 per cent. (1 to 1000) solution of edestin, a substance derived from hemp

[6] Berl. klin. Woch., 1908, p. 643.
[7] Münch. med. Woch., 1907, p. 1455.
[8] Amer. Med., February, 1908.

and belonging to the category of the globulins. The edestin is dissolved in a solution of dilute hydrochloric acid having an acidity of 30 (corresponding, as he says, to the free HCl found normally after a test breakfast of Ewald and Boas). Six test-tubes (preferably of a diameter of 1 c.c.) are filled with various quantities of gastric filtrate obtained after an Ewald and Boas test breakfast and diluted according to its degree of acidity from 1 to 100 to 1 to 10 (for anacidity). To each of these tubes 2 c.c. of the edestin solution is quickly added and the contents then allowed to digest for thirty minutes at the room temperature. At the expiration of the time named, strong ammonia is allowed to run down the sides of the tube until a layer is formed above the contents. The tubes are now examined by reflected light against a black background. If digestion is complete, nothing is seen, but if incomplete a distinct white ring of albumin is seen between the two layers—just as in Heller's test for urine.

By finding the test-tube in which a distinct ring ceases to be formed, the point where digestion is complete is determined, and by that the strength of the pepsin content of the given gastric juice. Thus, if the tube with 0.7 c.c. gastric filtrate has no ring, and the next tube with 0.4 c.c. thereof has a ring, it can be said that the gastric juice of that stomach has a pepsin strength of 0.7 in a 1 to 100 dilution. This can be recorded in another way: The dilution of the stomach contents is multiplied by the amount of the edestin solution used and the product divided by the amount of gastric filtrate placed in the tube; thus, 100 x 2.0 (read as 20), divided by 0.7, equals 285+. The pepsin value of that gastric juice is then said to be 285+.

Or the test can be carried out in this way: Instead of the ammonia, a 10 per cent. solution of sodium chloride is added to the tube. This will produce cloudiness proportionate to the degree of digestion that has taken place. The more ample the digestion, the lighter the cloudiness; in other words, the greater the quantity of the solution that will be required to render the contents of the tube cloudy. Thus, at the outset 0.5 c.c. of the salt solution may produce turbidity, while half an hour later it may require 2 c.c., or even more, to produce the same result. In this wise the test can be made with but a single tube.

It can be made in still another way: A 1 per cent. solution of edestin is prepared as above directed. Of this a sufficient quantity (5 or even 10 c.c.) is placed in a test-tube and digested with a not too strongly diluted gastric filtrate. At the expiration of half an hour solid sodium chloride is added, the tube well shaken, and the height of the precipitate measured. Levison says, in further explanation, that "the precipitated albumin very quickly assumes a definite volume, and as it exists under no considerable pressure, its volume is dependent upon the pepsin amount, and allows itself to be read off after standing a short time."

It will be at once apparent to the reader that this method also is not the simple one it is held out to be. All that has been said in criticism, in this one regard, of the method of Solms can be said of that of Fuld, namely, it is too complicated; the making of the principle solution, the dilution of the gastric filtrate, the filling of a number of tubes with minimal quantities which, to give reliable results, must be accurately measured (that it is not an easy matter to measure accurately 0.7 or 0.4 or 0.3 c.c. will be acknowledged by all laboratory workers, and is clearly shown by the numerous instructions given by the writer of the article as to the use of graduated tubes, pipettes with rubber bulb, etc.)—0.05 c.c. too much or too little, and the whole calculation goes wrong.

Then, after all the work has been done, what has the examiner learned? Nothing! His question, whether the gastric juice of a certain patient has enough pepsin content to carry on normally the process of digestion, remains unanswered.

A method to be clinical, that is, adapted to the needs of the man of large practice with not much time to spare, must be possessed of the following qualifications: (1) It must be simple in its technique, and every step thereof definite and accurately measured; and (2) it must give a result at once apparent, at once understood, without the necessity of long and complex calculations or of the translation by way of the imagination of hypothetical figures into those of a supposed actuality.

Of all the methods hitherto described, only the one devised by me measures up to this standard. It is as follows:

(*a*) Ten centigrams (exact weight) of egg albumen (white of egg[9]) is coagulated in the following manner: The egg is put into a pot of cold water, which is then covered with a lid and set on to cook. It is allowed to cook for ten minutes after the water has begun to boil—in all, twenty minutes from the time it was put on. The egg is then taken out and allowed to cool, either by just setting it out in a saucer or by putting it into cold water. Furthermore, to imitate the usual way in which food is or should be put into the stomach, the segment of albumen is divided into two parts. (Observation has shown—just as it has long been known clinically—that when it is thus subdivided the gastric juice can act upon it more quickly. The action being from all sides, it is naturally more effective when there are eight sides of attack for a given quantity than when there are but four.) (*b*) The two parts of coagulated egg albumen are put into 10 c.c. of the gastric filtrate from the stomach contents extracted one hour after an Ewald and Boas test breakfast; and (*c*) they are then placed in the thermostat, which is kept at 38° C. The time in which the 10 centigrams is digested entirely, partially, or not at all, will give us a correct idea as to the status of the pepsin secretion in the case under examination.

The results obtained with this method are well shown in the following tables:

[9] Hens' eggs only.

TABLE I—RAPID DIGESTION (less than the normal period.)

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion.	Remarks.
I F.	N. K.	42	62	Hyperacidity	2.23	
II M.	A. A.	38	48	Hyperacidity, subacute gastritis, constipation	3.00	15 centigrams in 10 c.c. filtrate digested fully in 5¼ hours.
III M.	F. H.	66	99	Hyperacidity, constipation	3.00	15 centigrams in 10 c.c. filtrate digested fully in 3½ hours.
IV M.	R. M.	56	77	Hyperacidity	3.10	
V M.	S. B.	62	77	Hyperacidity, constipation	3.10	
VI F.	M. B.	12	28	Deficient HCl; in the climacteric	3.15	Eats too heavy food. Her teeth are bad, and she cannot masticate well.
VII M.	A. E.	50	68	Hyperacidity, constipation	3.21	
VIII M.	J. K.	47	69	Eats too much and too heavy food	3.26	Adenoids and obstructed nose.
IX M.	A. K.	71	102	Hyperacidity and constipation	3.29	
X F.	B. C.	38	64	Hyperacidity, constipation	3.30	6 centigrams in 5.5 c.c. filtrate digested in 3½ hours.
XI F.	Y. G.	35	58	Constipation, inflamed hemorrhoids	3.30	
XII M.	J. F.	No reaction to phlor. gluc. v.	24	Constipation, colics, deficient HCl	3.30	With the addition of HCl c. p., gtt. ii. Without the acid at the end of 11 hours, the edges only slightly thinned.
XIII M.	J. L.	46	67	Constipation, hyperacidity	3.40	
XIV F.	E. A.	20	54	Constipation, dyspepsia	3.42	
XV M.	A. B. F.	40	57	Hyperacidity, constipation	3.49	His father suffered from hyperacidity, and finally died in an attack of hematemesis.
XVI M.	B. I.	29	58	Constipation	3.49	
XVII M.	B. G. C.	70	115	Hyperacidity	3.50	15 centigrams in 10 c.c. filtrate in 3 hours 50 minutes all digested except one small particle. That remained even after 4 additional hours.
XVIII M.	J. P.	39	54	Neurasthenia, constipation, hyperacidity	3.50	Three months later similar trial; 10 centigrams in 10 c.c. filtrate digested in 3 hours and 30 minutes.
XIX M.	N. W.	63	85	Hyperacidity, constipation	3.56	
XX M.	E. W.	41	59	Neurasthenia, hyperacidity	4.00	
XXI F.	S. M.	47	69	Hyperacidity, constipation	4.00	
XXII M.	E. M.	19	37	Atony of stomach	4.00	Lives in a warm climate Eats food that is too heavy.
XXIII M.	L. R.	56	87	Constipation, hyperacidity	4.00	
XXIV F.	M. D.	22	54	Subacute gastritis	4.00	Had a gastro-enterostomy made on her before I saw her.
XXV M.	G. W. F.	39	71	Hyperacidity	4.09	
XXVI M.	I. B.	17	31	Constipation, slight deficiency in HCl	4.12	
XXVII F.	S. W.	27	57	Subacute gastritis	4.15	
XXVIII M.	L. B.	22	46	Constipation, secondary atony of stomach	4.15	
XXIX M.	A. W.	41	64	Hyperacidity, constipation neurasthenia	4.15	

TABLE I.—Continued.

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion.	Remarks.
XXX M.	I. C.	48	79	Hyperacidity, constipation	4.19	
XXXI M.	A. M.	50	61	Tabes, (beginning of) hyperacidity	4.20	
XXXII M.	M. A.	45	61	Hyperacidity, constipation	4.20	
XXXIII F.	S. H.	08	24	General debility, neurasthenia	4.20	
XXXIV M.	G. M.	38	64	Hyperacidity	4.25	
XXXV M.	Ph. M.	29	50	Constipation, uric acid gravel	4.25	
XXXVI F.	Mrs. B.	28	55	Hyperacidity	4.29	Determined from the symptoms, which are the same as in the most marked cases.
XXXVII F.	F. S.	28	51	Neurasthenia, constipation	4.30	
XXXVIII M.	D. H.	53	81	Hyperacidity, constipation	4.30	
XXXIX M.	M. S.	25	50	Subacute gastritis	4.30	
XL F.	D. B.	20	40	Subacute gastritis, diarrhœa	4.30	
XLI M.	N. P.	29	61	General debility, constipation	4.30	Taught school and studied law.
XLII M.	S. B.	22	45	Neurasthenia	4.54	Repeated a month later. Same results.
XLIII M.	H. C. B.	45	64	Hyperacidity, constipation	4.35	
XLIV M.	B. M.	19	34	Dyspepsia, constipation	4.40	
XLV M.	H. R.	36	59	Hyperacidity	4.40	
XLVI M.	H. G.	23	81	Hyperacidity, subacute gastritis	4.45	
XLVII M.	S. Gr.	24	58	Stricture of pylorus	4.45	

As already stated elsewhere,[10] and for the reasons there given, I fixed upon 10 centigrams of albumen in 10 c.c. of filtrate, and five to five and one-half hours (at the farthest) to be taken as the normal standard for both quantity and time. Any deviation from this standard in either direction could then be readily recognized, and this deviation should then be, as it has been in all my cases, in harmony with the clinical history. That this method is correct for both quantity and time was borne out by the first series of cases reported,[11] and is fully confirmed by the tables here presented.

Table I shows a deviation downward. The 10 centigrams is digested more rapidly than usual, showing a greater digestive force in those particular gastric juices. This was proved in some of the cases (in those in which sufficient of the filtrate remained over for further examination) by a subsequent examination with 15 centigrams of the albumen to 10 c.c. of filtrate. In my first series of cases (coming under this same head), in all the instances in which a second examination with 15 centigrams was made the time required for the digestion of the greater quantity was the full normal time, five and one-half hours. In the present series we have three cases in which this examination

[10] Archiv. f. Verdauungskrkh., loc. cit.

[11] Loc. cit.

was made. In Case II the time required for the digestion of the 15 centigrams was five and one-quarter hours, the normal time. It indicated that in this case the pepsin in the gastric juice was augmented by 50 per cent. beyond the normal.

TABLE II.—DIGESTION IN NORMAL PERIOD.

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion.	Remarks.
I F.	L. H.	32	53	Constipation, hyperacidity	5.00	
II M.	M. C.	45	67	Hyperacidity, subacute gastritis, constipation	5.00	Suffers much with cramps.
III M.	M. J. C.	17	44	Constipation, dyspepsia	5.00	
IV F.	R. H.	24	48	Constipation; impaired respiratory activity	5.00	Nose very much obstructed.
V M.	S. B.	25	40	Stricture of œsophagus	5.00	
VI F.	L. C.	21	51	Constipation, colics	5.00	
VII M.	L. G.	26	60	Constipation	5.00	The constipation due to a wrong diet.
VIII F.	L. T.	19	40	Constipation	5.02	
IX M.	I. S.	84	98	Hyperacidity	5.03	
X M.	C. B.	35	65	Hyperacidity	5.05	
XI M.	J. H.	59	84	Hyperacidity, constipation	5.05	
XII M.	M. W.	26	64	Colics	5.09	
XIII F.	A. M. H.	28	56	Dyspepsia	5.10	
XIV M.	W. S.	25	49	Subacute gastritis, gastrosuccorrhœa	5.10	Subsequently obstruction of bile duct and jaundice.
XV M.	M. A.	46	62	Bright's disease	5.12	
XVI M.	C. N.	25	48	Hyperacidity, dyspepsia, constipation	5.12	
XVII M.	S. A. V.	37	53	Hyperacidity, subacute gastritis	5.13	
XVIII F.	N. D.	34	46	Constipation, tendency to hyperacidity	5.18	
XIX F.	Mrs. Z.	30	69	Neurasthenia	5.20	
XX F.	F. G.	30	45	Bad mastication, bad teeth, constipation	5.20	
XXI M.	A. B.	33	59	Tendency to hyperacidity, irritability of stomach, nervous	5.24	
XXII M.	S. R.	41	61	Hyperacidity	5.24	Nervous temperament.
XXIII M.	M. B.	34	59	Hyperacidity, constipation	5.30	
XXIV F.	F. R.	13	69	Constipation, dyspepsia	5.30	
XXV M.	F. B.	24	45	Rheumatism of stomach, subacute gastritis	5.30	Tendency to hyperacidity.
XXVI M.	M. G.	44	76	Hyperacidity	5.30	Much coffee.
XXVII F.	A. R.	19	39	Constipation, deficient HCl, nervousness	5.30	Leads an irregular life.
XXVIII M.	C. J. L.	055	25.5	Deficient HCl, irritability of intestinal tract	5.30	
XXIX M.	A. E.	30	52	Subacute gastritis	5.30	
XXX M.	S. J. P.	59	81	Hyperacidity, slight subacute gastritis	5.30	

In Case III the 15 centigrams was digested in three and one-half hours, showing that in this case the quantity of pepsin secreted was greater even than in Case II, possibly double the normal. I regretted

TABLE III.—DELAYED DIGESTION (more time than the normal period required).

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion.	Remarks.
I M.	M. P.	31	51	Constipation, tendency to hyperacidity	5.50	At the time of examination a question as to whether he had had a hepatitis shortly before or not.
II F.	M. M.	28	44	Constipation, secondary atony of stomach	6.00	
III F.	L. P.	22	51	Chronic diarrhœa	6.00	
IV M.	M. L. R.	26	71	Hyperacidity, constipation, angina pectoris	6.10	
V F.	N. B	03	37	Chronic gastric catarrh	6.10	Has lost very much flesh.
VI M.	M. S.	25	50	Hyperacidity, pseudo-angina pectoris	6.10	The symptoms pointed thereto (hyperacidity) and the dietary directions for this same brought rapid relief.
VII F.	L. K.	05	26	Defective gastric juice, costive	6.20	
VIII F.	F. D.	10	32	Defective gastric juice, constipation	6.20	
IX F.	P. W.	14	27	Slight catarrh of the stomach	6.20	
X M.	F. D.	13	45	Defective gastric juice, constipation	6.30	
XI M.	J. C. L.	10	29	Defective gastric juice, constipation	7.00	
XII M.	L. M.	45	65	Hyperacidity, constipation	7.03	
XIII F.	L. K.	05	28	General impairment of the digestive organs reflexly from ailments of the genital tract.	7.15	
XIV F.	B. K.	No reaction to Congo, none to ph. gluc. v.		Chronic gastric catarrh, tendency to epileptic seizures.	10.20	

very much that for lack of filtrate I could not carry the examination farther, to 17.5 centigrams, and if this was digested in less than the normal time, to 20 centigrams.

In Case XVII the greater part of the 15 centigrams was digested in three hours and fifty minutes; a small particle remained over and was still there and as large after four hours more in the thermostat. This demonstrated that the digestive capacity of these 10 c.c. of filtrate was beyond 10 centigrams, but not quite up to 15. Here also the lack of filtrate prevented a reëxamination with a less quantity, namely, 13 centigrams (the particle remaining over in the second examination I judged to be about 2 centigrams).

TABLE IV.—INSUFFICIENCY OF PEPSIN (in all these cases the heating was continued for 4 or 5 hours more after the time named, but no further digestion was effected).

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion and remarks.
I M.	F. R.	43	69	Hyperacidity, constipation	In 3 hours 39 minutes all digested except two or three minute particles.
II F.	B. B.	23	69	Stricture of pylorus (benign)	In 4 hours all digested except a number of minute particles.
III M.	J. H. C.	47	72	Hyperacidity, constipation	In 4 hours 10 minutes all digested except one fine scale. At the end of 7 hours scale still present.
IV M.	S. L. J.	48	71	Hyperacidity, costiveness	In 4 hours 30 minutes all digested except three minute particles.
V F.	L. S.	16	37	Deficient HCl, constipation	In 4 hours 40 minutes all digested except a few minute particles.
VI F.	Mrs. H.	14	51	Constipation, flatulence	In 5 hours all digested except two minute particles.
VII M.	B. S.	73	93	Hyperacidity	In 5 hours all digested except one particle.
VIII M.	M. C.	41	60	Hyperacidity, constipation	In 5 hours all digested except two minute particles.
IX F.	L. S.	08	27	Defective gastric juice, constipation	In 5 hours all digested except one minute particle.
X F.	L. J.	15	38	Gastralgia, constipation, defective gastric juice	In 5 hours all digested except a few minute particles. Five centigrams in 10 c.c., all digested in 3 hours 30 minutes.
XI M.	L. B.	32	58	Hyperacidity, constipation	In 5 hours 7 minutes all digested except two or three particles.
XII F.	V. B.	21	51	Cystic tumor of right ovary	In 5 hours 7 minutes all digested except four or five minute particles.
XIII M.	J. G	48	64	Hyperacidity	In 5 hours 25 minutes all digested except one faint scale.
XIV M.	H. G.	77	96	Hyperacidity, constipation	In 5 hours 40 minutes all digested except a few minute particles.
XV F.	A. C.	12	34	Defective gastric juice, constipation	In 6 hours all digested except three or four particles.
XVI F.	A. R.	12	42	Defective gastric juice, constipation	In 6 hours all digested except three or four minute particles
XVII F.	R. L.	27	50	Status epilepticus	In 6 hours 29 minutes all digested except two minute particles
XVIII F.	H. H.	06	21	Defective gastric juice, constipation	In 6 hours 30 minutes all digested except one small piece, thinned at edges only.
XIX M.	W. K.	55	70	Hyperacidity	In 6 hours 35 minutes all digested except two small particles.
XX M.	J. M.	53	72	Hyperacidity, nervous	In 6 hours 41 minutes all digested except two particles.
XXI M.	D. U.	41	65	Neurasthenia, hyperacidity	After 7 hours a scale of one segment (about one-third of it) remained undigested.
XXII F.	F. J.	10	58	Defective gastric juice, atony of stomach, hyperesthesia of intestinal mucous membrane	After 7 hours 13 minutes a large part of the one segment (though borders were very much thinned down) and a large scale of the other segment remaining over.
XXIII F.	Mrs. H.	13	28	Defective gastric juice, constipation, irregular eating, rich food	After 7 hours 15 minutes a very small particle and several minute ones over.
XXIV M.	L. D.	11	32	Defective gastric juice, slight catarrh of stomach	After 7 hours 30 minutes a number of minute particles still present.
XXV F.	J. C.	27	51	Chronic ovaritis, chronic appendicitis	After 7 hours 45 minutes a number of minute particles still present.
XXVI F.	J. S.	32	49	Atony of stomach	After 7 hours 48 minutes two small scales still present.
XXVII M.	M. R.	29	43	Constipation	After 8 hours several minute particles still present.
XXVIII M.	J. F	34	63	Hyperacidity, atony of stomach second degree, atony of bowels	After 8 hours one small particle and several minute ones still over. (Test repeated; same result.)

TABLE IV.—Continued.

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion and remarks.
XXIX F.	Miss L.	14	43	Defective gastric juice, atony of stomach	After 8 hours all digested except one thin scale.
XXX M.	S. J. P.	26	41	Gastralgia	After 8 hours several small particles still present.
XXXI F.	U. J. K.	02	..	Chronic gastric catarrh	After 8 hours 30 minutes 2.5 centigrams over.
XXXII M.	W. A. J.	58	77	Hyperacidity	After 8 hours 40 minutes two or three particles still present.
XXXIII M.	J. B.	43	78	Hyperacidity (bad teeth, bad mastication)	After 8 hours 43 minutes four small particles still there.
XXXIV M.	S. P.	21	47	Gastralgia	After 9 hours a number of minute particles still present.
XXXV M.	S. E.	17	49	Sarcoma of intestines	After 10 hours a scale of each segment left.
XXXVI F.	R. B.	16	26	Gastralgia, constipation	After 10 hours a minute particle still present.
XXXVII M.	J. W.	21	37	Beginning gastric catarrh	After 10 hours 30 minutes two or three minute particles still present.
XXXVIII	B. Th.	40	52	Hyperacidity, constipation	After 11 hours a minute scale still present.
XXXIX	A. K.	09	39	Defective gastric juice, constipation	After 11 hours two or three minute particles still present.
XL F.	J. R. W.	10	41	Defective gastric juice	After 12 hours three minute particles still over.
XLI F.	B. K.	10	28	Chronic gastric catarrh	After 13 hours four minute particles still present.

TABLE V.—VERY LITTLE DIGESTION OR NONE AT ALL.

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Time.
I F.	C. T.	No reaction to Congo	..	Chronic gastritis	10 centigrams in 10 c.c. filtrate + gtt. ii HCl c.p. After 9 hours unaffected.
II M.	E. N.	Reaction: blue litmus + Congo — resorcin —	..	Chronic gastro-intestinal catarrh	10 centigrams + 10 c.c., no effect. 10 centigrams + 10 c.c. + HCl gtt. ii c.p. After 11 hours digested down to one large scale and several small particles (in all about one-third, left.
III F.	S. W.	Reaction: blue litmus — Congo —	..	Cirrhosis ventriculi	10 centigrams + 10 c.c., no effect. 10 centigrams + 10 c.c. + HCl c.p. gtt. ii, no effect.
IV F.	S. R.	Reaction: Congo + filtered Congo — ph. gluc. v. —	18	Neurasthenia, constipation	10 centigrams + 10 c.c., no effect. 10 centigrams + 10 c.c. + HCl c.p. gtt. ii, digested in 6 hours.
V M.	N. S.	Reaction: blue litmus + Congo —	..	Nervous prostration	Without HCl, no effect; with HCl, digested all but one particle in 6 hours.
VI M.	A. St.	Reaction: blue litmus + Congo —	..	Cirrhosis ventriculi	10 centigrams with HCl gtt. ii at end of 5 hours untouched.
VII M.	H. W.	Reaction: blue litmus + Congo —	..	Cirrhosis ventriculi	10 centigrams with HCl at end of 10¾ hours untouched.
VIII M.	N. R.	Reaction: blue litmus + Congo —	..	Chronic gastric catarrh	10 centigrams without HCl after 10¾ hours no digestion at all. With HCl at end of 10¾ hours one large scale and a few particles over, altogether about 3 centigrams left over.
IX F.	S. F.	Reaction: blue litmus + Congo —	..	Chronic gastric catarrh	Without HCl, unaffected. With HCl at end of 8 hours, one segment slightly thinned at edges.
X M.	S. D.	0.1	2.2	Chronic gastric catarrh	Without HCl after 9 hours and 7 minutes, the four borders of each segment thinned down, translucent almost. With HCl, all digested in 9 hours and 7 minutes, except 2 small fine scales.
XI F.	J. V.	Filtered. No reaction to ph. gluc. v.	3.0	Constipation, neurasthenia	Without HCl, not affected. With HCl at end of 5 hours and 7 minutes all digested except two minute particles.

The points of special interest in this table are the indications (*a*) that in many cases of hyperacidity, perhaps in the greater number, there is also an increased secretion of pepsin,[12] and (*b*) that it is not by the excess of acid that the digestion is hastened. Thus, in Case XIV, with a degree of acidity, both free and combined, considerably lower than many of the others that follow, the digestive act was complete in much less time than was required, say, in Case XVII, with more than three times the quantity of free acid and more than double the quantity in the total acidity. The same can be seen in a number of other cases in this same table, and it is very clearly shown when comparisons are made with some of the cases of hyperacidity (Cases IX, XI, and XXVI) of Table II, and most strikingly so when the comparisons are made with some of the cases of hyperacidity of Table III (Cases IV and XII) and of Table IV (Cases IV, VII, and VIII).

There is yet something more that this table seems to disclose. It is the possibility that just as there may be an increased secretion of HCl without a corresponding increase in the secretion of the pepsin, so there may be an increased secretion of pepsin without any corresponding increase in the secretion of HCl. Cases XIV, XXII, XXVI, and XXXIII seem to point thereto.

In Table II the cases all conform to the standard in quantity and time. Here also we have a very interesting observation, the corollary of that noted above, namely, that there may be an increased secretion of HCl without a corresponding increase in the pepsin.[13] The patients with hyperacidity of Table I had, with all their complaints, a fair to good appetite, and could, when properly directed as to diet, eat again four hours after a hearty meal; those of Table II did not have that early hunger—in fact, some of them complained of poor appetites. In some of the cases of this table the greater part of the 10 centigrams was digested in four hours or a little over; still, as long as a speck remained, the act was not counted complete.

Tables III, IV, and V show us the deviations upward, there being a deficiency in the quantity of pepsin secreted. In Table III we note a deficiency; the time required for a completion of the process of digestion is more or less greater than the normal time. Nevertheless, the deficiency can be but slight, as there is still enough pepsin secreted to digest completely the test quantity. In Table IV the deficiency is greater, and part of the test quantity remains undigested. How much this is can be readily estimated either by simply judging it by the eye, which is rather unscientific, or by the more accurate method of removing it from the beaker to a filter and weighing it.

Still another fact of great interest is presented to us by these tables, namely, that we may have hyperacidity associated with a deficient

[12] This was first definitely demonstrated by my tables.

[13] Ibid.

secretion of pepsin, from the minimal deficiency of Table III, causing only a protraction of the act of digestion, *i. e.*, requiring a longer time for its completion, to the larger one of Table IV, entailing a marked diminution of the digestive capacity of that gastric juice.

In Table V we observe a total or almost total absence of pepsin and pepsinogen. Here we have a further demonstration of the fact that the HCl constituent alone does not effect digestion. In Cases I, III, VI, and VII pepsin and pepsinogen were altogether wanting, and the addition of sufficient HCl (c. p.) to give free acid 22 did not in any way change the result.

To summarize, it may be claimed for this method:

1. That it is simple and eminently practical.

2. That from the simplicity and exactness of its preliminaries it is well adapted for comparative study.

3. That it is directly demonstrative, in that the examiner has a direct ocular demonstration, as it were, of the digestive capacity of a given gastric juice, and can thus at once, without any long mathematical calculations, which not infrequently have only a fictitious basis, determine whether in the case under examination there is:

Hyperpepsinia: Requiring only three to four hours for digestion. Not necessarily in a pathological sense, only to indicate a secretion of pepsin greater than usual, which may, however, be normal for that case.

Normal pepsinia: Requiring only five to five and one-half hours (the normal period) for digestion.

Hypopepsinia: Requiring more than the normal time for digestion (deficiency in the pepsin secretion).

Apepsinia: No digestion at all (total absence of pepsin and pepsinogen).

TABLE VI.—DIGESTION IN LESS THAN THE NORMAL PERIOD. Less than 10 c.c.

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Quantity.	Time.
I M.	E. G.	45	85	Hyperacidity	4 centigrams to 4 c.c. filtrate	3.55
II F.	P. K.	11	22	Deficient HCl, constipation, nervousness	5 centigrams to 5 c.c. filtrate	3.55
III M.	J. L.	42	74	Hyperacidity, intestinal colic	5 centigrams to 5 c.c. filtrate	3.30
IV F.	B. C.	38	64	Hyperacidity, constipation	6 centigrams to 5.5 c.c. filtrate	3.30
V M.	J. St.	65	92	Hyperacidity, constipation	7 centigrams to 7 c.c. filtrate	3.35

But a mathematically still more closely defined result can be obtained with this method. In the course of this work it occurred to me, and the reasons therefor are self-evident, that possibly each of the 10 c.c. possessed the power of digesting individually 1 centi-

gram of the white of egg above described. A number of trials were made, and the results proved this to be so.

Additional confirmation was afforded by the results obtained in some other cases:

In Case X of Table IV the 10 c.c. were not able to digest the 10 centigrams entirely; a few minute particles remained over, and allowing the beakers to remain in the thermostat for four or five hours more than the normal time did not change the result; but 5 centigrams was digested by 10 c.c. in three and one-half hours, two hours less than the normal time. It was clearly apparent, therefore, that though the 10 c.c. was not quite equal to 10 centigrams (in other words 1 c.c. to 1 centigram), it was more than equal to 5 centigrams (or 1 c.c. to more than 0.5 centigram). If more filtrate had been left the examination could have been continued until the exact limit for quantity had been reached; judging by what was left over on the first trial, I should say that this would have been about 9 to 9.5 centigrams.

F. A. G., reaction to blue litmus +	6 centigrams in 6 c.c. after 9 hours 15 minutes, edges only slightly thinned.
reaction to Congo +	6 centigrams in 6 c.c. + HCl (c. p.) gtt. ii. Complete digestion in five hours.
Filtered, reaction to Congo —	

Here, when we have the pepsin activated by the addition of HCl, we get a striking verification of our proposition. It can, therefore, be said that the normal quantity of pepsin in the ordinary normal stomach is equivalent to the digestion by each cubic centimeter of the gastric juice of 1 centigram of white of egg, prepared as above described, in from five to five and one-half hours.

ADENOFIBROMA OF THE MALE BREAST.

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of Chicago.

(From the Pathologic-Anatomical Institute of the University of Strassburg, Prof. Chiari, Director.)

In March, 1907, there was sent to the laboratory a tumor mass which had been extirpated from the breast of a boy. This was put into my hands for study by Prof. Chiari. It had a fibro-epithelial structure corresponding to that of ordinary adenofibroma, such as may occur in the female breast. In attempting to ascertain the frequency of such growths in the male breast some difficulty was encountered, because of the uncertainty which still prevails in the literature

regarding the use of terms covering a group of affections of the male breast, all of which are fibro-epithelial in structure. Thus the terms adenofibroma, gynecomastia, adolescent tumor, mastitis, etc., are applied by different writers to the most variable affections. This seems to be due to a lack of data concerning the finer structure of the affections in question, a lack due in its turn to scarcity of pathological material, because all of these affections are uncommon and of such nature that they seldom demand surgical treatment.

A second case like the first one appeared in one of the surgical clinics a few weeks later, and Prof. Chiari then unearthed from the museum specimens representing five more cases of the same general group. With this rather unusual material at hand, it was thought worth while, in view of the afore-mentioned considerations, to record the findings and make them the basis for a comparative study of the members of the group of non-malignant fibro-epithelial tumors which affect the male breast.

Case I.—(The history was provided by the physician in charge of the case, Dr. Schaller, of Barr, in Alsatia.) Alphonse S., aged fifteen years, a gardener's apprentice, had always been well until one year ago. At this time he noticed a lump the size of a bean under the left nipple, and a few weeks later a similar enlargement under the right. Both were painful and grew quite rapidly until they reached the size of hazelnuts. After four to six weeks they disappeared spontaneously. Toward the end of January, 1907, about a year later, a new tumor appeared under the left nipple. This increased steadily in size, becoming more and more painful until the patient sought the advice of a doctor. He complained then of pain in the left breast which was increased when pressure was made over the areola. There was no history of traumatism.

Physical Examination. The patient is a very thin, lightly built, but healthy looking boy. All organs are normal except the breast. Under the left nipple is an elongated mass, 6 to 8 cm. long and 3 to 4 cm. broad, its long axis extending toward the axilla. Its consistence is firm, its surface somewhat knobbed; it can be moved freely on the underlying structures. The overlying skin is not adherent. The papilla itself, however, is fixed to the mass. There is marked tenderness on pressure. The right breast is normal. A potassium iodide salve was prescribed. February 26 (four weeks later): The breast is more painful. Two bean-sized glands are now palpable in the axilla of the affected side. A compression bandage applied. March 11: The tumor has increased somewhat in size and is still more painful. Extirpation of the mass together with the nipple under local anesthesia. Rapid healing. March 19: The patient reports that he is free from symptoms. Since this time there has been no recurrence.

Macroscopy. The tissue sent to the laboratory in alcohol and formaldehyde consists of a flat, dense, whitish disk (2.5 to 3.0 cm.

broad, 0.5 to 0.8 cm. thick. Its surface is smooth but somewhat knobbed, with all evidence to show that it had been sharply defined from the surrounding tissues. Its cut surface is white with small punctate or linear markings corresponding to gland ducts. These radiate downward and outward from the region of the papilla to the periphery of the disk.

Microscopic examination shows a predominance of fibrous stroma arranged in broad trabeculæ, which support in their interspaces glandular structures (Fig. 2). The latter consist of gland ducts cut in various directions, and their acinus-like terminals. The large ducts are nearly all dilated and show great irregularity of form due to projections of the walls into the capacious lumens. These projections are sometimes purely epithelial; more often they appear to be caused by invaginations of the surrounding stroma. Where both processes combine, true papillary structures are produced (as pictured in Fig. 4 from Case II). The lumens are generally empty. A few contain scattered epithelial cells, clear serum-like fluid, or detritus. Lining the lumens four to six irregular layers of epithelial cells may often be found, while at times, as above noted, solid epithelial projections occur, leaving no doubt that genuine proliferation of these cells has taken place. The ducts after three to four bifurcations terminate in groups of end-sacs analogous to those seen in the breast of a non-parous girl. They vary in number from two to eight in a group. The epithelium everywhere rests on a basement structure which is usually thickened. This membrane is not always "structureless" as Kölliker[1] observes for normal glands. Favorable sections show plainly that it is made up of very long, narrow cells laid in imbricated series and apparently analogous to the layer of myo-epithelium which occurs in sweat glands. Outside of the basement structure comes the fibrous stroma. This is moderately vascular and well supplied with nuclei which stain deeply. (For comparison of stroma with that of the normal gland of a fifteen-year-old male, see Figs. 1 and 2.) In the zones immediately adjacent to the gland structures one notices even greater cell richness and vascularity than elsewhere, also occasional plump cells with large oval nuclei like young connective tissue cells. (These are pictured from Case II, in Fig. 4, D.) The close proximity of these to the thickened basement structure and the occurence of large cells in the latter make it difficult to tell where stroma leaves off and basement structure begins. In hemalum- or hematoxylin-stained specimens the periglandular zones are rendered conspiciously blue owing to the presence there of a fluid which takes that color. Blue zones are especially marked about the terminals and finer ducts of the glands where the parenchymatous parts are invested in loose-meshed

[1] Beiträge zur Kenntnis der Brustdrüse. Verhandl. der physikalisch-medizinischen Gesellschaft in Würzburg, 1880, xiv.

areolar tissue mantels which fill the interspaces between the firm stroma and the epithelial structures. The acini and fine ducts appear then to be surrounded by myxomatous tissue. The fluid, however, gives no characteristic color reactions when stained for

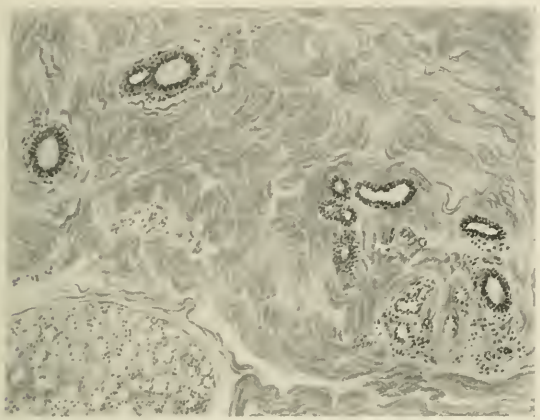

Fig. 1.—Normal breast from a fifteen-year-old boy dead of pneumonia. To show the nature of the normal stroma and epithelium. (Zeiss ocular 4, objective AA.)

Fig. 2.—Case I. Shows dense rich stroma and proliferated epithelium. (Zeiss ocular 4, objective AA.)

mucin or for fibrin. In adenofibroma of the female breast Ribbert refers to it as more in the nature of œdema. There are no collections of round cells. Elastic tissue is not demonstrable except in the bloodvessels. Fat tissue occurs peripherally in narrow islands

parallel to the fiber course. Centrally one sees single fat cells or small collections of them.

Diagnosis. The following affections require consideration:

I. Simple inflammation (mastitis).

II. Gynecomastia.

III. Neoplasm (adenofibroma).

IV. Adolescent tumor ("mastitis adolescentium," etc.)[2]

I. Mastitis. There are no evidences of simple acute inflammation. There is no marked vascular engorgement, no cellular nor fibrin-containing exudate. The mass as a whole, was sharply circumscribed. The only feature suggesting inflammation in any way was the enlarged axillary gland. The great difficulty that may arise in trying to differentiate chronic inflammation from tumor in the breast in general is recognized. It has been emphasized by Billroth,[3] Borst,[4] Ribbert,[5] Fabian[6] and others. In this case nothing points unequivocally toward inflammation in the ordinary sense.

II. Gynecomastia. This term, originated by Paul d'Egine, which has been loosely applied to nearly every disease to which the male breast is heir, refers literally to cases in which a male possesses a breast or breasts like those of a woman. Lack of pathological material for accurate study due to the rarity of the affection[7] and its mild nature, which only exceptionally demands surgical treatment, has been responsible for confusion as to its relationship to other diseases of the male breast and misconception as to the degree of real similarity which can exist between male and female breasts in general. Before proceeding with the differential diagnosis, I shall attempt to define clearly what the term gynecomastia properly embraces.

It was at one time supposed and still often stated that such glands can secrete milk.[8] The case of a man who suckled his own child continually recurs in the literature. The weight of scientific evidence is all against such occurrences. No case is on record in which the secretion of milk has been verified by chemical tests,[9] the reports of

[2] Peculiarities of the normal male breast capable of confusion with disease processes are thoroughly treated by Gruber, Luschka, Kölliker, and Langer.

[3] Die allgemeine chirurgische Pathologie und Therapie, 1893, 902.

[4] Die Lehre der Geschwülste. [5] Lehrbuch der speziellen Pathologie, 1902, 564.

[6] Die Bindegewebshyperplasie im Fibrom und im Fibroadenom der Mamma, Arch. f. klin. Chirurgie, lxv.

[7] According to Duplay and Reclus, only one case was found by Peuch in the course of the examination of 13,000 conscripts.

[8] Lydston, Jour. Amer. Med. Assoc., December 12, 1908, makes this statement again apropos of an article by Gellhorn concerning Abnormal Secretions from the Mammary Glands in Non-pregnant Women (same Journal, 1908, li, 1839).

[9] A simple chemical test is to demonstrate the presence of lactose. The poorest true milk contains a percentage of lactose not approached by any other body fluid. If the de-albuminized fluid reduces the ordinary copper solutions, but fails to show fermentation with yeast, lactose is probably present. Further proof of lactose would be afforded by precipitation of phenyl-lactosazon in the usual manner of osazon preparation, separation of the crystals, and determination of their melting point.

milk secretion having arisen through the not infrequent observation of whitish fluids expressible from the nipple which on exact examination have proved to be cystic, purulent, or serous. Further, no careful microscopic examination of the male breast has ever demonstrated acini of the type which occur in a breast which has functionated. A true resemblance between a mature female mamma and that of a male, therefore, does not exist. However, the finer structure of a virgin female breast differs little from that of the male. Let a male breast develop sufficiently in size, and it may truly resemble a nulliparous female breast.[10] Such an enlargement constitutes gynecomastia. It may occur as one manifestation of a general feminine tendency, and be accompanied by other anomalies, such as high voice, female distribution of hair, etc.; it may be associated with cryptorchism, hypospadias, hermaphroditism, and many other anomalies of the genitalia; it may follow removal of the testicles even late in life, or occur in the course of diseases, such as carcinoma, which destroy these organs; or, finally, it may be an apparently isolated anomaly occurring in otherwise healthy men of strong masculine habitus. It may be unilateral or bilateral.

The histology of such glands has been accurately studied in only a few cases (Volkman,[11] Stieda,[12] Israel,[13] Scheiber[14]). Apart from slight differences in their findings, which might have been due to differences in the age of the disease or to the presence or absence of slight superposed pathological processes, such as inflammations, these authors agree in assigning to such glands the structure of hypertrophic male breasts. They are anomalies of growth.

It must be remembered that a hypertrophic male breast is subject to disease. Owing to the nature of a man's pursuits it is more likely to suffer from traumatism than a female breast of the same size. Failure to discriminate between "healthy" and "diseased" gynecomastias has led to many doubtful statements regarding their characteristics, both pathological and clinical. A hard, dense breast is never pure gynecomastia, nor are simple gynecomastias "exquisitely tender" nor painful any more than normal female breasts. Histological reports bearing on such complicated cases have little value. (For examples, see Gaillet,[15] Reny,[16] Baillet,[17] Kauffmann.[18])

Limiting the term gynecomastia, then, to hypertrophies of the

[10] Provided, of course, the nullipara had never secreted true milk. For a discussion of milk secretion by virgins, see Gellhorn, loc. cit., footnote 1.

[11] In Schuchardt's Collection.

[12] Beitrag zur hist. Kenntnis der sog. Gynäkomastie, Beiträge zur klin. Chir. (Bruns), 1895, xiv, 179.

[13] Zwei Fälle von Hypertrophie der männlichen Brustdrüse, Inaugural Dissertation, Berlin, 1894.

[14] Einige angeborene Anomalien beobachtet im path. anat. Inst. zu Bukarest, No. 7 in Med. Jahrbücher von der k. k. Gesellschaft, Wien, 1875, p. 261.

[15] Soc. de biol., 1850.

[16] Thèse de Velpeau, Paris, 1880, p. 35.

[17] Bull. de la soc. anat., 1890, p. 532.

[18] Lehrbuch der spez. path. Anat., 1904, p. 955.

male breast, such as may occur as independent anomalies early in life, or as accompaniments of other signs of female habitus, such as feminine voice, female hair distribution, etc., or in association with cryptorchism, hypospadias, hermaphroditism, or other anomaly of the genital tract, or such hypertrophy as may occur at any time as a result of castration or disease which destroys the testicles, we may say that the specimen under consideration is not gynecomastia. It differs in that it was painful and tender from the outset. The mass was sharply circumscribed and dense. Under the microscope the stroma is seen to be rich and active, the epithelium to be in a marked state of proliferation.

These observations were controlled by comparison with a genuine case of gynecomastia which Prof. Chiari kindly lent from his private collection, and also by comparison with a second case of gynecomastia herein described (Case VII). It may be stated here that these cases confirm the findings of Stieda, Israel, and others.

III. Adenofibroma. The density, circumscription, and histological structure tally closely with those of an ordinary adenofibroma, such as may occur in the breast of a young woman. The symptoms and course also offer nothing incompatible with such. The only difficulty is encountered when one attempts to make such a diagnosis fit in with the clinical facts which have been published concerning that affection known as "mastitis adolescentium," which also closely resembles this case, but which is supposedly a self-limited affection, it being alleged that most cases recover spontaneously. Further discussion will follow a short presentation of the facts concerning this latter disease.

IV. Adolescent Tumor. (Mastitis adolescentium, mastitis pubescentium virilis, Albers.[19] Areolar and mammillary tumor, Cooper.[20]) Hochenegg[21] says: "Mastitis adolescentium constitutes an inflammation of problematical etiology which affects the mammary glands of young men." The observed characteristics of the disease will be evident from the following citations taken mainly from Schuchardt's[22] review of male breast affections.

C. Langer[23] states: "At puberty there very often occurs a dull, easily borne pain in the breast, during which time the organ enlarges somewhat, the nipple projects, and the areola becomes deeply pigmented or even reddened. After two to three weeks all symptoms disappear, but the nipple may remain prominent from that time forth." This is common knowledge. Severer cases may be marked by exaggeration of the pain and prolongation of the course. Albers

[19] Mastitis pubescentium virilis, Med. Korrespondenzbl. Rhein. und Westphäl-Aerzte 1843, No. 13.

[20] Lectures on Surgery, 1836.

[21] Lehrbuch der spez. Chir., 1907, i, 869.

[22] Ueber die Vergrösserungen der männlichen Brüste, Langenbeck's Archiv, 1885, xxxi, 159.

[23] Ueber den Bau und die Entwickelung der Milchdrüse bei beidern Geschlechtern. Denkschrift der Kais. Akad. der Wissens. (math. wissens. Klasse), 1852, iii.

found the gland enlarged in the form of "a flat hard callus" lying behind the nipple and tender on pressure.

A case of Lauenstein's[24] reported by Schuchardt ran as follows: The patient, aged twenty-six years, entered the hospital, October 13, 1884, on account of a tumor in the left breast which had developed within five months. No history of syphilis or traumatism. The tumor had never been painful until about the time he sought medical advice. The region of the left breast bulged somewhat. The centre of the left mamma was occupied by a flat tumor the size of a 5-mark piece which seemed not to be fixed either to the skin or to the pectoralis. No pain on pressure. . . . The patient had in each axilla an enlarged freely movable lymph gland, and on the right side a swollen cubital gland. Extirpation October 14. The tumor included the centre of the gland; it was the size of a 5-mark piece and 1 cm. thick. In substance it was dense like a fibroma and sharply defined from its surroundings. On cut surface it looked grayish white. . . . Examination of sections made after hardening showed excessive amounts of connective tissue ground substance, in which gland tubules were here and there introduced and in which in addition there occurred numerous smooth-walled irregular cavities which gave the whole section the appearance of being perforated like Swiss cheese. The vascular development in the tumor was not very rich. The diagnosis *fibro-adenoma cysticum*, herewith made, was confirmed by Prosector Dr. Fränkel, of Hamburg. The patient had no further trouble. Schuchardt looks on this case as one of adolescent mastitis.

Stümcke states that the disease lasts until adult life is established; that its course will be shorter the less time there remains before that event. He never saw a case that did not get well spontaneously.[25]

Risel[26] also reports a case like Lauenstein's, in which the tumor was much larger but nevertheless disk-shaped, attached to the nipple, otherwise free, sharply circumscribed, dense, etc. Risel says he too has seen other cases of the disease, but never had to operate except in this instance.

Such cases as Lauenstein's and Risel's are undoubtedly instances of the same disease as Case I. They were also looked upon by the writers as identical, for a time at least, with adolescent mastitis. If such cases as these were capable of spontaneous healing like the rest, it would be difficult to think of them as tumors, in spite of their structural resemblance to adenofibroma. But it will be noticed that those cases which were operated upon and showed tumor structure had already demonstrated clinically that they were dif-

[24] In Schuchardt's Collection, loc. cit.

[25] It has also been said that onanists and those who have exercised the sexual function are seldom affected.

[26] In Schuchardt's Collection, loc. cit.

ferent from the ordinary adolescent mastitis, and were operated upon for this very reason, that is, their lack of self-healing tendencies.

Microscopic examinations have never been made in such cases as get well by themselves. If made, it is probable that the picture would be found to differ from that of such cases as Case I. This unknown picture would be that of adolescent mastitis.

Briefly summarized, Case I is an instance of adenofibroma in the male breast. What "adolescent mastitis" is, we do not know. Case I throws no new light on it, unless it be taken to show that "adolescent mastitis" may occasionally give rise to true adenofibroma.

A review of available literature made for the purpose of learning how frequent these cases of adenofibroma are, showed that while malignant tumors occur in the male breast about one-fiftieth as often as in the female breast, and are hence not extremely rare, non-malignant neoplasms (in which we do not include adolescent tumor) occur much less frequently. Pure undoubted adenoma has been reported three times only (Römer[27]), and one of these reports was suggestive of carcinoma. As stated at the beginning of the paper, reports of "adenofibroma" have included many sorts of affections, so that it is difficult to judge of their frequency. Some half-dozen cases in the literature had characteristics which made it evident they were identical with the one in hand. But probably they are commoner than the literature indicates.

Case II.—Museum specimen 3923, dated June 10, 1902. Histological Protocol 1756, Dr. Boeckel.

Clinical History. Edward S., aged ten years, was first examined by Dr. B., June 6, 1902. The mother said that a tumor began in the left mamma eight months before, and that recently it had begun to grow larger (no mention is made of pain). Examination showed a tumor in the upper part of the left mamma the size of a small apple. The nipple protruded. Axillary glands not enlarged. Clinical diagnosis adenoma. Extirpation June 10. Complete healing of wound. No recurrence after four years.

The tissue consists of a roughly oval disk with one surface a little concave, the other convex. Dimensions, 8.5 x 5 x 2 cm. The surfaces are everywhere smooth, in places slightly knobbed. All signs point to a sharp circumscription. The cut surface is marked by connective tissue trabeculæ, which subdivide the whole into small irregular areas in which the tissue is darker and more opaque than the rest, corresponding to ducts and terminals.

Microscopic examination shows trabeculæ of relatively dense connective tissue provided with numerous, fairly plump nuclei, and moderate numbers of bloodvessels (Figs. 3 and 4). Fat tissue occurs in the stroma as long islands running parallel to the course of the fibers and as single fat cells or small clusters of the same. The

[27] Inaugural Dissertation, München, 1880.

glandular parts show the same characteristics as those seen in Case I, except that in this case the acini are better developed, groups of eight, ten, and twelve being not uncommon. These are

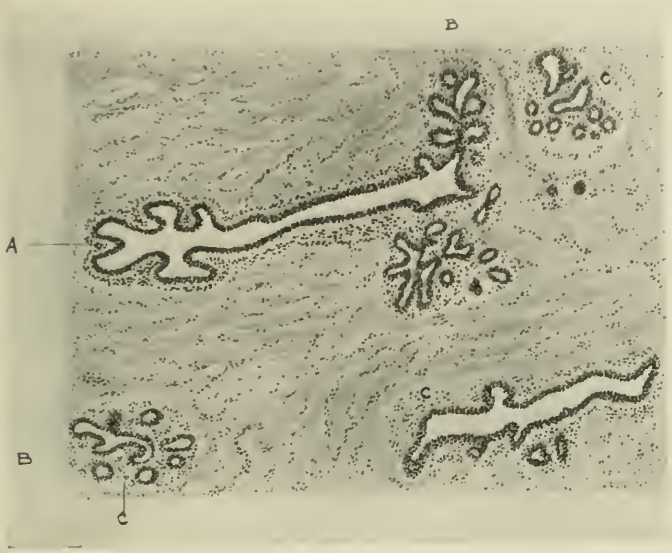

Fig. 3.—Case II. *A*, zone of active proliferation of stroma cells; *B*, group of characteristic end-sacs—virgin type; *C*, mantles of loose tissue containing blue-staining fluid. (Zeiss ocular 2, objective AA.)

Fig. 4.—Case II. Shows true epithelial proliferation *A*, and papillary invaginations in a duct *B*. *C* shows the character of the blue-staining periglandular mantle. *D*, young connective cells. *E*, basement structure. (Zeiss ocular 4, objective DD.)

larger than those seen in Case I, though not like those of a functionating female breast. The glands are surrounded by the same blue-stained mesh containing numerous young connective-tissue

cells. Elastic fibers are absent. The bloodvessels are full of blood, and many show in their vicinity a slight sprinkling of leukocytes. The gland ducts contain gray detritus and drops of coagulated fluid, which stain deeply with eosin.

This case differs in no essential from the foregoing except for the larger size of the tumor and for the slight round-cell infiltration. The high-grade development of rudimentary acini is no more than has been observed in normal breasts. Diagnosis, Adenofibroma.

Case III.—Museum specimen 3877, dated June 13, 1899. Label: Adenofibroma mammæ dextræ.

Clinical History. A tumor developed in the breast of a soldier, aged nineteen years, during the course of a year. The patient was examined by Dr. Eichel, who found in place of the right mamma a disk of firm consistence measuring 7 x 7 x 2 cm. It was not well defined from the surrounding tissues. The nipple was normal, the areola pigmented deeply. The axillary glands were slightly enlarged. The tumor was extirpated, the axilla opened, and the enlarged lymph glands removed.

The tissue of the tumor is dense and white. It sends prolongations into the fat tissue, which in turn projects into the tumor. Examination of the cut surface, which is of a translucent gray-white color, reveals irregularly distributed linear, y-, or glove-shaped flecks of yellowish color corresponding to gland structures.

Microscopically fibrous tissue predominates to a greater extent than in either of the foregoing cases. It is of uniform consistence throughout and sparingly beset with thin, narrow nuclei. Bloodvessels are small and few in number. The glandular parts consist mainly of long dilated ducts branched dichotomously. Occasionally they show lateral twigs. End-sacs occur in groups of only two to four. The papillary structures described in Cases I and II are less marked. Fat is scarce, elastic tissue absent. Scatterings of leukocytes occur in several places. The lymph glands show areas of hyperplasia.

In this case the patient's age is the same as in Cases I and II. While there is no specific history of traumatism, it is not at all improbable that such had occurred, since a tightly clothed soldier in the course of drill is likely to rub or bruise the chest. The sharp circumscription of Cases I and II is lacking, nor is the stroma so active nor the epithelial changes so marked. There is, moreover, a diffuse leukocytic infiltration, while the axillary glands examined showed hyperplasic areas.

Another case, with no clinical history available, but, so far as ascertainable, like the last, may be introduced here and discussed along with it.

Case IV.—Museum specimen 3925. Male, aged eighteen years. Dated, June 11, 1905: labelled, Adenofibroma mammæ. The specimen consists of an ellipse of skin, including a prominent nipple with

a pigmented areola. Beneath the nipple is an ill-defined, tough, whitish mass, which shows under the microscope the same structures as Case III.

Such cases as these (III and IV) differ less markedly from inflammatory processes than do such cases as I and II. Morphologically they stand on the dividing line between inflammation and tumor. They are entirely analogous to the more diffuse adenofibroma-like growths which occur at various times of life in the female breast. The completeness of the series of forms intermediate between inflammation and tumor in the female breast has even been made the basis for the view that direct metamorphosis of fixed tissue cells into tumor cells may occur (Fabian). This view is only quoted to illustrate how many the intermediate forms are, and to point out that in the male breast also we should expect to find a series of tumor-like growths, some showing more, some less, resemblance to pure inflammation, on the one hand, or to adenofibroma, on the other. Such cases as the foregoing are taken as evidence that such a series does exist in males as in the other sex. They throw no new light on the real nature of such.

The four cases so far described occurred in youth, and seem to fall into two groups, two sharply defined tumors, two ill-defined, the former associated with no history of traumatism, the latter perhaps traumatic. The two following cases are of frankly post-traumatic enlargements in adult breasts and seem to resemble Cases III and IV rather than I and II.

Case V.—Museum preparation 3926. Male, aged fifty-six years.

Clinical History. Nine months ago patient fell, striking against the left breast, after which a tumor developed very slowly. Lately it has shown a more rapid growth. October 11, 1898, extirpation by Dr. Lange, of Strassburg, who found the mamma represented by a tumor the size of a hen's egg, not sharply defined from the surrounding tissues. The nipple was prominent and adherent.

The tissue is a dense yellowish mass 4.5 x 2 x 2 cm., which blends intimately with the fat tissue surrounding it. The cut surface shows trabecular markings, the interspaces between which are of darker color and irregular shape.

Microscopic Examination. Dense connective tissue stroma predominates. It is fairly vascular and beset with numerous elongated but not extremely thin nuclei. Gland elements occur as ducts and terminals, as described in Cases I to IV, varying according to the region from which sections come. They are enveloped in broad mantels of loose meshed tissue composed of narrow spindle cells with fibrous extremities, in the interspaces between which, especially near the basement membrane, young connective tissue cells occur. The whole mesh also contains the blue-staining substance described in Case I. Fat tissue is seen in the dense tissues

in moderate amounts, especially near the periphery, where it occurs in large islands. Toward the centre one sees only single fat cells or small groups of them. The glandular parts correspond closely to those of Cases I, II, and IV. Elastic tissue is lacking. A few scatterings of leukocytes occur.

Case VI.—Johann S., aged forty-two years, has noticed a tumor in the left breast since a fall three months ago, in which the breast was bruised. It has grown a little ever since. The patient is otherwise normal, of strong build, and decided masculine habitus. Examination reveals a palm-sized tumor, which is freely movable, on the deep tissues and under the skin. The nipple and areola look large, but are not adherent. May 7, 1907, extirpation. Healing.

The tissue sent to the laboratory consists of an ellipse of skin containing nipple and areola, both of which are prominent but not fixed to the mass which lies below. Under the skin is a piece of tissue 8 cm. wide and 5 cm. thick, in the centre of which is an indurated mass corresponding to the mammary gland. The latter blends all around with the fat tissue. It is firm and consists mainly of fibrous tissue enclosing elongated, semitransparent, colloid-like areas. In the centres of these areas the gland ducts are visible as clefts, or punctate orifices which can be sounded with a bristle. Microscopically, the findings are essentially the same as in Cases III and IV.

In their lack of circumscription these two cases (V and VI) resemble the two preceding cases (III and IV). The only difference between them and Cases III and IV is that the latter occurred earlier in life, while their histories fail to mention the occurrence or non-occurrence of traumatism as an exciting cause, while Cases V and VI arose in advanced life as a result of definite injury. Probably Cases III, IV, V, and VI all belong to the same category, that is, the post-traumatic enlargement just discussed.

These cases differ from adenofibromas like Cases I and II in that the latter began spontaneously as affections which, seen early, would have been called "adolescent mastitis," and developed into sharply defined disk-like tumors.

Consideration of these cases in conjunction with case reports in the literature suggests the view that such sharply defined adenofibromas as Cases I and II always originate in this same way, whereas the ill-defined less certainly newgrowth-like enlargements are frequently a result of traumatism. The sharply defined growths are closely analogous to the painful adenofibromas which occur in young female breasts, which it may be incidentally noted, also frequently begin at about the time of puberty as a result of an affection resembling in all ways "adolescent mastitis." Jopson, Speese, and White have recently noted the relatively great frequency of fibro-epithelial enlargements of the breasts of children at the

time of puberty as against other periods in both sexes. The ill-defined growths are analogous in every way to non-malignant indurations which occur in adult, less often in young, female breasts. Both can be looked upon, however, as adenofibromas because of their characteristic structure and progressive growth.

The last case, an example of gynecomastia, is here recorded because reports are still needed definitely to establish the nature of this rare affection. Stieda's two cases in 1895, with six more collected by him from the literature, form the bulk of what is known concerning the histology of the genuine affection. It also offers favorable opportunity for direct comparison of this process with the other cases of the adenofibroma-like group herein described.

Case VII.—Museum specimen 3359, from the Pathologic-Anatomical Institute. Labelled Hyperplasia mammæ.

Autopsy protocol, November 17, 1894; male, aged fifty-nine years. Cause of death, cirrhosis of liver; also, hypertrichosis and enlargement of both mammæ. The specimen consists of an ellipse of skin including the areola 2.5 to 3 cm. in diameter, and a prominent nipple 3 mm. thick. Around the edge of the areola is a growth of long, coarse hairs. Under the skin is a mass of fat tissue including a gland 6 x 7 x 1.5 cm. not more sharply defined from the surroundings than a normal gland. Its consistence is much less firm than that of Cases I to VI. More cannot be said, owing to the preserving fluid.

Microscopic sections show even to the naked eye a light loose stroma especially marked in the deeper parts of the gland where the ducts bifurcate, so that the peripheral branches of the glandular elements seem to be supported in a light mesh. Under the microscope the relatively dense stroma directly under the nipple is found to consist of wavy bundles of coarse fibers with scanty numbers of long thin nuclei and few bloodvessels. Deeper down such stroma occurs only in trabecular strands, between which, and hence enclosing the gland-parts proper, are areas of very loose fibrous tissue provided with very few nuclei. The whole is more lightly built than the normal breast of a fifteen-year-old boy, pictured in Fig. 1. The ducts show one to four layers of low columnar or polygonal epithelial cells resting on the basement membrane. There are none of the epithelial prominences described in Cases I to VI, nor any invaginations of the duct walls into the lumens comparable to papillæ. Fat is seen in patches lying parallel to the course of the fiber bundles, much more plentiful peripherally than in the centre of the gland. No leukocytes are found.

This specimen differs from the normal only as regards gross measurements, and the texture of its stroma. The latter may depend on the different ages of the subjects compared. The differences between this case and Cases I to VI are essentially the same as the differences between the normal gland and Cases I to VI. These

findings are virtually in accord with those of Israel, Scheiber, Stieda, and Volkmann. They confirm the view that gynecomastia is simply hypertrophy.

Summary. Seven cases of non-malignant enlargement of the male breast are reported, affording opportunity for a comparative study of the members of a rather rare and little studied group of affections. Six of the cases are adenofibromas as here interpreted, but they admit of division into two groups:

1. Sharply circumscribed, firm, dense tumors, which arise without traumatism in the breasts of young men, run a painful clinical course, and show under the microscope little or no evidence of inflammation, but all the characteristics of ordinary adenofibromas, such as may occur in the breasts of young women, and to which they are here looked upon as analogous. Cases I and II are of this type.

2. Diffuse or ill-defined growths which may occur at any age as a result of traumatism. Microscopically, these enlargements have virtually the same structure as those of Group I. Signs of inflammation are more in evidence, however, so that their differentiation from chronic inflammation may not be easy (Cases III to VI inclusive). They are here called adenofibromas, because of their progressive growth and microscopic structure. This group corresponds to the "traumatic indurations" of some writers.

Cases like I and II are rare. Cases of the type of V and VI are probably more common than is generally supposed. The early stages of cases such as I and II closely correspond to the disease of problematic nature and unrecorded histology known as "adolescent mastitis." Case I in particular with its small disk-like tumors showed all of the characteristics which have been assigned to this affection.

What the relationship is between adolescent mastitis, so-called, and adenofibromas cannot be settled until histological examinations of the tissue in the former disease have been made. Certainly a close relationship exists between adolescent mastitis and tumors such as are included in Group I. Probably adolescent mastitis is a separate affection, as a sequel to which adenofibroma may develop in exceptional instances. Nearly all cases of sharply circumscribed adenofibromas of the male breast heretofore described have developed in youth in the course of what appeared to be "adolescent mastitis." Further study of this affection is needed. Case VII is an example of gynecomastia. Its histological structure is simply that of an hypertrophy which confirms the few exsisting reports on the histology of genuine unmixed examples of this rare condition. The term gynecomastia might well be dropped in favor of hypertrophy.

OCULOMOTOR PARALYSIS ACCOMPANIED BY FACIAL PALSY, NEUROPARALYTIC KERATITIS, AND HEMIPLEGIA.[1]

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Through the kindness of Dr. Schwenk I am allowed to report the following case, interesting on account of the multiplicity of important symptoms, the vagaries in the modes of their production, and the marked regain of power by the patient.

On August 24, 1905, a tall negro woman came to the hospital complaining of the sudden closure of her left eye. The eye had been inflamed for six weeks past. Two weeks before coming to the hospital the sight was totally obscured by the falling of the lid over it. The woman was aged thirty years, a housekeeper, and had been married for eight years. She had been pregnant only once, six years ago. Two days before the end of term she fell down stairs and gave birth to a dead child. She denied having specific disease. She had been in good health until March, 1905, when she was seized with violent pains in her head, and was treated for rheumatism. In July her left eye became inflamed, and she had applied for treatment at another hospital; this inflammation was not very painful.

On examination the left brow and cheek were seen to be discolored, and near the outer angle of the orbit there was a large circular maculation, which had resulted from long blistering. The eye was completely covered by the upper lid. There was facial palsy on the left side and the mouth was drawn toward the right. When the lid was raised the globe was seen to be rotated outward. It was deeply injected, and the epithelium of the cornea was macerated and steamy over nearly the entire surface. The summit was œdematous and infiltrated; and it stained deeply when fluorescin solution was instilled. No view of the fundus could be obtained, nor could the iris be seen. Atropine suspended in oil was prescribed, and the patient was instructed to take a saturated solution of potassium iodide, beginning with fifteen drops and rapidly increasing the dose.

The periphery of the cornea was less œdematous in two days, and soon became sparkling. The ulcerous area rapidly became sharply outlined. In a few days, on forced effort, there was slight ability to raise the upper lid, but no effort could overcome the paresis of the internal rectus, though there was a faint reaction of the superior oblique muscle. By September 21 the ball remained divergent and fixed at the outer extremity. Facial paralysis was complete on the left side, and in the distribution over the superior and inferior

[1] Read at a meeting of the Wills Hospital Ophthalmic Society, Philadelphia, March 2, 1909.

maxillary branches of the fifth nerve there was anesthesia. The cornea continued hazy and anesthetic. About this time the patient's left arm and leg became numb and powerless. She was seized suddenly in the night in the midst of a severe headache; in the morning she could not lift her arm nor raise her leg in walking.

On September 28 the patient was sent to Dr. Wm. G. Spiller, at the Polyclinic hospital, for an examination of her general state. His letter, dated October 12, reported that the patient had cerebral syphilis. On October 24 the solution of atropine was discontinued, and by November 11 the "mixed solution" which had been ordered three weeks before was withdrawn, and mercurial ointment substituted. By November 28 the patient showed signs of ptyalism, and all medicines were withheld. Toward the end of December, when efforts were forcibly made the globe could be brought to the vertical line, but not within the meridian, and when the exertions were made it was noticed that the palpebral fissure widened so that the globe was entirely uncovered by the lid. At this time marked increase in the general muscular power began; the facial palsy was reduced, and the leg and arm regained much of their lost functions.

On January 5, 1906, the cornea again became denuded. To allay the irritation the lids were sealed with plaster and a firm bandage was kept on for two days; on the removal of this the exposed area was seen to be closed, but the surface was shaggy, as though it were studded with epithelial shreds like the remains of ruptured herpes. These shreds were examined by Dr. Goldberg and found to consist of epithelial cells only. Toward the end of the month the epithelium became restored. From this time on the return of power was marked. The ptosis disappeared almost entirely. The eye could be rotated to the primary position, but not within the vertical nor above or below the horizontal meridian. The woman could walk with greater comfort. A certain childishness manifested in the past gave place to a decidely acute intelligence. Before the first of February, 1906, the patient ceased her visits, and did not return again until January, 1909, in response to a letter from me. By this time she had regained all her lost weight and much more besides, for she weighed 215 pounds. In the past three years she had been able to keep house and to work as a laundress. She has had a constant dull ache over her left brow, and, when overtired, she has attacks of headache, and the arm and leg feel like "pins and needles." She is very much annoyed by diplopia when she attempts to look up or down, yet even in the primary position there are two images, the left being the false one and the higher. The sight is good, but with the left eye objects appear far away and small. There is now partial ptosis when at rest, but by wrinkling her forehead she can draw the lid up to the limbus. There is paralysis of the superior and inferior recti, the inferior oblique, and the superior oblique muscles; the ocular excursions are limited to the extent of free adduc-

tion and abduction, while supraduction is abolished and infraduction is only faintly perceptible. The diplopia is upward. There is no accommodation power, for while the woman can read types 0.50 D. with the right eye, she has great difficulty in seeing type 2 D. with her left. The vision, however, in each eye equals 5/10. The pupil of the left eye is not circular and is apparently fixed. The fundus is healthy. The cornea is perfectly smooth, and, even with the stereoscopic loupe, nothing is found marring the clearness of the membrane. It is, however, anesthetic, and so is the conjunctiva. There is a free flow of tears. The adjustment of spherical lenses + 1.25 D. with which she sees 5/5, has relieved the woman in the past month of this annoying epiphora. The facial movements have been regained; she can wrinkle her brows and protrude her tongue straight. The face is anesthetic to pin pricks, including the nose, tongue, all of the upper part of the head except that portion comprised in a line from the lobe of the ear and ramus of the jaw forward to about three-fourths of an inch from the meridian line of the chin up beyond the angle of the mouth, thence obliquely to the ear.

In order to know the patient's general condition, I sent her to Dr. T. H. Weisenberg for an examination. He fortunately remembers having seen the woman in Dr. Spiller's clinic at the Polyclinic hospital four years ago. As Dr. Weisenberg points out, the separate occurrence of left hemiplegic with left oculomotor palsy is most unusual. It is probable that the hemiplegia was caused by vascular disturbance in the right side of the brain, while the oculomotor palsy arose through a disturbance at the base on the left side, in advance of the nerve fibers supplying the extremities. The hemiplegia has almost entirely disappeared; it is rather strange, therefore, that the oculomotor disturbance remains. This palsy has been most extensive, affecting both the intrinsic and the extrinsic mechanisms. The widening of the palpebral fissure during the middle period of the ptosis was rather startling in appearance, and was the first instance of that phenomenon in my experinece. The facial palsy has all but disappeared, the anesthesia being the most annoying subjective symptom in this connection. It is most fortunate that the early keratitis did not extend to the true corneal tissue.

INSANITY, RESPONSIBILITY, AND PUNISHMENT FOR CRIME.

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To most people it seems very startling when they are told that experts in mental and nervous diseases are agreed that no absolute definition for insanity can be given. Insanity, which means etymologically ill health, has now come to be limited exclusively to mental ill health. It is very evident that this is a term of extremely wide comprehension. It shades off on all sides into normal conditions so elusively that it is impossible properly to set limits to it. The expert may not quite agree that "half the world is off and the other half not quite on," nor with that expression of the French that "we are all a little cracked, only some of us conceal the crack better than others;" but he does not like to be pinned down to a formula of words according to which the sanity or insanity of individuals may be judged with absolute assurance. As a matter of fact, no definition will stand the light of criticism and discussion and application to all the varying cases that are seen.

The confession that there is no satisfactory definition for sanity or insanity even by acknowledged authorities in mental diseases, loses most of its original startlingness as soon as we are brought face to face with the fact that there is also no definition for health or illness of body. After all, the two problems are very nearly alike. The one is mental and the other physical ill or well being. Sanity of mind cannot be defined, but neither can sanity of body. Here once more there are so many elements that enter into the problem, differing in each individual, that they cannot all be combined in a single formula. This is, after all, nature's way in practically everything. Plants and animals differ from one another ordinarily quite as strikingly as sane and insane individuals, yet no absolute definition by which plants may be distinguished from animals in that borderland where the two kingdoms of living things touch, can be framed. Nature evidently did not intend that there should be such precise delimitations in nature. Things grade so insensibly into one another that it is impossible to make fine accurate distinctions. It is the individual that must be studied and not the class, and the various expressions of nature animate and inanimate have an individuality all their own.

It is evident then when we talk of insanity as a defence for crime we are turning to an excuse which may be extremely difficult to control and keep from being abused. It is often said that an insane person is not a criminal and that he must not be held to criminal responsibility for his actions. While the medical expert will not and

cannot give a definition of insanity, and does not hope ever to be able to give one, he does not hesitate to tell us who are or are not insane. It is evident, however, that in the haziness of the subject it is extremely easy for the expert, even in good faith, to be led when his interest and that of his client, for it is as client rather than as patient that the medical expert's employer must be considered, sway his judgment, that he may be easily led into expressions which can only serve to be a source of confusion to the court and above all to the jury, and not at all a source of such expert knowledge as would prove to be of service. Here is where the difficulty lies in the great abuse of the plea of insanity, which has been allowed to creep into our criminal procedures in recent years.

Between this and the readiness with which lawyers find legal quibbles and technicalities to delay and defeat justice, these two abuses have brought about a very serious state of affairs. The number of murderers who are punished in this country is very small indeed. A prominent divine in New York pointed out not long since that it was much safer to be a murderer in this country than to be an employee of a railroad. When his statement was questioned, he showed by the statistics of recent years that to the whole number of brakemen employed in this country, more were killed every year than of murderers were punished to the number of murders. One of the things that most impressed Ambassador White when he returned to this country was the great number of murders committed and the few murderers that are punished. He called attention to the fact, which is not pleasant for American ears to hear, that human life is cheaper in this country than in any other portion of the civilized world, with the possible exception of Vendetta-ridden Corsica or Sicily. It is evident that without realizing it we have been slipping back into a barbarism none the less to be deplored because it happens to be associated with an over-refinement, which it must not be forgotten has characterized the history of all nations which, having reached an acme of progress, have begun to decay.

Of course, it is realized that many of the murders committed in this country are due to an immigrant population and to the fact that these peoples have not as yet come under the influence of the civilizing process at work in this country. It must not be forgotten, however, that many of our most prominent murders are committed by native-born citizens of good education and with all the advantages that our civilization is supposed to give. Most of these murderers, however, escape on the plea of insanity. We are in the midst of a reaction in this matter. Time was when the rule was an eye for an eye and a tooth for a tooth, and for a human life a human life, as if somehow a crime must be compensated for. We do not care to make the punishments fit the crime now, but the criminal, and we have gone so far to the other extreme that whenever there is anything suspicious in a man's previous history we are prone to think that this may

have impaired his responsibility to such a degree that he does not deserve punishment. It has been easy, therefore, for the medical experts to lead us into a state of mind where criminals escape punishment very readily on the plea of insanity—and then secure their release from insane asylums because they are sane.

There is a tendency to blame physicians for this state of affairs, and to say that is it due to a commercializing of professional dignity. Perhaps there is some truth in this, but it would be entirely unjust to lay the blame at the door of the medical profession alone. The physician is bound by a professional duty to protect the public and secure the ends of justice. There is no doubt of this, but he is not bound as formally to secure the enforcement of law and the ends of justice as is the lawyer. It is clear that at the present moment somehow the legal profession has been diverted from its true purpose to a very large extent. This is so true that ordinarily it is quite startling for the community to be reminded as it was so forcibly by Governor Hughes in his address to the graduates of Fordham University Law School, in the spring of 1908, that the lawyer on his admission to the bar becomes a sworn officer of the court for the securing of justice. He is bound by oath to strive only for what is just for his client, absolutely nothing more. In all matters in which life and death are not in question he is bound by formal oath to see that justice is done, no matter what may be the consequence to his client.

In spite of this, lawyers find ways and means to defeat justice and to delay it, so that President Taft has declared that injustice to the poor is constantly done. If this is done in mere questions of dollars and cents, it is not urprising that physicians upholding the significance of their profession should, for the purpose of saving life, have exaggerated the meaning of their knowledge of crime and responsibility, somewhat perverted our idea of insanity, and so brought about an abuse of legal procedure that has sadly defeated the ends of justice. Professional life has indeed been tainted by commercialization, but the humanitarian motives that guided the tendency to the present abuse of medical knowledge cannot be impugned. The abuse is not as great as that to be found in the legal profession, and its purpose, at least originally, was much higher. There is much need of reform in the matter of the medico-legal expert, but we may as well start with the realization that this is only a part of the reform that is sadly needed in legal procedures in this country, and by no means the most important part.

The question of responsibility for actions, and above all for criminal actions, is not so simple as many people would like to proclaim it. No two men are free to perform an act or not to perform it in quite the same way. Familiar examples are ready to hand: One man finds no difficulty at all in resisting the inclination to take spirituous liquor to excess; another finds it a most difficult feat, often apparently

impossible for him to refrain from indulging to excess almost whenever the opportunity offers, or at least whenever he gets a taste of liquor. This difference between the two men is founded in their very nature. It would be utterly a mistake to praise the one for his abstinence or to blame the other under certain circumstances for his indulgence. Between these two classes there are others quite different individually. Some of them have a slight tendency, and, fearing the worst, do not indulge in it; some of them have a marked tendency which they are able to resist under most circumstances without very much difficulty once they have made up their minds; some are sorely tempted, fall occasionally, yet never become habitual drunkards. For each of these men there is a different responsibility, and so far as they are to be punished a different punishment must be meted out, for it is our effort in the modern time to make the punishment fit the criminal and not the crime.

This same thing holds true for many other forms of crime. Some men readily lose sight of the distinction between mine and thine, and possess themselves of their neighbors' goods almost without realizing that they have done wrong. They are rare, and we have been accustomed to call these people kleptomaniacs. Between these and the man who hesitates to steal, even when starving or for his starving children, there are many degrees of inclination and disinclination toward stealing. The same thing is true to a more noteworthy degree with regard to anger. Anger, the old saw says, is a brief madness. In English we say very frankly that a man who is very angry is mad. In this brief madness he may be led to do things which he would not do at all in his sober senses. Some men easily get into one of these awful fits of anger in which their responsibility is lessened, while others have a calm phlegmatic disposition from which they are scarcely aroused even by the worst forms of abuse or injury, or even physical suffering.

It is evident in all these cases that in order to measure how much of punishment ought to be meted out for acts committed it is more necessary to know the individual than his act. This often becomes an extremely difficult matter, for after the commission of crime every effort is made to make out as little responsibility as possible for the criminal. The easiest way to do this has been to use the insanity plea. As already stated, we have no definition of insanity. It is easy to understand then that there will a disagreement among physicians as to who is or is not insane, and the result is almost sure to create doubt in the minds of the jury that prevents the proper punishment of crime. Now this system is founded on certain wrong principles as regards the administration of justice. While it is difficult to decide with regard to a man's insanity or sanity, it is not difficult to decide with regard to his punishment when the ordinary purposes of punishment are kept well in view.

The old idea of punishment used to be that of revenge. A man

had done a wrong, and what would ordinarily be held a wrong had to be done to him in order that the scales of justice should be maintained level. At the present time we have no such idea at all. Punishment has two main purposes—the prevention of further disturbance of social order by the particular criminal, and the deterrence of others from like acts. If a man takes away the life of another we do not take away his because thus justice will be obtained, but we take it away to prevent him from ever doing anything of the same kind again. A man who has committed murder is more likely to do it again than another. He has committed one breach of social order; we shall prevent him forever from committing another of the same kind. This is the very best deterrent to such crimes that there is. It will be said, of course, that these men could not refrain from doing their acts. It is doubtful, however, whether this contention is true in the great majority of cases, and the proper punishment of such as occur furnishes the best possible motive to help others from the commission of like acts.

This holds true for children at a time when their sense of responsibility for their acts is as yet undeveloped. They can be taught, even very in early life, by properly applied punishment, that need not be severe, that they must not do certain things, and then they will not do them, or at least, will do them much less. This is true not only for perfectly rational childern, but also for those that are to some degree irrational. Punishment is of great importance in the training of children of low grade intelligence, and there is scarcely any child, however wanting it may be in intellect, that cannot be disciplined into conduct that makes it much less bothersome than would ordinarily be the case. This is well known and it is also well known that the attempt to manage such children without punishment would be extremely difficult, not to say impossible. They do not reason about the thing, they are not quite responsible for their acts, but they do connect punishment with what they have done, and are in many cases deterred from doing it again, especially while they realize that authority is near them and that punishment is inevitable. These are the principles on which the adjudication of punishment for crime must be measured. There is nothing else that can be done if society would preserve itself and its members from those who are irresponsible even in minor degrees.

In this matter practical experience is well worth the while. The lower order of creatures, the animals, we do not consider responsible for their acts in the same way as human beings. We know the value, however, of punishment in deterring them. A dog, for instance, by being whipped a few times when he is young, can be taught not to steal things to eat, and taught that there is an inevitable connection between the taking of such things and the infliction of such punishment. I shall not soon forget my first lesson in philosophy from a dear old professor, who, talking of the memory of animals, demon-

strated that they had a memory, from the ordinary experience of mankind with regard to them. "If a cat does something naughty in your room," he said, "you rub its nose in it, and it will not do it again." The cat had no idea that it was doing wrong. According to its way of life it was not doing wrong. It learned, however, from sensory experience that it must not do this sort of thing under special circumstances, and after the lesson has been once thoroughly learned there is no more trouble of this kind.

Individuals who are of less mental stability than normal require, indeed, more careful discipline than average men. The rational may be managed by sweet reasonableness. The defective child must be made to realize that certain actions will surely be followed by painful punishment, though, of course, the main purpose of modern care for such children is to watch over them so diligently as to prevent them getting into mischief. This is after all what we do with the animals, and we realize the necessity for it. Defective human beings approach the animal in their lessened power to resist impulses, and they must be treated in the same way. If we were to save the animals in an excess of tenderness toward them, because we held to the notion either that they did not know any better or else could not resist their impulses, and then permitted them to do things without punishment, we should either have to get rid of animals entirely, or else life would be one continuous readjustment of things to animal ways. Since defectives occur in the general population, it must be realized that far from being less rigid with them in the matter of meting out punishment for things they do that are harmful to others, we must be even more strict with them. Otherwise, we will have to take the bitter consequences of our own foolishness.

It does not make so much difference if the thoroughly rational individual occasionally escapes punishment for something done, but whenever the subrational escapes, he is encouraged to do it again. More than that, the example of his punishment is needed for others. So far as possible, punishment must inevitably follow crime in the world, in order to impress the subrational and deter them from yielding to impulses. Far from being less deserving of punishment in every sense in which a modern penologist cares to inflict punishment, these individuals are more impressed by it, and, above all, need to be more impressed by it. When the subrational know that they can do things without being severely punished for them, they will always abuse that state of affairs. The thoroughly rational man may be depended on to do his duty as a rule without the need of punishment hanging over him. This is not true for the others, and hence the greater increase in crime, and above all in murder, which has made human life cheaper in this than in any other country in the world, as the direct consequence of recent abuses in our penal system.

It has become very clear now that in recent years we have come

to take entirely too lenient a view in these matters, and that many criminals who deserved to be punished, both because in this way they would be prevented from future crime and others deterred by the knowledge of their punishment, have been allowed to escape justice. The tendency is toward too great mercifulness, which spoils the character of the nation, just as leniency to the developing child spoils individual character. Men may very well be insane, in the broad meaning of that term, in the sense that they have done irrational things, but then there is almost no one who has not. The responsibility of most men for a definite action is quite clear in the sense that if they are punished they will not do it again, or will be less likely to do it again, while if they are not punished their escape becomes a suggestion to themselves and to others to repeat such acts. It is for the subrational that we most need to insist on punishment. The cunning of the insane is proverbial, and this extends also to the subrational, and many of these folk realize that their difference from others, their queerness, as their folks call it, is quite enough to make a verdict of insanity in their case assured with the present lax enforcement of law. If the present state of affairs continues in this matter, we are simply allowing ourselves to be led by the nose by these cunning people into the perpetuation of a state of affairs in which they may do what they like because we have become foolishly oversensitive in the matter of inflicting punishment.

On the principle that punishment deters, a man who has killed another man, even under conditions that seriously impaired his responsibility for the act and with evidence of previous lowered mentality, must never again be free to live the ordinary life of men. He must be under surveillance, and should be confined for life in an institution for the criminally insane. For the subrational such a sentence, if known to be inevitable, would usually be more deterrent than even imprisonment in an ordinary prison for life with all the possibilities for freedom which are presented by executive clemency, pardoning boards, and the like. It is absurd to say that a man may have such an attack of mental unsoundness as will lead him to do so serious an act as taking away human life, and then be expected to get over his mental condition so as not to be likely to do the same thing again. Every alienist knows that this is not true. Such acts, when really due to mental instability, occur either in depressed or maniacal conditions and these, as is now well known from statistics very carefully collected, inevitably recur. Society must be protected from such individuals, and this constitutes the most important reason for punishment. It is no longer a personal matter, but a social requirement.

Conclusions. 1. The term insanity is so vague that its use as a plea to enable the criminal to escape punishment is not justifiable in the present state of our knowledge.

2. Responsibility differs in different individuals, but it is never

quite eliminated except in the absolute idiot. For those of lowered mentality, even the animals, punishment has a good effect.

3. Punishment is not revenge, but is meant to deter the individual criminal, and above all to deter others tempted to criminal acts.

4. Punishment is more needed for those of lowered mentality, of whom the expert may well declare that they are insane, than it is for the normal.

5. Subrational individuals with the cunning of the insane will take advantage of our leniency if present conditions are allowed to continue, and we shall have a riot of crime by personal violence.

REVIEWS.

Constipation and Intestinal Obstruction (Obstipation). By Samuel Goodwin Gant, M.D., LL.D., Professor of Diseases of the Rectum and Anus in the New York Post-Graduate Medical School. Pp. 559; 250 original illustrations. Philadelphia: W. B. Saunders & Co., 1909.

The purpose the author had in mind in the preparation of this work is perhaps better expressed at the beginning of the short chapter on the medical treatment of constipation than it is in the preface. He says: "I believe that drugs properly administered accomplish a very great deal toward the relief and cure of *acute* and *spastic* constipation, but, with a few exceptions, consider them inefficient in the curative treatment of chronic *atonic* and *mechanical* constipation. I am of the opinion that chronic costiveness can be cured in the vast majority of cases without the aid of medicines by means of *education, exercise, psychotherapy, dieting, hydrotherapy, massage, mechanical vibration,* and *electricity*—procedures with which I have already dealt. My experience justifies the belief that drugs are helpful in the treatment of this complaint in certain cases when employed in conjunction with the therapeutic measures named, and further, that they are rarely effectual when used alone."

The non-medicinal methods he mentions are taken up seriatim, a chapter or two being devoted to each. The section on the etiology of constipation emphasizes the facts that digestion may be impaired and the act of defecation delayed or prevented through the influence of the mind upon the gastro-intestinal mechanism when disturbed by anger, fright, sorrow, worry, or depression; and also that the act of defecation may be facilitated by concentration of the mind upon this one object, or delayed when it is diverted by reading or in other ways while in the toilet.

In the chapter on psychic (moral) treatment (the title of which—in conjunction with constipation—irresistibly suggests an "Emmanuel movement") the author repeats the above list of mental factors in the production of constipation, and adds: "In exceptional cases sufferers from constipation become so very anxious to have an evacuation that the anxiety, through its influence upon the nerve centres, produces an inhibitory action upon the motor mechanism of the bowel, and in this way delays or prevents a movement. Such

patients should be advised to occupy and divert their mind in some way as the hour for defecation approaches, and to attempt evacuating the bowel at the very first manifestation of the desire." He says, further, that constipation has been cured by hypnotic suggestion, and also by worshipping and praying to idols, religious relics, the Holy Virgin of Lourdes, by wearing chains, kissing precious stones, or by carrying about amulets which have a reputation for healing certain ailments. His discussion of these subjects is interesting, and presents a pleasing variation in the literary treatment of a subject which does not lend itself readily to fancy or romance.

It must not be thought, however, that the book is not a valuable and practical addition to the literature of the subject. Perhaps when it takes in—as it does—a consideration of splanchnoptosis it is going somewhat beyond the promise of its title. But those sections also are well done, and represent the latest views as to causation, symptoms, and treatment of enteroptosis generally. The chapter on massage is admirable. The teachings as to diet and exercise are among the clearest and most practical with which we are familiar. The various methods and appliances for mechanical vibration, for the use of hydrotherapy and of electricity, and other physical therapeutic procedures, are described exhaustively.

The author is obviously an enthusiast in the direction of the drugless management of constipation and of obstipation—which he defines as "a special variety of constipation brought about as the result of some *mechanical* obstruction to the free passage of the feces through the intestines, as, for example, congenital deformities, stricture, pressure by neighboring organs, tumors, foreign bodies, hypertrophy of the rectal valves, angulation, etc." But his enthusiasm has not made him unreasonable or impractical, and the book may be warmly commended as likely to be of great use to both the surgeon and the general practitioner. J. W. W.

American Practice of Surgery. Edited by Joseph D. Bryant, M.D., LL.D., and Albert H. Buck, M.D., of New York City. Vol. V; pp. 965, with 452 illustrations. New York: William Wood & Co., 1908.

In the fifth volume of the *American Practice of Surgery*, regional surgery, comprising Part XVI of the entire work, is commenced. Surgical affections and wounds of the head, by Edward Archibald, M.D., of Montreal, is the opening monograph, and occupies 375 pages; the surgery of the cranial nerves is discussed in 37 pages by Charles H. Frazier, M.D., of Philadelphia; Dr. Charles B. G. de Nancrède covers the subject of surgical diseases, certain abnormities,

and wounds of the face, in a satisfactory and as entertaining a manner as the subject permits; there follow articles on harelip and cleft palate, by James S. Stone, M.D., of Boston; on surgical diseases and wounds of the eye, by George C. Harlan, M.D., of Philadelphia; on surgical diseases and wounds of the ear, by Robert Lewis, Jr., M.D., and on sinus thrombosis of otitic origin, and suppurative disease of the labyrinth, by John D. Richards, M.D., both of New York City; pyogenic diseases of the brain of otitic origin, by Henry Ottridge Reik, M.D., of Baltimore; surgical diseases and wounds of the pharynx, by Charles H. Knight, M.D.; surgical diseases and wounds of the larynx and trachea, by James E. Newcomb, M.D., and on laryngectomy, by Frank Hartley, M.D., all of New York City.

Dr. Archibald has given us, in his monograph on the surgery of the head, an article encyclopedic in character and well illustrated by statistics from the Royal Victoria Hospital, including autopsy and operative investigations of much value. He gives an excellent historical and critical account of the mechanism of skull fractures; and traces our knowledge of the pathogenesis of compression of the brain up to Cushing's work, through the previous observations of von Bergmann, von Schultén, and Leonard Hill. He lays stress on the value of blood pressure in diagnosis and prognosis in cases of compression, and on the fact that the "vagus pulse" is no indication of the *degree* of compression present, merely testifying to its existence; he insists on the value of the depth and rhythm of the respirations as an aid in diagnosis of the degree of compression; and on the fact that though failure of the respiratory centre usually precedes failure of heart action, yet that the former is always due to anemia of the vasomotor centre. The subject of concussion of the brain remains in a much more obscure state than that in which compression has been put by modern investigation, the main symptoms being unconsciousness, with a moderate or rapid pulse, slow and later normal respiration, and low blood pressure. Archibald holds that the immediate unconsciousness is due to anemia of the brain, and that prolonged unconsciousness is caused by a prolonged anemia due to the marked depression of the vasomotor tone, and to capillary thromboses and cerebral (intracortical) œdema; he contends that these symptoms cannot be due to direct injury of the ganglionar cells, since in such case the symptoms would be permanent; he concludes, finally, that the lesions commonly classed as those of contusion are, if present in cases of concussion, mere by-products, not causes themselves of the symptoms of concussion, because they are present also when no symptoms of concussion exist. Should the symptoms of compression appear after those of concussion subside, he urges decompressive craniectomy even in the absence of all focal signs. This is a matter about which surgeons are not agreed; and no doubt more harm than good would be done were the average

surgeon to adopt decompression as a routine treatment. We say the average surgeon, not doubting his mere operative skill, but questioning his ability or willingness to study his cases of head injury so carefully as to enable him to reach a rational conclusion as to which patients he might reasonably hope to save by operation; and we think Archibald does well to insist on the propriety of the surgeon himself exhaustively examining and attentively observing for prolonged intervals patients who are too often left to the entire care of an interne or a nurse, because thought on superficial examination to be hopeless cases. Were surgeons to investigate the mortality of their unoperated as well as of their operative cases of severe head injury, they would be surprised at the mortality, and would be more ready in the future to study in person even apparently doomed cases with a view to their operative relief. These remarks apply as well to fractures as to compression without fracture, and to meningeal hemorrhages due to trauma.

Archibald considers intradural much more frequent than extradural hemorrhages, the former including subdural or extrapial hemorrhages. In discussing the subject of pulsating exophthalmos no notice is taken of the work of de Schweinitz and Holloway (1907), in which they came to the conclusion that ligation of the orbital veins is the best treatment. Archibald still recommends ligation of the internal carotid. He concludes his article with an excellent account of brain tumors, and the technique of cerebral operations.

Frazier's contribution to the surgery of the cranial nerves covers the subject in a satisfactory manner, but the illustrations of excision of the Gasserian ganglion are rather disappointing, making the ganglion appear altogether too easy of access and too near the surface. He gives the mortality of the modern operation as only 3.7 per cent. It is a pity that no bibliography accompanies the text; most of the articles are so supplemented.

Stone recommends that operation for harelip be done between the age of six weeks and three months, and that for cleft palate between six and ten or fifteen months.

The inclusion in a modern system of surgery of articles on the so-called specialties (eye, ear, etc.) is often adversely criticised, but we think the editors are quite justified in not excluding these subjects. This system of surgery doubtless reaches many who have neither modern text-books on these specialties nor specialists at hand to whom they can refer their patients on short notice; and the authors of these sections, with few exceptions, have been careful to confine themselves to such affections as any general surgeon may be called upon to treat. The longest article, that by Harlan, on the eye, is quite an encyclopedia in itself, and, in addition to epitomizing the literature of the subject, is enriched both pictorially and textually from the author's own ample experience. Lewis warns the surgeon in undertaking a "mastoid operation" always to be prepared to open

the lateral sinus, to excise the jugular vein, or to open the skull and explore for cerebral or cerebellar abscess. That this is good advice is not to be heedlessly denied; but it is quite apparent to the general surgeon that the otologist sometimes pushes his operative intervention too far in the case of patients acutely ill; and that better ultimate results will be obtained if at the first operation the mastoid cells and antrum are merely drained, and if the complete operation be postponed until more strength is gained. A. P. C. A.

Arteriosclerosis: Etiology, Pathology, Diagnosis, Prognosis, Prophylaxis, and Treatment. By Louis M. Warfield, A.B., M.D., Instructor in Medicine in the Washington University Medical Department, St. Louis, Mo. With an introduction by W. S. Thayer, M.D., Professor of Clinical Medicine in the Johns Hopkins University, Baltimore. Pp. 165; 8 illustrations. St. Louis, Mo.: C. V. Mosby Medical Book Company, 1908.

Monograph writing has been sadly neglected by the American physicians, yet books dealing with a single subject may be of the utmost value. An essential requisite, however, is that the book should be authoritative. This does not necessarily mean that a name distinguished in medical literature should be upon the title page, but that the writer display a mastery of his subject, not merely the result of reading, but also of personal familiarity.

The present volume does not meet this requirement. It is largely a compilation, and yet only occasionally is the source mentioned. The arrangement is not good. The author does not seem to have understood clearly in some places just what part of the subject he is discussing. Thus, in the chapter on etiology, his paragraph on the congenital form is a mere statement of its existence. The symptoms and physical signs are sadly jumbled, and in different parts of the chapters devoted to them, indeed, in different parts of the book, there is much repetition. It is true that the symptoms and signs of arteriosclerosis are at best somewhat indefinite, but this renders more necessary a clear presentation of the facilities and limitations of our present methods of diagnosis. An unfortunate feature is a tendency to make a statement, and then to retract or qualify it somewhat farther along, leaving the reader in a confused state. The treatment of hypertension throughout the book illustrates this defect.

In the section on prophylaxis, there are a good many familiar platitudes: (page 120) "The struggle for existence is keen," etc.; (page 121) "We live rapidly, burning the candle at both ends," etc. The author lays great stress upon the importance of early diagnosis

and is an enthusiastic advocate of golf in the treatment of the early stage of the disease. He takes a rational view of some of the smaller vices, such as tobacco and alcohol, but believes strongly in the deleterious effects of butcher's meat, and seems to prefer a low proteid diet. Among the drugs, he speaks with approval of two, the iodides and nitrites. To these he adds morphine to meet certain severe indications and an occasional stimulant for heart failure. In the final chapter, entitled "Practical Suggestions," there is considerable preaching. "It is a wise maxim never to drive a horse too far. Apply this to a human being, and the rule holds equally well," and much more of the same sort. The book could have been compressed into less than 165 pages, and yet all the information it contains be well presented. It could have been expanded to a great many more pages with advantage by a more thorough treatment of its subject. The introduction by Dr. Thayer is an interesting piece of writing. J. S.

PARCIMONY IN NUTRITION By SIR JAMES CRICHTON-BROWNE, M.D., LL.D., F.R.S., Lord Chancellor's Visitor in Lunacy, London. Pp. 111. London and New York: Funk & Wagnalls Company, 1909.

To an American accustomed to the orthography taught in his native schools, the first word as spelled by Sir James in the title of his book appears rather startling; to an American taught to regard Professor Chittenden's researches in metabolism, as he is wont to regard Cæsar's wife, above suspicion and reproach, the contents of Sir James' little monograph appear more startling.

Parcimony in Nutrition is a revised and expanded version of the author's presidential address to the section of preventive medicine at the meeting of the Royal Institute of Public Health, at Buxton, England, in July, 1908. As the title suggests, the book is more or less of a satirical invective against the present-day theory that man has for years been eating in excess of all natural requirements, as elaborated by Chittenden. We hardly turn the second page before the inspirer of the address and its present elaboration can be plainly seen between the lines. But ere our noted physiologist is openly assailed, the avenue of attack lies over the body of an American gentleman, who has "literally chewed himself back into health after being middle aged, obese, dyspeptic, in failing health, rejected by an insurance company, and unaided by many medical men consulted." Sir James with cruel pen and biting sarcasm slays Horace Fletcher in the first chapter, and apart from sporadic resurrections of his victim devotes the remainder of his book to the combat with Chittenden.

It is impossible to review *Parcimony in Nutrition* without a shade of levity. Not that the book is *not* serious, for we believe the author is sincere in his statements, but wittingly or not, he has infused an informal, chatty atmosphere into his work which instructs while it amuses, informs us while we smile. Certain of Chittenden's deductions, arrived at from his painstaking studies, are swept aside with a swish of the pen, and mere statements, unproved by research, are offered in their place. On the other hand, statistics and argument are presented which, it must be confessed, seem difficult to refute. The author points out that Chittenden's contention that the Japanese subsist on a low protein diet is wrong, and he shows that when body weight and body surface are considered there is not much difference between the individual food supply of the Japanese and Voit's standard. The Greeks he shows later on to have been large eaters of meat, despite which fact the group of statesmen, artists, and literary men, who were born 530 to 430 B.C., has never been equalled in any race.

Statistics are not lacking: Chapter III is devoted to the subject of prison experiences (collected from authorities not personal), which show what abundant rations are supplied the convicts; and it concludes with the epigram, "Nothing is more demoralizing than chronic hunger." The author quotes a disciple of Fletcher as saying the British navvy is of little use after forty, and questions, pertinently, "If excesses in diet are this slow in exacting their penalties, may it not be that a subliminal diet may be also tardy in manifesting its untoward effect? May not imperceptible undermining be going on for a long time before there is any obvious collapse?" He places all Chittenden's scientific discoveries against empirical observation and states that "farmers have valued and cultivated leguminous crops, the four-course rotation of turnip, barley, clover, wheat having been popular two thousand years ago, but it is not twenty years since Hellriegel and Wilfarth revealed to us that leguminous plants bear on their roots nodosities abounding in bacteria capable of fixing atmospheric nitrogen and thus supplying the nitrates essential to grain crops. From the dawn of history men have been engaged in adapting domestic animals to their uses, by making crosses between varieties with differentiating characteristics, . . . but it was only in 1866 that Mendel formulated his law of inheritance in hybrid varieties."

There is an abundance of similar arguments in the book, with a corresponding lack of scientific experimentation to prove the author's point, which is that practical experience and physiological requirements of mankind in general more than offset all the protocols of metabolic studies. The book is well worth the perusal of those desiring to be amused and instructed by this opponent of parcimony in nutrition. E. H. G.

A Text-book of Surgical Anatomy. By William Francis Campbell, M.D., Professor of Anatomy in the Long Island College Hospital, New York. Pp. 675; 319 original illustrations. Philadelphia and London: W. B. Saunders Co., 1908.

Writers on surgical anatomy tread on dangerous ground; their path is narrow and strewn with stones; on one side lies the barren field of systematic anatomy, from which even the stones have been removed, and on the other is the flowery mead of surgery; and the temptation to step aside momentarily, or even to walk persistently on one side or other of the path usually is too strong to be overcome. The hard task set for himself by a writer on applied anatomy is not only to keep in the path, but to clear it of the obstructions which pure anatomists have placed there, perhaps unintentionally, by their failure to explain the why and the wherefore of the construction of the human body. The applied anatomist attempts in a very real sense "to justify the ways of God to man."

This volume is divided into six parts, treating of the head and neck, the thorax, the upper extremity, the abdomen and pelvis, the spine, and the lower extremity. In general, each subject is discussed under the headings of (*a*) surface anatomy, (*b*) regional anatomy (*e.g.*, shoulder arm, elbow, forearm, etc.); and under each region discussed are to be found short paragraphs on surgical anatomy; but Dr. Campbell pays almost too much attention to pure anatomy, and when he does venture into the fields of surgery it is as one seemingly unfamiliar with many of the practical details of which he writes. This is evidenced, for instance, by his unconquerable habit of making hard and fast rules of surgical procedure based on theories unsupported by clinical experience. Thus, he advises the retention of a plaster cast for several weeks after the reduction of a congenital dislocation of the hip; many months is the usual time. He utters the edict that tuberculous disease of the tarsus requires amputation, and that resections must be confined to traumatic affections; whereas it is well known that recovery is frequent after conservative operations for tuberculosis of the tarsal bones. In doing a tenotomy of the tendo Achillis, he warns against injury of the posterior tibial vessels and nerve; we wonder that he does not caution the student to beware of the external iliac artery in operating for appendicitis! He classifies every tuberculous abscess of the tibia as a "Brodie's abscess."

That the author is not thoroughly familiar with modern surgical literature is shown by his describing gastro-enterostomy as a "drainage" operation, in which the "vicious circle" may be prevented by making the anastomosis at the *most dependent portion* of the stomach; and by his quoting, as if of recent utterance, remarks in approval of Jonnesco's operation for exophthalmic goitre.

In discussing the surgical anatomy of the extremities, Dr. Camp-

bell has done better: his accounts of dislocations and fractures are usually sufficient, if not illuminating; some attention has been paid to the lymphatics; the descriptions of surface anatomy and landmarks are clear; and the illustrations show what is intended. Although all the illustrations are called "original" on the title page, the majority are so only in so far as they have been redrawn from other works. They are all painfully diagrammatic; a few are truly original; and they are the only original things in the entire volume.

A. P. C. A.

Gonorrhœa in Women. By Palmer Findley, M.D., Professor of Gynecology in the College of Medicine of the University of Nebraska. Pp. 112. St. Louis: C. V. Mosby Medical Book and Publishing Co., 1908.

This monograph, dedicated to Neisser, has been prepared by the author to stimulate professional interest in a very important and much neglected subject. He has appended an extensive bibliography, which forms a very valuable feature of the work. It seems remarkable that some such book has not appeared before, since the importance of the subject and the very vague ideas held by the great mass of the profession with regard to many of the problems of sanitation, prophylaxis, and treatment have certainly demanded it. However, the book has at last appeared, and the fact of its publication gives us great satisfaction, since there is no subject in the domain of present-day medicine which needs more careful elucidation. The author offers as an introduction a short but sufficiently complete historical account of gonorrhœa, the next two chapters being concerned with its etiology and pathogenesis; an interesting section upon the pathology is followed by a ten-page description of the course of gonorrheal infection, while chapters upon its diagnosis, frequency, sociological bearings, and treatment follow. The book concludes with a three-page account of the systemic gonorrheal infection and the bibliography before mentioned. In the section devoted to treatment the author has included the conclusions of Butler and Long, based upon their experience with the vaccine method of treatment in the vulvo-vaginitis in childhood. A considerable portion of this monograph is composed of excerpts from the writings of various authors. In many instances they have been quoted verbatim. This occasions some repetition and renders the book a little less readable than would have been the case had the author incorporated the substance of their work alone. While to the specialist there is nothing new in the book, except possibly the vaccine method of treatment, and while certain advice relative to intra-uterine treatment may well be objected to, there is, on the other hand, a very great deal which to the general

practitioner will come as new thought and which will well repay his careful digestion. We feel that Dr. Findley has done a good work in the preparation of this book, and hope that its circulation will be widespread. W. R. N.

SUGGESTIVE THERAPEUTICS, APPLIED HYPNOTISM, PSYCHIC SCIENCE. By HENRY S. MUNRO, M.D. Second edition, pp. 360. St. Louis, Mo.: C. V. Mosby, 1908.

IN his preface the author states that he not only brings to the consideration of the medical profession the results of his personal experience and clinical evidence as proof of the value of suggestive therapeutics in the general practice of medicine, but also a detailed explanation of how to apply suggestion efficaciously, both with and without hypnotism as a therapeutic adjunct. He also states, with a view of forestalling criticism, that his book is not intended principally or even mainly for neurologists or psychotherapeutists, but for the vast mass of the profession to whom this entire field is new.

To criticise this book or even adequately to review it is beyond the ability of the reviewer, who is merely a neurologist. For while it is acknowledged that there is room for an adequate presentation of such a subject one's mature judgment is immediately put in abeyance, and in spite of whatever self command one has, it is almost impossible to read such a book without becoming incensed at such a preposterous presentation. There are so many books which are written nowadays with the presumption that the poor medical man who is asked to buy and read the book knows absolutely nothing. The author, evidently, is one of those who not only thinks so, but actually proves it, for in his text, the better to emphasize to his reader what he wants him to infer, for he presumes that the reader does not use his intelligence, he uses heavy type. As an example of his method we may quote a paragraph taken at random on page 36: "Tell him to relax. Sit down. Sleep on. Doctor, suggest to your subject that when you rub this medicine on his arm three times it will be dead and have no feeling in it as long as he is in this room. Tell him that when he is awake you can thrust a pin in the skin of that arm while he is looking at it, and he will have no feeling in it, that his arm will be perfectly dead as long as he is in this room."

It is a matter of regret that the cure of patients by hypnotism, the use of which the author takes as a matter of course, as if everybody employed it dozens of times a day without the slightest ill effects now or hereafter, in the experience of most neurologists, is not so easy as the author seems to assume. The conclusion that one arrives at after having somewhat painfully perused this book,

is that Dr. Munro, after all, is the only person who really does know anything at all of suggestive therapeutics, applied hypnotism, and psychic science. It is a pity that he did not consider the vexing question of the difference between Christian Science and the so-called Emanuel movement, to say nothing of thought healers, mechanoneurotherapists, osteopaths, and the latest cults of soul massage. And yet this book is now in the second edition!

T. H. W.

The Muscles of the Eye. By Lucien Howe, M.A., M.D. Professor of Ophthalmology in the University of Buffalo, New York. Vol. II, Pathology and Treatment, pp. 481; illustrated. New York and London: G. P. Putnam's Sons, The Knickerbocker Press, 1908.

The second volume of Howe's exhaustive work deals with imbalance of the intra-ocular muscles, extra-ocular muscles (heterophoria), strabismus, paralysis, and atypical movements (nystagmus, etc.), and inflammations and injuries of the muscles themselves. The concluding portion is devoted to operations. An extensive bibliography is added, and pictures and short biographical summaries of one-half dozen eminent ophthalmologists are appended.

The book is not easy reading, but this is not the fault of the writer; but because a degree of preliminary knowledge such as not every oculist possesses is necessary. Careful study of the first volume will supply such deficit to a certain extent, but the questions considered in this volume are more difficult, involving as they do the same problems of anatomy and physiology plus the action of disease and anomaly, much of which is novel and unknown.

Haphazard and rule of thumb methods find no favor with Dr. Howe, and he is entirely correct in the statement that for a proper recognition and appreciation of the importance of the subject of heterophoria, thorough anatomical and physiological knowledge obtained from actual dissections and experiment, the proper appliances, an open mind, with time and patience, are prerequisites.

In view of the exaggerations that have been exploited in some quarters regarding this matter we note with pleasure the sanity of Howe's views. He is not in favor of actively treating every form of muscular deviation which can be recognized, and he admits the limitation of all the means at our command for correcting some of them. The oculist is also admonished not to be oblivious to the effect of general conditions upon the muscles of the eye as well as the reverse.

These two volumes will no doubt find a place upon the shelves of every physician who engages in this kind of special work. There

is room for differences of opinion, but the views expressed in the work should be carefully pondered by all who attempt to treat the anomalies of the ocular muscles. It is much better to pass these by entirely than to attempt their correction in a hit or miss fashion without adequate knowledge of all the factors of the problem. T. B. S.

Handbook for Attendants on the Insane. By the Medico-Psychological Association, Fifth edition; pp. 390; 19 illustrations. Chicago: W. T. Keener & Co., 1907.

The English Medico-Psychological Association has filled a distinct need by taking into its own hands the publication of a handbook for the use of attendants on the insane. This is the fifth revised edition. It was not until the second edition that the association took direct charge, intrusting it to a committee of eleven members. The scope of the book has been enlarged in each edition, since the training already given in different institutions has created a demand for some advanced teaching which this book attempts to fill. Its contents are well arranged and include the ordinary instruction in anatomy and physiology of general and nervous diseases and care of the sick. It is needless to review a small book of this kind any more than to say that it is well done and highly to be commended.

T. H. W.

Action de la Contraction Utérine sur l'Œuf Humain, Phénomenès Passifs de la Grossesse et du Travail. By Dr. Paul Bouquet, Médicin de la Maternité de Brest, Directeur du Cours Départmental d'Accouchement. Paris: G. Steinheil, 1908.

This monograph covers a subject of considerable theoretical and practical interest to obstetricians. It is written in a discursive style, and its practical conclusions are not such as to add greatly to our therapeutic resources. It should, however, find place in obstetric libraries, and may be consulted to advantage by those studying and writing upon obstetrics.

Bouquet's idea has been to study by references in literature the results of uterine pressure upon the human ovum in its various stages of development. He endeavors to ascertain the effect of the pressure upon the lower pole of the ovum, and considers the results of such pressure in a low insertion of the placenta and in placenta prævia. He further considers the results of painless and painful uterine contractions upon the ovum at different stages of its develop-

ment until birth begins. The phenomena of engagement and dilatation receive considerable attention. The point brought out most strongly by the monograph is the fact that before the rupture of the membranes the fœtus and its envelopes constitute a body of little elasticity; uterine pressure is then exerted without much counterpressure and becomes essentially a mechanical phenomenon. This observation Bonquet applies to the various phenomena of dilatation and labor.

The practical deduction from his observation is to emphasize the protection given to the fœtus by the membranes and amniotic liquid, and to call renewed attention to the fact that before the membranes rupture resistance of the uterine contents must influence the blood tension of the mother. The author quotes extensively from Varnier, Tarnier, Mauricean, and others. He offers no original experiments or detailed clinical observation of his own. E. P. D.

Manual of the Course of Study at the Bancroft Training School for Mentally Subnormal Children, Haddonfield, N. J. Arranged by Margaret Bancroft and E. A. Farrington, B.S., M.D. Pp. 120. Philadelphia: Ware Brothers Company, 1909.

Within recent years much attention has been paid to the training of subnormal children, and as a result many institutions have been created for this purpose. Of these, the Bancroft Training School is one of the best. The manual under review has been arranged by Margaret Bancroft and Dr. E. A. Farrington, and encompasses the details of instruction used in that school. There is nothing original about it. It is, however, well worked out and shows careful preparation. T. H. W.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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A Simple Procedure for the Recognition of Blood in the Urine.—Albarran and Heitz-Boyer (*Presse méd.*, 1909, xl, 361) point out the need of an easy method for the recognition of blood in the urine, in addition to the test for albumin and the microscopic examination of the centrifugalized sediment. To give the proper interpretation to albumin in the urine the absence of blood must be definitely determined. The microscopic detection of red blood corpuscles is the best, but in vain in case of complete hemolysis. The reagent used is an alkaline solution of phenolpthalein, prepared by boiling phenolpthalein, 2 grams; anhydrous potassium, 20 grams; powdered zinc, 10 grams, in distilled water, 100 grams. The clear, colorless filtrate will keep for months. One c.c. of this reagent is shaken with 2 c.c. of the urine to be tested and 3 or 4 drops of ordinary oxygenated water is added. A red fuchsin color is positive, the intensity depending upon the amount of blood present. It is positive for blood in the dilution of 1 to 100,000. It is specific for blood or hemoglobin, and obtained as well in alkaline as in acid urines. Albumin, pus, sugar, bile pigments, chloroform, or thymol do not affect the test. Uric acid, urates, acetone, indican have no influence, nor do such drugs as the iodides, bromides, salicylic acid, morphine, phenacetin, or urotropin. In a word, of easy execution, specific and delicate, this reaction is an excellent clinical procedure for the recognition of blood in the urine.

Alcoholic Epilepsy.—Redlich (*Epilepsia*, 1909, i, 41) states that there are certain cases of alcoholic epilepsy in which the abuse of spirits is the only excitant in a person disposed thereto, either through heredity or by a "trauma." In other cases, the use of alcohol may bring about epilepsy

without direct, natural predisposition, either in the form of true alcoholic epilepsy (Bratz) fits appearing under influence of intoxication, or in the form of genuine, constitutional epilepsy, whereby fits also appear, irrespective of spirits being taken or not. Moreover, there are intermediate forms of these types. Redlich discusses especially alcoholic epilepsy. Here the epileptic attack may appear at the moment of maximal intoxication. Emotion and trauma of the head will heighten the action of alcohol. In some cases the fit occurs only during the after-intoxication period, twelve to twenty-four hours after the last dose of spirits. In common epilepsy the attacks very frequently appear at that time. Delirium tremens may simultaneously occur and either this or epilepsy follow on an alcoholic bout. These two conditions are closely related both in cause and nautre. It is a question whether abstention from alcohol may cause fits or not.

Purpura Hæmorrhagica and Hereditary Syphilis.—Sabrazes and Duperie (*Arch. d. mal. d. cœur, d. vaisseaux, et du sang*, 1909, v, 257) report the case of a subicteric infant, with fissure at the corners of the mouth, who succumbed to œdema and hemorrhagic effusions. Eight hours after birth the liver, spleen, adrenals, lungs, and skin presented multiple effusions of blood, old and new. They contained spirochetes to the exclusion of other organisms. The multiple hemorrhages are due to rupture of distended capillaries resulting from a mechanical obstruction by the interstitial granulomatous lesions of the organism. The injury done by this septicemia of spirochetes was evident in other organs. The thymus was sclerotic; the kidney glomeruli, and the panceras showed signs of irritation; the myocardium and vasa vasorum of the great vessels contained spirochetes. The relation of hemorrhages to hereditary syphilis has been observed by Levaditi and others, but Sabrazés and Dupérié feel that this case explains more clearly the mechanical origin of the œdemas and hemorrhages. In all these cases the ubiquitous presence of the spirochete is remarkable. If the infant survives, there will remain in the organs traces more or less profound of the multiple lesions —scleroses, defective vessels, collateral circulations tending to hemorrhages, hypoplasia of parenchymas. The infinite variety reveals the vast field of functional insufficiencies, isolated or combined, which may have as origin an hereditary syphilis recognized or overlooked. Rational treatment of the parents and most active and precocious therapy of the newborn are the salvation.

The Small Heart of Cirrhosis.—In three cases of pure alcoholic cirrhosis of the liver, without tuberculosis or arteriosclerosis, Carnot (*Progrès méd.*, 1909, v, 61) has found at autopsy the heart diminished in size and weighing, respectively, 125, 150, and 183 grams instead of the average 250 grams. In the last case the wall of the left ventricle measured 10.5 mm. instead of 12 mm., and the circumference of the aorta 57 mm. instead of about 65 mm. The small size is readily made out by percussion *intra vitam*. The existence of renal or arterial disease is the exception; when present, causing hypertension and cardiac hypertrophy. The small size of the heart must be in relation to the hypotension in the arteries as pointed out by Gilbert and Garnier. Both are dependent upon the hepatic obstruction which causes a portal hypertension and a

diminution in the volume of blood coursing through the heart in a unit of time. Ludwig, Garnier, and Gilbert have noted a fall in the carotid pressure immediately after ligation of the vena porta. This hypotension lessens the work of the heart, and the diminished size is its adaptation to the lessened function.

Oatmeal in the Curative Treatment of Diabetes.—Von Noorden (*Medical Press*, 1909, lxxxvii, 446) explains the method of procedure in the oatmeal cure, emphasizing the necessity of certain definite rules and the fact that only a certain number of patients are benefited. In 28 per cent. of 400 cases he obtained excellent results. These were cases of medium severity. The treatment does not cure the diabetes, but considerably diminishes its dangers. It is of the greatest importance that from time to time the patients should assimilate large quantities of carbohydrates. Thereby the danger of acid poisoning is averted. For this reason the treatment should be repeated four or five times a year. It requires only a week, as a rule, three oatmeal days, and two vegetable days. Experimental glycosuria was produced in dogs by injecting adrenalin. If a small amount of extract of oats were added, no glycosuria resulted. As there is no doubt that adrenalin diminishes the internal secretion of the pancreatic gland, the probability arises that the oat extract has the opposite effect, *i. e.*, that the extract is a stimulus for the pancreatic secretion. After extirpation of the pancreas in animals, the oatmeal treatment has a very bad effect, and the oat extract does not diminish the glycosuria. This also seems to prove that the oat extract only acts by influencing the internal pancreatic secretion in a direct and specific manner. Therefore, von Noorden presumes that the astonishing effect of the oatmeal treatment is due to the circumstance that with the oats is introduced into the body small amounts of a substance which acts as a stimulant upon the internal secretion of the pancreatic gland.

A Note on the Absorption of Fat.—Whitehead (*Amer. Jour. Phys.*, 1909, xxiv, 294) undertook to determine if emulsified fat was absorbed as such by the intestinal mucosa. The work of Kastle and Loevenhart, demonstrating the universal presence of lipase in the tissues and its reversibility of action to synthesize as well as split fat, has strengthened the view that fat is absorbed in the form of soluble soaps. Decomposed in the intestine it is built up again in the villi. On the other hand, there are no *a priori* reasons why fine emulsions may not be absorbed, and it is well known that the intestinal contents are such as to favor the formation of emulsions. In view of Loevenhart's work, the finding of fat droplets in the epithelium and lacteals of villi does not prove that the fat was absorbed as such. Whitehead fed a cat with butter stained in Sudan III, killed the animal, and sectioned the small intestine. There was an entire absence of red globules of fat in the villi, although masses of red butter still unsplit were evident in the lumen of the intestine. Either the fat had not been absorbed as such or not at all. But by staining these same sections in Sudan III fat was demonstrated in great abundance in the epithelium and lacteals of the villi, in the form of minute red globules. Undoubtedly the fat was absorbed, inasmuch as the animal had been previously starved. And it is evident that it was not absorbed as such but taken up in water-soluble forms

of its constituents. Presumably, the fatty acids entered the villi in the form of soaps: for the villi were stained a diffuse red, which was readily decolorized by weak alcohol, and it is well known that oleic acid stained with this dye is decolorized with much greater difficulty than soaps.

Two Cases of Gonococcic Septicemia Terminating in Cure, and followed by Typhoid Fever.—Dieulafoy (*Presse méd.*, 1909, xl, 353) reports two cases of gonococcic septicemia following an acute specific urethritis. In the first case the gonococcus was obtained from the urethra and the blood on two different occasions. Gonococcic vaccine was employed as treatment. Recovery occurred after the third injection. The scar of a mitral endocarditis remained. After two days of apyrexia there followed a short attack of typhoid fever with positive blood culture and serum reactions. The second case ran a similar course with blood cultures positive for the gonococcus and B. typhosus in their respective attacks. Dieulafoy remarks upon the infrequency of cure of gonococcic septicemia, and comments upon the possibilities of the pathogenesis of the typhoid infection. Ruling out the possibilities of the gonococcic vaccine being contaminated by typhoid bacilli, of "house" infection in the wards, and the typhoid bacillus being associated with the gonococcus in the first febrile attacks, he considers it most likely that these patients bore the bacillus in a latent state, possibly in the biliary passages; and that the gonococcal septicemia resulted in a condition favorable for their explosion.

Viscosity of the Blood and Iodine.—Boveri (*Presse méd.*, 1908, lxiii, 546) studied the effect of iodine upon the viscosity of the blood in twelve patients with arteriosclerosis and hypertension. He gave them sajodin, iodipin, and iodogelatin in the ordinary doses, and measured the viscosity of the blood and serum at intervals of ten, twenty, thirty, and forty hours. A very definite diminution in the viscosity of the blood followed in eight of the cases. The serum did not show the same changes as the blood in toto. It remained stationary. The arterial tension fell during the treatment, as much as 65 mm. of mercury (by the Riva-Rocci apparatus) in some instances. The same results were obtained in rabbits by the administration of the iodide of sodium and potassium. The viscosity of the blood was found to be increased or diminished constantly with the increase and decrease in number of red blood corpuscles. In a general way there seems to exist a parallelism between arteriosclerosis, viscosity of the blood, and response to treatment. The greater viscosity is found in the more advanced arteriosclerosis; whereas iodine accomplishes a diminution in viscosity in inverse proportion to the severity of the disease.

The Compensatory Role of Accidental Subcutaneous Emphysema in Two Cases of Pneumothorax with Valvular Opening.—Perrin (*Arch. gén. de méd.*, 1909, iii, 193) calls attention to the fact that subcutaneous emphysema following the puncture of a pneumothorax is considered a serious complication, especially when the latter is of the valvular type. The emphysema may become generalized and require pleurotomy, or a cellulitis may be set up by extension from the septic pleurisy.

Fortunately, it is not always of such bad prognosis, and the author reports his two cases as evidence of this fact. In both cases the subcutaneous emphysema was produced accidentally by thoracentesis. The puncture wound acted as a veritable safety-valve, compensating for the continual accumulation of air in the pleural cavity by evacuating it into the cellular tissues where it was absorbed. Cure resulted spontaneously. However, Perrin emphasizes the severity of this complication in the majority of cases. The likelihood of the accident is much less if a very fine, sharp needle is used for puncture, so that the opening of the puncture may be easily obliterated. These patients should be watched very carefully in order to interfere if suffocation should be imminent or if the emphysema appears to spread very rapidly. It might be advisable to leave in place a needle with valves as devised by Boinet, which would allow the escape of air but no inflow. Perrin suggests this instrument as useful in the systematic treatment of pneumothorax of the valvular type.

Fixation Abscess in the Grave Accidents of Saturnism.—Carles (*Bull. gén. de thér.*, 1909, February 8) advances for trial an interesting medication for the more grave accidents due to the accumulation of lead in the organism. By producing a fixation abscess through the subcutaneous injection of turpentine, he hoped to withdraw from the body sufficient lead to make a difference in the symptoms of saturnine intoxication. He carried this out in dogs poisoned with red lead, and from the pus in every instance obtained more lead than in equal amounts of the blood, brain, and liver This was equally successful in a dog poisoned over a period of two months. The clinical application was made in only two cases, one of bronchitis, and one of lead colic. Again, lead was extracted from the pus of the abscess. However, the quantity was small and the clinical results not sure. His explanation is a phagocytosis of the visceral lead by the leukocytes and its transportation to the fixation abscess.

Carnot (*Progrès méd.*, 1909, xii, 159) points out that these results are confirmatory of his own, published in 1894, when he contended that metallic salts in circulation are localized in the region of inflamed organs and that it suffices to produce a traumatism or local suppuration in order to fix the lead salts absorbed. This elective fixation of circulating poisons in the region of inflammatory lesions is comparable to that of microörganisms. It permits the explication of an attack of gout in the region of an injured joint by the elective fixation of uric acid. It explains certain localizations of medicines in the course of local inflammations, as salicylates in rheumatic foci and mercury and iodides in syphilitic lesions. Carnot agrees with Carles that the method may have therapeutic usefulness in a case in which it is urgent to disintoxicate the organism.

Pathological Diagnosis of Mitral Valve Lesions.—Schabert (*Deutsch. Arch. f. klin. Med.*, 1909, xcvi, 116) has studied all hearts for the last two years by the "closure test" (Schliessprobe). The wall of the auricle is slit down to the auriculoventricular opening, exposing the mitral valves. An opening is then made in the apex of the left ventricle and a stream of water is directed into the ventricle, while the valves are directly observed.

In this way one can, as a rule, readily differentiate between normal, insufficient, and stenotic valves. Weighing the ventricles separately, according to the method of Müller is also of great aid, and in combined mitral lesions the weights of the ventricles and the closure test allow of closer analysis than has heretofore been possible. The closure test has revealed a surprising number of cases of pure mitral stenosis. Schabert concludes: (1) The closure test is satisfactory in the great majority of cases examined, and permits of far more certain decision as to the presence of stenosis or insufficiency than the usual inspection of the valves. (2) The results of the closure test should be controlled by weighing the ventricles separately. (3) Cases of pure stenosis are only recognizable by the closure test. (4) In pure mitral insufficiency an hypertrophy of the left ventricle is always present.

Myeloid Chloroma.—Jacobaeus (*Deutsch. Arch. f. klin. Med.*, 1909, xcvi, 7) reports two new cases of myeloid chloroma or chloroleukemia, as Naegeli calls the disease. This brings the total number of myeloid chloromas to ten since Turk's original case in 1903. In both of Jacobaeus' cases myelocytes and myeloblasts were conspicuous in the blood picture. The diagnosis was made antemortem in the first case by the greenish skin metastases, in the second by blood examination; 5 c.c. of blood was treated with citrate and centrifugalized. The resulting layer of leukocytes had a faint greenish tint. Controls from six normal individuals and chronic myeloid leukemias, which were examined similarly, showed no trace of green in the leukocyte layer. Thus, for the first time, examination of the blood alone has led to the correct diagnosis of chloroma, as the postmortem finding subsequently proved.

SURGERY.

UNDER THE CHARGE OF

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The Bone Fragility and Eburnation of Rachitis.—Blanchard (*Amer. Jour. Orthop. Surg.*, 1909, vi, 616) says that normal bone ossifies slowly. Normal unossified bone bends under the pressure of the osteoclast. Rachitic bone may remain friable indefinitely, but with favoring conditions eburnates suddenly. Eburnated bone breaks hard, and is not amenable to any pressure of iron braces or a plaster of Paris. Very mild bow-leg that is not at all or only slightly rachitic may be autocorrected. Rachitic bow-leg, knock-knee, and anterior bent tibia are never autocorrecting and frequently grow worse in bed. Badly deform-

ing, rachitic knock-knee and bow-leg are occasionally seen in children that have never walked. Rachitic bow-leg predisposes to rachitic coxa vara. Rachitic knock-knee predisposes to rachitic coxa valga. Knock-knee is distributed distortion of all the long bones of the legs, having its usual inception in an inbend of the upper tibial shaft. Bow-leg is a distributed distortion of all the long bones of the leg, having its usual inception in an outbend of the femoral shaft, though, as the deformity progresses, the secondary outbend of the tibia frequently becomes the most pronounced. The skiagraphic observations of a large number of cases would seem to prove quite conclusively that these deformities closely follow fixed lines of development. Rapid osteoclasis is the safest and quickest means of correcting most of the rachitic deformities of the legs. The term rapid osteoclasis is used because osteoclasis that is not rapid is always a failure and brings the osteoclast into disrepute. These conclusions are drawn from nearly 1000 cases of knock-knees, bow-legs, and anterior bent tibiæ corrected by rapid osteoclasis with the Grattan osteoclast.

A New Method of Treating Hydrocele,—Marcozzi (*Ann. d. mal. org. gén.-urin.*, 1909, i, 739) says that the tunica vaginalis is important for the function of the testicle. A certain number of hydroceles, especially those of recent origin, show only slight distention and thickening of the tunic, and for this reason have a tendency to cure. In such cases it would be best to try first some non-operative treatment, such as the injection into the sac of some suitable medicament, like iodine or silver nitrate. It does not always cure, but is frequently followed by recurrence. This is probably due to the fact that the effect of the injected substance is too fleeting to permit it to act long enough upon the chronically inflamed serous sac. Marcozzi has employed in 10 cases an injection of fragmented magnesium thread. This is first washed in ether and then sterilized in distilled water. After being injected it is easily absorbed and produces a durable stimulation and reaction of the serous surfaces. As the result of his success in the use of this method, he concludes that it is more advantageous than all others. It produces no pain and permits the patient to continue at his usual occupation. There is no risk of necrosis of the scrotal tissues, such as has followed the employment of the tincture of iodine.

Partial Sequestration of a Transplanted Piece of Bone.—Axhausen (*Arch. f. klin. Chir.*, 1909, lxxxix, 281) resected the upper end of the humerus and the joint capsule for a secondary cancer in this situation, which had followed an operation for the removal of a cancer of the breast, two and one-half years before. There was no recurrence in the scar or in the lymph glands. After removal of the upper end of the humerus, the second, right metatarsal bone was extirpated and its attached muscles carefully removed. Then several incisions were made through its periosteum and its proximal end was freshened and pointed. Its pointed end was then driven into the medullary cavity of the upper end of the divided humerus. The two attached bones were then replaced so that the cartilaginous surface of the metatarsal bone fitted into the glenoid cavity of the scapula. The muscles, which had been detached from the humerus, were sutured to the periosteum of the upper divided

end of this bone and the wound closed with the introduction of a small drain. Moderate infection followed, but although the transplanted bone was bathed in pus, it did not become entirely necrotic, but was only partially sequestered. Recurrence of the carcinoma in the humerus made another resection necessary, so that a histological examination was possible. This showed that the original transplanted bone substance had died completely, and that the greater part of the periosteum had lived and had given rise to abundant bone formation. Some of the medullary substance also remained, especially in the region of union, where there was considerable callus formation. The new bone which had taken the place of the dead bone almost completely, came only from the periosteum and medullary substance. Lacunar resorption and apposition played an important part in the process. In the presence of a moderate infection, although the whole transplanted bone becomes necrotic, the greater part may be retained and only a part be extruded as a sequestrum, that is, only a partial sequestration of the total necrotic bone occurs.

A Case of Stasis Cyanosis following an Epileptic Seizure, Simulating Traumatic Asphyxia.—Alexander (*Annals of Surgery*, 1909, xlix, 762) reports the case of a young man who fell to the floor in an epileptic attack and was taken to the hospital. At the time of the fit it was observed that his face was blue, that his eyes were bloodshot, and that he remained unconscious several minutes. On examination at the hospital, his face and neck were found to show a diffuse, bilateral, bluish, and slightly punctiform discoloration of the skin, that disappeared but slightly, if at all, on pressure. This discoloration stopped with a decided line of demarcation just where the tight collar passed around the neck. Below this line the skin was of a normal color. The eyes showed a marked subconjunctival ecchymosis, more marked on the left. The chest and abdomen failed to show any signs of recent injury. Fourteen days after the accident the discoloration had almost disappeared. Alexander thinks that the factors producing this condition are similar to those producing traumatic asphyxia, namely, a fixed thorax, a closed epiglottis, an increased intrathoracic pressure, a lack of aëration of the blood, and the incompetent and absent valves of the jugular, subclavian, and facial veins.

Pancreatic Surgery.—Rosenbach (*Arch. f. klin. Chir.*, 1909, lxxxix, 303) says that in recent years we have gained a more exact clinical knowledge of pancreatitis and pancreatic necrosis, and, particularly, we have learned to estimate more correctly their relation to diesases of neighboring organs, as the gall-bladder and stomach. We have also advanced considerably in our knowledge and successful treatment of wounds of the pancreas. Isolated subcutaneous injuries are rare. Up to 1905 only 8 uncomplicated cases had been reported, and all these, whether operated on or not, died. Garré, in 1905, while operating for what he thought was a ruptured intestine, found a transverse tear of the pancreas, which he sutured and which was the first cure. Since then Heinecke collected 10 cases, 8 of which were cured by operation, and more recently 8 other cases have been reported, of which 4 were saved by operation and 4 died. Rosenbach reports an additional case, which recovered after operation. A woman, aged forty-seven years, was struck in the abdomen by the pole

of a wagon, and fell unconscious. On recovering consciousness, she complained of severe pain in the abdomen especially in the region of the stomach. Soon there was vomiting of bile, without blood, and for three days there was passage of flatus, but no feces. Distention of the abdomen was marked, especially in the epigastrium. The recti were rigid and the whole abdomen very tender. Above the umbilicus was detected a feeling of resistance not well defined. The whole clinical picture suggested an intra-abdominal injury, probably of the stomach. On opening the abdomen an encapsulated mass, about the size of an apple, was found below the liver and above the stomach. After separating some adhesions a brownish red, thin fluid escaped. On raising the omentum and transverse colon there was seen near the spine, in the mesocolon, a mass of yellowish gray necrotic material. Many areas of fat necrosis were seen in the omentum and mesentery. An incision was made through the gastrohepatic omentum, that is, above the stomach, into the above-mentioned encapsulated mass. A quantity of brownish red old blood, but no fresh blood escaped. No tear in the pancreas was detected by the finger, and an exact inspection was impossible. The wound was tamponed and drained. Frequent examination of the urine showed no sugar. From the wound there was an abundant discharge, which upon examination was recognized as pancreatic secretion. It required nearly four months for the fistula to close.

Traumatic Asphyxia.—Despard (*Annals of Surgery*, 1909, xlix, 751) says that the term traumatic asphyxia has been applied to the series of phenomena following the suspension of the respiratory function for a more or less prolonged period by forcible compression of the thorax or abdomen, or both. These consist of the usual symptoms of suffocation, associated with a peculiar bluish mottling of the skin of the face and neck, sometimes extending to the upper part of the thorax, and occasionally to one or both arms, accompanied by subconjunctival hemorrhages, and frequently bleeding from ears, nose, and pharynx. He reports the case of a man who was compressed by a moving elevator for about five minutes. The line of compression extended from the left side of the base of the neck to the right lumbar region. When found his face was said to have been almost black, eyes bulging, mouth open, and tongue protruding, and he was completely unconscious. Examination later showed both arms to be apparently paralyzed. Over the forehead, face, and neck, and extending to the right shoulder anteriorly and posteriorly, were what seemed to be small ecchymotic spots, dark red in color, which did not disappear on pressure and were separated by normal skin. Gradually the patient recovered, so that in about three weeks after the accident he had so far recovered as to be permitted to leave the hospital, to return to the out-patient department for electrical treatment. Six weeks later he had fairly recovered the use of the right arm, but there was still some muscular weakness. In this form of suffocation not only is the air prevented from entering the lungs by their inability to expand, but the contents of the thoracic vessels may be forced out and, in the case of the veins, the current is reversed. The valves are then overcome and the blood is dammed back into the capillaries. If the force acting is sufficiently great, it is conceivable that the capillary vessels would be dilated to a point where paresis would ensue.

The Technique of Implanting the Ureter into the Intestine.—Ssimzemsky (*Zentralbl. f. Chir.*, 1909, xxxvi, 706) says that experimental attempts to implant the ureter into the large intestine have given unfavorable results. Mirotworzew reported that the implantation on both sides in dogs was fatal in all his cases, and when only one ureter was implanted more than half of the dogs died. Of 28 patients operated on by Tichow, 9 died; and both patients operated on by Diakanow also died. Yet the indications for operation are so pressing that undoubtedly these cases will continue to be operated on in the future. Therefore, perfection of the technique should be sought. The usual causes of death are ascending pyelonephritis and urinary peritonitis. Ssimzemsky offers the following technique: The ureter is divided at a convenient situation on the posterior wall of the small pelvis, and without isolating it from the surrounding tissue, it is taken up with small flaps of the overlying peritoneum and the attached connective tissue to the extent of about 6 or 7 cm. Then a suitable forceps is introduced into the rectum and the wall of the intestine at the site of the anastomosis is made to protrude on the points of the forceps. A small incision is made over this prominence, the forceps is protruded, the divided end of the ureter seized and drawn into the bowel about 1 to 1.5 cm. The flaps of peritoneum covering the ureter are then applied to the serous surface of the intestine around the seat of anastomosis. Two stitches fix the ureter in the small intestinal wound and grasp the serous and muscular coat of the ureter and surrounding cellular tissue and the serous coat of the ureter. The peritoneal flaps of the ureter are then sutured to the serous surface of the intestine. After such an anastomosis on the cadaver, the kidney was isolated, the pelvis of the ureter opened, and water under considerable pressure forced through the ureter into the intestine. After the water had somewhat distended the intestine it escaped through a tube introduced into the rectum. None escaped into the abdominal cavity.

The Operative Treatment of Old Empyema.—Goldmann (*Zentralbl. f. Chir.*, 1909, xxxvi, 785) calls attention to the fact that the Schede operation requires a free exposure of the whole cavity of the empyema, and above all the removal of all bony covering. The Delorme decortication of the lung fails in many cases, and in some the removal of the thickened pleura must be combined with the classic Schede resection, in order to obliterate the cavity. He concludes that the ideal healing will be promoted the more rapidly: (1) The more completely the overlying chest wall is resected and the thickened pleura is extirpated; (2) the more thoroughly are our efforts made to cause the decorticated lung to expand; and (3) the more quickly we bring about the permanent unfolding of the lung by causing a prmiary adhesion of the lung to the overlying soft parts. He reports the case of a young woman, who had a pneumonic empyema June 2, 1906. On June 28 a rib resection (2 ribs) was done. The wound was healed November 9, but later broke open again. April 11, 1907, another resection was done (7 cm. of one rib) on account of the persisting fistula. Later a Schede operation was performed and forced expiratory gymnastics carried out, without success. After determining, by probing, the presence of a cavity about the size of an apple, Goldmann, on May 6, 1908, freely exposed this cavity, removing the sixth rib. He then removed the lining of the chest wall and the lung pleura.

The wound was contracted by sutures until there was left only a small drainage track. August 10 the fistula was reopened and the lining wall of the now smaller cavity was again removed. This was repeated November 30, and the lung was now drawn out by a forceps and fixed by sutures to the margins of the cavity. A flap was made from the overlying skin and soft tissues with its base toward the spine and drawn over the presenting lung tissue. For a few days there was high fever. Six days after the operation there was evacuated from the upper angle of the wound a large quantity of almost clear exudate, free of bacteria. Then the temperautre fell and the wound closed firmly. Over the whole area of the old cavity, vesicular breathing could be heard. There was a good curve to the thorax, with no sinking in of the skin and no scoliosis.

The Utilization of the Coagulation Time of the Blood in Surgery.—Denk and Hellmann (*Mitt. a. d. Grenzgeb. d. Med. u. Chir.*, 1909, xx, 218) say that the Wright method of determining the coagulation time of the blood, with careful attention to sources of error, is the most reliable method of determining comparatively slight deviations from the normal, which may be regarded as pathological. The practical application of this examination is of great value to surgeons in detecting hemophilia, even of the mildest grades. Operations on bleeders should be undertaken only after prophylactic treatment with calcium salts or serum injections, when the effects of these substances are to be controlled by the determination ot the coagulation time of blood. Operations that are not urgent should be declined in severe cases of hemophilia, in which the clotting time is higher than normal in spite of preliminary treatment. In the after-treatment of operations, in which the coagulation time is normal or accelerated, milk and all other foods which hasten coagulation should be avoided. In such cases citric acid should be given in large doses, on account of the possible relationship between rapid coagulation and emboli formation. Milk should be replaced by foods lacking in calcium and, therefore, having no effect on the time of clotting.

Thyroid Gland and Vascular Surgery.—Carrell (*Surg., Gynec., and Obst.*, 1909, viii, 606) says that among the operations able to modify the functions of the thyroid, the ligature of the arteries alone has been used in clinical surgery for the treatment of exophthalmic goitre. Other diseases of the thyroid, especially the "formes frustes" of hyperthyroidism and hypothyroidism may be improved by surgical modifications of the circulation of the gland. It is very logical to suppose that we can stimulate or diminish the activity of a gland by increasing or diminishing its circulation, and perhaps also modify its pathological reactions. But it is premature to think at present of clinical applications along these lines. The study of the question is still at its beginning and a great many of more precise experiments have still to be done. The treatment of hypothyroidism and athyroidism by the transplantation of the thyroid is more immediately promising of practical results. The feasibility of the operation has been demonstrated. The actual problem is to render it ordinarily successful. Success in transplantation depends on two factors, biological, and surgical. We are still ignorant of the biological factors of success and failure in transplantation of tissue between animals

of the same species. We do know exactly what are the interactions of the organism and its new organ. It is certain that, at least, in some cases, the gland adapts itself to its new owner, does not undergo any marked histological changes, and functionates normally. But we do not know how frequent are the cases in which the new tissue and its host will have a pernicious influence on each other. The surgical factors can be probably controlled. It must be hoped that the technique of the graft by simple implantation will be improved. The operation is very much simpler than the transplantation with anastomoses of the vessels. However, if it is found that it cannot frequently be successful the transplantation of large pieces of thyroid with union of their vessels to the vessels of the patient must be attempted. In this indirect manner vascular surgery may improve the results of the treatment of hypothyroidism and athyroidism.

THERAPEUTICS.

UNDER THE CHARGE OF

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The Physical Treatment of Bronchial Asthma.—Strasser (*Monats. f. d. physikal.-diät. Heilmethod*, 1909, i) has made use of hydrotherapeutic measures for the treatment of asthma for sixteen years. These measures include general douches, local douches applied to the legs, simple and Scotch douches to the epigastrium, etc. Strasser has had good results from their use even in severe cases complicated by bronchitis. They all diminish the reflex nervous irritability, which seems to play such an important role in the etiology of asthma. Von Strümpell has advocated the use of electric light baths for the treatment of asthma, and Strasser confirms his view regarding their beneficial action. Von Strümpell has his patients remain in bed for two hours after the electric light applications. Strasser prefers the use of some of the milder hydrotherapeutic measures following the electric light baths. He does not think it advisable to allow the patients to remain for two hours bathed in perspiration. In mild cases, Strasser follows electric light bath of ten minutes' duration with a douche or light rubbing down and very light exercise. In severer cases the electric light baths are made longer in duration and the hydrotherapeutic measures following are milder. The patients are then made to rest in bed for one or two hours. Strasser regulates the length of the baths by the subjective condition of the patient. As a rule, the initial bath is not more than six or eight minutes in duration, and, subsequently, never more than fifteen minutes. Daily baths are sometimes exhausting, and if so, they should be given every other day, or on two days out of each three. On the intervening days the treatment is intermitted or general mildly tonic hydrotherapy is given. Von Strüm-

pell does not recommend the baths at the height of the attacks, but Strasser has found them very useful even in acute attacks. Strasser agrees with v. Strümpell that probably the most important action of the baths is the diaphoresis induced by them. He thinks it also may be true that the electric lights have a specific action upon the bronchial mucous membrane, increasing the secretion from the glands and epithelium. By virtue of these actions they may aid in the elimination of the toxic substances which are the inciting agents of asthma. Strasser also suggests that they may have a direct antispasmodic action upon the skin and internal organs. The effect of heat upon various hysterical spasmodic affections is well known, and Strasser bases his theory upon this premise.

General Principles in the Treatment of Diseases of the Heart.— Barr (*Brit. Med. Jour.*, 1909, i, 989) introduces his paper by discussing the preventative treatment of cardiac disease occurring as a result of the acute infections, particularly rheumatism, or as a result of the degenerative lesions of the heart and bloodvessels so common in later life. He deprecates the tendency for the indiscriminate use of cardiac tonics in heart disease. He says that many of the cardiac tonics cause an increase of arterial pressure which, if long maintained, leads to irreparable damage to the aorta. He believes that measures which lessen the work of the heart are of much greater value than cardiac tonics. When the balance between the two sides of the heart is well maintained drugs are usually unnecessary. Among the measures which lighten the work of the heart he mentions rest, graduated exercise, reduction of intake of fluids, reduction of amount of food, sweating, diuresis, use of cathartics, and various hydrotherapeutic measures. With regard to exercise, Barr does not prefer any routine system, but believes that the exercise must be fitted to the individual. Exercise increases oxidation and hastens the elimination of waste products that are the main causes of arterial degeneration. Properly graduated exercise increases the tone of the cardiac muscle and at the same time increases the general muscular capacity. If the blood pressure is high, Barr prefers the use of decalcifying agents and mild cathartics over long periods of time in order to maintain the pressure at a constant lower level. If there is any tendency to thrombosis or embolism, he believes that decalcifying agents, such as phosphoric and citric acids and their soluble salts, should be used. In cases in which there are urgent symptoms indicating cardiac thrombosis or pulmonary embolism, strong ammonia should be freely used. Barr gives a mixture of five minims each of liquor ammoniæ fortior and spiritus chloroformi in a wineglassful of barley water every half-hour or hour for this purpose. He states that a free hemoptysis in a case of mitral stenosis is often the best form of bloodletting to relieve the distended right heart, and, therefore, it should not be checked too quickly. He condemns the use of hemostatics and advocates the use of nitroglycerin and atropine. In all of the spasmodic affections associated with a high arterial tension, such as angina pectoris, the best remedies are nitroglycerin, morphine, atropine, amyl nitrite, and the iodide preparations. Barr says that, so far as we know, the presence of free calcium ions in the blood and tissues is necessary for effective muscular contractions. He cites Bell's work, who showed that when a solution of calcium chloride was injected into the veins of a rabbit, the rhythmical

contractions continued and their amplitude increased while their frequency diminished. Barr believes that a renewed supply of calcium ions is indicated in febrile conditions because of the increase of their excretion in such conditions. An inordinate supply of calcium ions increases the viscosity of the blood, raises the blood pressure, hastens the formation of scar tissue, and leads to degenerative changes in the heart and bloodvessels. When the supply of calcium ions in the blood is too great, Barr states that the cardiac contractions tend to become slow though still effective. Because of the greater muscularity of the left ventricle, there then occurs an asynchronous action of the right side of the heart with consequent irregularities and intermissions. Therefore, in such conditions as Stokes-Adams disease Barr advises an elimination of the lime salts and the use of decalcifying agents.

Salt-poor Diet as a Therapeutic Measure.—Mendel (*Münch. med. Woch.*, 1909, ix, 433, and x, 516) believes that the good effects of many of the dietetic methods for the treatment of circulatory disturbances and obesity are to be ascribed simply to the low sodium chloride content of the diets used. Thus, he explains the beneficial effects of the Karell milk cure with its various modifications. Mendel has obtained the same results by the use of a mixed diet low in sodium chloride content. He reviews some of the recent experimental work and clinical observations of the results obtained by the use of salt-poor diet in various affections. He mentions particularly Finklestein's results in the treatment of eczema of children by such a diet. Mendel says that even normal amounts of salt in the diet of healthy individuals cause a retention of blood in the body of from 1.5 to 3 liters. An excessive intake of salt may cause a marked retention of water even in the healthy individual with sound kidneys. However, in normal individuals, there is no transudation or exudation of fluid. Mendel cites various animal experiments performed by Cohnheim and Lichtheim, who found that normal animals could eliminate large quantities of salt solution introduced intravenously without the production of any œdema. If, however, they produced an artificial inflammation, a marked œdema always occurred in the neighborhood of the inflammation. Consequently, there must be some abnormal condition of blood or lymph vessels before œdema can be produced. They were also able to produce œdema in case they prevented the venous return by a ligature. Mendel believes that retention of salt with consequent retention of water not only alters the clinical picture of inflammation, but has an injurious action on its course. This effect, he believes, is more striking in various skin affections and in inflammations of serous membranes. He believes that an excess of salt increases the accumulation of fluid in pleurisy and in synovitis. These accumulations of fluid compress the lymphatics and capillaries, and thus prolong the course of the inflammation. Mendel describes some very favorable results obtained by the reduction of salt in the diet of various affections. The cases he treated in this manner comprise cases of burn, of eczema, of ascites with cirrhosis of liver, of pleurisy, of gonorrhœal epididymitis, of joint affections, of venous thrombosis, and of arteriosclerosis. He states that in parenchymatous nephritis, the most notable results can be obtained by a salt-poor diet. A salt-poor diet relieves peripheral resistance in heart affections, increasing the amount of urine, and thus reliev-

ing stasis. Mendel says that it is not necessary to exclude all salt, but to reduce the amount to from 30 to 60 grains a day. He has found that a milk diet is the simplest and easiest form of a salt-poor diet.

The Results of Drug Treatment in Five Hundred Cases of Delirium Tremens.—Ranson (*Jour. Amer. Med. Assoc.*, 1909, lvi, 1224) summarizes his conclusions with the use of drugs in the treatment of 500 cases of delirium tremens as follows: (1) In incipient cases the patients respond readily to treatment with chloral, ergot, bromides, and whiskey, the drugs being mentioned in the order of their value. (2) Delirious patients are very resistant to treatment. In the cases studied the administration of the sedative drugs increased the mortality. This was most evident when scopolamin was used, that drug increasing the mortality 13 per cent. These unsatisfactory results with the sedative drugs were due to the large doses used. It will be shown that small quantities, for example, 15 to 30 grains of chloral in twenty-four hours, may be given with good results, but that when larger doses are given the death rate increases with the amount of sedative administered. (3) The only drug which reduced the mortality was ergot. By its use the death rate was reduced 21.6 per cent. (4) When whiskey was given the mortality was increased 1.8 per cent. The 500 cases which serve as a basis for Ranson's article were treated at the Cook County Hospital, Chicago, over a period of three years. They were divided into two classes, incipient and delirious. The incipient cases were those which showed only insomnia, restlessness, and tremor. The fully developed cases were noisy, delirious, and had well-defined hallucinations. Of the sedative drugs the bromides were most extensively used. A mixture of sodium, potassium, and ammonium bromide, in doses of from 10 to 30 grains each every four hours, was used in 198 cases with delirium. This series showed a mortality of 45.5 per cent. In 103 similar cases treated without bromides, the mortality was 40.8 per cent. In 190 incipient cases treated with bromides, only 32.6 per cent. of the patients became delirious; while of 156 similar cases not treated with bromides, 52.8 per cent. of the patients became delirious. Chloral was also extensively used and frequently was given in combination with the bromides. Ranson believes that it is of no value after active delirium has set in. In fact, it seemed to increase the mortality by 2 per cent. However, he believes that chloral is of great value in the treatment of incipient cases. It reduced the percentage of cases developing delirium in the hospital from 48.9 per cent. to 20.6 per cent. Ranson believes chloral superior to the bromides in the treatment of incipient cases because of the greater speed of its action. He found morphine of very little value, having no effect at all in delirous cases and far inferior to small repeated doses of the other sedatives in the incipient cases. Scopolamin was given in single doses of $\frac{1}{100}$ to $\frac{1}{50}$ of a grain to a series of incipient cases. Like the morphine series, this series of cases seemed to show that single doses of sedatives have no controlling action upon the incipient cases. In addition, Ranson found that scopolamin was extremely dangerous when given to actively delirious cases. He speaks particularly of the good effects of ergot in the treatment of these cases. Ergot was given in the form of the fluid extract, in dram doses, repeated every four hours, and often in combination with whiskey. In those cases in which ergot was used the mortality was only 30 per cent.,

while among those patients treated without ergot it was 51.6 per cent. Ranson says that this result is striking when we consider that usually the sedatives increase the mortality. Ranson likewise found ergot of value in the incipient cases, the percentage of patients developing delirium being reduced 23.3 per cent. by its use. He says that any explanation of its action would be pure speculation, but speaks of the possibility that it may decrease the cerebral hyperemia. Ranson also believes that whiskey is of use in the treatment of incipient cases, lowering the percentage of patients becoming delirious by 20.2 per cent. In the delirious patients, he believes it is useless.

The Pathogenesis and Therapeutics of Diarrhœa.—A. Schmidt (*Med. Klin.*, 1909, xiii, 457) reviews briefly some of the causes of diarrhœa, and mentions particularly a form with serous, foul-smelling stools. This form, he believes, is due to an abnormal secretion of an albuminous fluid which putrifies readily. He discusses briefly the treatment of the different forms of diarrhœa, classifying the remedies into the opiates, the astringents, and the intestinal antiseptics. The opiates are believed to be beneficial by reason of their antiperistaltic action, and consequently they are used in cases of diarrhœa due to increased peristaltic action. Schmidt believes they should be used only in cases of chronic diarrhœa when all other measures fail, never for acute cases. He believes that the opiates are an example of symptomatic therapy and have no relation to rational therapeutics. The use of the astringents rests upon a much firmer etiological basis, but their application is limited to certain forms of diarrhœa, *e. g.*, those showing signs of a subacute or chronic inflammation of the mucous membrane. Schmidt says that he has made many clinical observations and experiments in order to determine the most efficient intestinal antiseptic, and he believes that hydrogen dioxide is the most efficient and least harmful. He found that pure agar-agar was able to take up from 10 to 12 per cent. of hydrogen dioxide, and that it yielded it up very slowly in the form of nascent oxygen. He used this hydrogen-dioxide-agar for the treatment of diarrhœa with serous foul-smelling stools, with good results. It had no apparent effect upon diarrhœas originating in the large intestine or those due to intestinal tuberculosis. Schmidt concludes by saying that since the hydrogen-dioxide-agar is perfectly harmless, it should be more widely used, in order to determine its further uses and limitations.

PEDIATRICS.

UNDER THE CHARGE OF

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Staphylococcic Infection Treated with Killed Bacteria.—Soper (*Pediatrics*, xxi, No. 3) reports observations on the application of opsonic therapy to bacterial infections in infancy and childhood. The

majority of his cases were furunculosis in children, ranging in age from two months to seven years. In each case the staphylococcus was isolated before treatment was begun, and no medicinal treatment was used. The opsonic index was not followed. Twelve cases received on an average two inoculations each; 6 received three, and 2 cases one each. Most of the cases were cured in from one to four weeks; a few were not improved. A bacterial emulsion was prepared in the following way: a twenty-four-hour growth of pure culture on plain slant agar was washed with 3 c.c. of sterile normal salt solution and placed in a water bath at 65° C. for one hour. The number of cocci per c.c. was estimated by the Wright method of standardization, or by counting in a Thoma-Zeiss red blood counter, using a solution of methylene blue as a stain. Sterility of the emulsion was proved by inoculating with it a tube of slant agar and incubating for twenty-four hours at 37° C. If no growth appeared, it was considered sterile enough for subcutaneous inoculation. Using the same amount of salt solution and the same sized stroke on the agar produced fairly constant quantities of bacteria per c.c; 0.5 to 1 c.c. usually gives a dosage of 50,000,000 to 100,000,000. Local inflammatory reaction was slight or absent. Comparison of stock vaccines with autogenous showed little difference in behavior, autogenous vaccine curing each of 5 cases, and stock vaccine 75 per cent. of 15 cases. Usually a small dose, 30,000,000, was given at first, and later the dose was increased up to 100,000,000. In some of the older children 250,000,000 were used at first and increased to 500,000,000.

Teratoma of the Testicle.—Lorrain (*Bull. et mém. de la soc. anat. de Paris*, 1909, lxxxiv, 65) reports the case of a boy, aged fifteen years, whose left testicle had been noticed to be enlarged since he was six months old; it grew slowly until his ninth year, at which time he had measles. Since then it had grown more rapidly. At the time of the report the right testicle was of normal size, the left twice as large. At the base of the testicle was to be felt a hard mass, regular in outline, not transparent and slightly tender; the epididymis was intact. The diagnosis rested between tuberculosis and cyst. After removal of the testicle it was noticed that the testicle and the tumor were separated from each other by a constriction, the tumor mass, however, extending into the interior of the testicle. The mass contained sebaceous material, glands, and four teeth, thus showing the tumor to be a teratoma.

The Distribution of Tuberculous Lesions in Infants.—Martha Wollstein (*Archiv. Int. Med.*, April, 1909) has investigated the manner of infection and the localization of tuberculous lesions in children, by analyzing the autopsies of 185 children showing tuberculosis. These children came from unhygienic tenement districts in New York and were all under three years of age. From this series of cases it appears that of all infants under one year coming to autopsy, 12 per cent. showed tuberculous lesions; 36 per cent. of those between one and two years, and 32 per cent. of those over two years. Of infants under three months only 1.8 per cent. had tuberculosis. In these 185 cases there were 13 in which the lesions were limited to the respiratory tract, and only 1 case involving the intestines alone. The point of entrance of the bacillus

was clearly 13 times in the lungs and once in the intestinal tract. Four additional cases were of undoubted intestinal origin, 40 of respiratory origin, and one of mixed origin. In 8 cases the mesenteric lymph nodes were involved without a lesion in the intestines, but with marked pulmonary involvement. The ulcers in the intestines were most frequent and advanced in the lower ileum. In the kidneys young miliary tubercles were found in 67 cases, usually in both kindeys. The pulmonary lesions varied from discrete miliary tubercles to cheesy areas and cavity formation. The right upper lobe was involved oftener than the other lobes. This study of cases under three years shows the rarity of undoubted cases of intestinal tuberculosis, and that tuberculosis of respiratory origin predominated over that due to ingestion of the bacillus. The tendency of the tuberculous process in lymph nodes in young children is toward progressive degeneration rather than healing, as caseation and suppuration were found to be the rule in these cases.

Gonorrhœa in Little Girls.—Scheuer (*Wien. klin. Woch.*, 1909, No. 18) reports 39 cases of gonorrhœa in little girls. In 15 cases the urethra was involved. In no case was he able subsequently to determine any involvement of the pelvic peritoneum or adnexa. Treatment consisted in rest in bed and sitz baths. The vagina was washed out three times a day, with 0.5 to 2 per cent. protargol solution or with a 0.1 per cent. ichthyol preparation. In treating the urethritis he employed thin protargol bougies. The course of the disease was usually found to be chronic, and in many cases a cure was not obtained until after three months of treatment.

The Cutaneous Test for Tuberculosis.—Herrman (*Pediatrics*, 1909, xxi, No. 3) discusses the preference for the cutaneous test. He states that while a positive reaction does not necessarily show the presence of active tuberculous lesions, this test is simple, universally applicable, and free from danger. Autopsy findings prove it as trustworthy as any of the tests recently employed. It is more valuable in infants and children than in adults, and a positive reaction more significant, because there is less likelihood of a healed tuberculous lesion being present. He notes the objections to the various tests. Injections of tuberculin are painful; can be used only when there is no fever; and the temperature must be taken at short intervals. The puncture test is painful and has no advantage over the cutaneous test. The conjunctival test is not without danger, and requires a normal eye and conjunctiva. It requires a specially purified preparation of tuberculin, and in infants the tuberculin may be conveyed to the opposite eye by rubbing of the eyes. Doubtful reactions are frequent. Inunctions of tuberculin require a special preparation, and results are not in any way more reliable. Advantages of the cutaneous method are simplicity, ease of application, and freedom from danger; it requires no special preparation, crude tuberculin being used. Patients need not be reëxamined for twenty-four or forty-eight hours. Pain caused by scarification is slight. He uses the following method. The forearm is cleansed and two new sewing needles are held together and dipped into tuberculin (undiluted); a small quantity is thus held between the needles by capillary attraction. Two short scarifications are made at right angles and the tuberculin adhering to the needles is rubbed in, or the drop is placed on the skin first, and scarification made through

it. New needles are used for each patient, and a separate applicator is unnecessary. No control scarification is used. If the reaction is doubtful it is of no value. Herrman has never seen a control which could be mistaken for a positive reaction.

A Case of External Masculine Pseudohermaphrodism; Sarcoma of the Ovary.—Broca (*Bull. et mém. de la soc. de chir. de Paris*, 1909, xxxv, 294) reports the case of a boy, aged seven years, looking like a boy of ten. There was very little of feminine facies and characteristics about him. He had an abdominal tumor, which had existed for six months. During his sixth year the following surprising condition developed: the penis became quite large, the voice deepened; he developed pubic hair; and his breasts became as large as those of a girl thirteen to fourteen. He had a hypospadia; the left testicle was missing from the scrotum; on the right side a hernia was noted containing a mass resembling a testicle. The abdominal tumor resembled a uterine or ovarian fibroid, but as there was no doubt as to the sex of the patient, it was believed to be a fibroid of the mesentery. The operation revealed a solid ovarian tumor with a normal tube and an atrophic uterus. A fibrous cord represented the vagina. The uterus and left ovary were removed; the right ovary and tube were contained within the hernia, and had no uterine connection. A radical operation was performed for the hernia, the ovary and tube having been reduced into the abdominal cavity. Pathologically the tumor was found to be an endotheliofibromyxosarcoma of the ovary. The penis, as stated above, was well developed; it had a normal glans and prepuce, the urethral orifice being situated 3 cm. back of the glans. A cord passing along its under surface caused an incurving of the glans. The pubic hair was long and very dark. The scrotum was of normal appearance, excepting that instead of hanging below the penis, it surrounded it, the latter seemingly growing out of it, as is the case with the clitoris and the labia majora. There was no sign of vaginal orifice. Four years later the child was still in excellent health; there was no sign of menstrual congestion; the child was of normal stature. The pelvis and thorax presented the characteristics of a male; the breasts were much less developed in proportion than at the time of the operation—in fact, they look like those of a boy. The external genitalia were as before, and there was no sign of a recurrence of the tumor. He was at the head of his class, and was able to take part in the games of boys of his age.

Adenoids, Nocturnal Incontinence, and the Thyroid Gland.—Williams (*Lancet*, 1909, i, 1245) had the adenoids of a nine-year-old boy removed, hoping it would cure him of nocturnal incontinence. It made the boy much worse, however, and believing that the removal of the adenoids deprived the boy of a necessary internal secretion, he then gave him thyroid extract, one-half grain twice daily. The result was instantaneous and complete, the boy no further wetting the bed. Twenty-four other cases were thus treated, with but one failure. He concludes that adenoid vegetations should no longer be considered as a cause of nocturnal incontinence; in fact, Williams believes that adenoids afford some measure of protection against this weakness, and he further believes that if the two co-exist, it is due to a common cause—insufficiency of the internal secretion of the thyroid gland.

OBSTETRICS.

UNDER THE CHARGE OF
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Pain and Hemorrhage as Symptoms of Ectopic Gestation.—Eden (*Brit. Med. Jour.*, April 17, 1909) reports eight cases of ectopic gestation, and calls attention to the importance of pain and hemorrhage as symptoms. He calls attention to the fact that symptoms associated with ectopic gestation arise not directly from the presence of the growing ovum, but from secondary lesions, traumatic or inflammatory. These are bleeding from tubal abortion, or rupture, the blood passing into the peritoneal cavity, or bleeding into the Fallopian tube, causing acute distention, the abdominal end remaining closed, or slowly progressive, or recurrent hemorrhage leading to encysted collections of blood, or infection of the gravid tube, or some encysted collection of blood leading to suppuration. He calls attention to the importance in diagnosis of hemorrhage from the uterus, the discharge being dark and persistent in character, as a symptom of ectopic gestation. When free bleeding occurs into the peritoneal cavity, the symptoms are acute abdominal pain, fainting, and constitutional signs of hemorrhage, and shock attended by vomiting, gradually improving, usually occurs after such hemorrhage, which differentiates these cases from perforation of the stomach or intestine, in which the poisonous contents, escaping through the point of rupture, cause the patient to grow steadily worse. The intense abdominal pain seen in ruptured ectopic gestation is caused not by the bursting of the Fallopian tube, but by irritation of the peritoneum by extravasated blood. Recurring attacks of intense pain indicate repeated intraperitoneal hemorrhage. Attacks of acute abdominal pain may also follow tubal abortion, and Eden reports a typical case, in which, before operation, it was thought that the pain arose from the bursting of a pyosalpinx and the escape of its pus into the peritoneal cavity. Pain and nervous disturbance resembling that following intraperitoneal hemorrhage may occur when a Fallopian tube is pregnant without rupture. In a case reported by Eden, a firm, elastic swelling was found in Douglas' cul-de-sac, the patient suffering from shock and abdominal pain. Her pulse, however, was good; her temperature very little disturbed, and the cardinal signs of dangerous hemorrhage were lacking. At operation an unruptured gravid tube was found and readily removed. An encysted collection of blood or hematocele is often found in those cases in which the patient has irregular uterine hemorrhage, with pain in the abdomen or pelvis, this phenomenon recurring at irregular intervals over a considerable period of time; in the writer's case a peritubal hematocele surrounding the left tube was present. In these cases it is not sufficient to put the patient at absolute rest to prevent progressive hemorrhage and the enlargement of the hematocele; operation only will permanently check slow and insidious bleeding. In some cases of pregnancy in a bicornate uterus,

where the ovum lodges in one side of the womb, it develops to a considerable extent and may rupture without hemorrhage. The writer describes a five months' pregnancy in the left cornu of the uterus, where rupture occurred and partial extrusion of the fetus and retention of the placenta. Very little free blood was found in the peritoneal cavity. Eden calls attention to the fact that the symptoms of ectopic gestation may be perfectly simulated by other conditions. He describes the case of a patient with disturbed menstruation, bleeding, discharge of clots, pain, and recurrences; a soft, elastic swelling was found in Douglas' pouch, extending into the left fornix. The case seemed typical of ectopic gestation. At operation the swelling turned out to be a septic ovary, composed of a corpus luteum cyst, into which hemorrhage had occurred. The left tube was inflamed and contained pus; the right tube inflamed without suppuration.

In a paper entitled "Three Recent Cases of Tubal Pregnancy," Purslow (*Brit. Med. Jour.*, April 17, 1909) emphasizes the fact that in more than 50 per cent. of cases amenorrhœa does not precede the development of ectopic gestation. He considers the cardinal symptoms of this condition to be irregular and unusual vaginal hemorrhage, intermittent attacks of pain in the lower abdomen, or the presence of a tender swelling in the pelvis.

Adrenalin in the Treatment of Osteomalacia.—Engländer (*Zentralbl. f. Gyn.*, 1909, No. 13) reports 6 cases of osteomalacia treated by the injection of adrenalin. Solution from 1 to 1000 was employed; injections were made daily, in the forenoon under strict antiseptic precautions, in the subcutaneous tissue of the abdominal wall. The temperature and pulse of the patient and her blood tension were observed and recorded daily. The doses varied from 0.5 to 1 c.c. Although the injections were somewhat painful, no local reaction followed. Some of the patients showed decided reaction in the redness and injection of the face, with altered conditions in the pupil. Most of the patients received 40 or more injections. Considerable improvement followed this treatment, the symptoms becoming less pronounced, and the patient being better able to do her work.

The Treatment of Placenta Prævia.—Krönig (*Zentralbl. f. Gyn.*, 1909, No. 15) reports twenty cases of placenta prævia treated by combined and internal version, and contrasts the results obtained by this method with those following the treatment of placenta prævia by the use of a dilating bag. The significant feature in these cases is the fact that, without exception, version followed by bring down the body of the child stopped hemorrhage. This control of bleeding pertained during labor only, while the child was in the process of extraction, its body remaining within the genital canal of the mother. After the expulsion of the child, in most cases, hemorrhage of varying degree of severity occurred. Créde's method in many cases was inefficient in producing the discharge of the placenta, and it was necessary to empty the uterus by the introduction of the hand. The situation of the patient was critical for an hour or two after the delivery of the placenta. This postpartum bleeding is most severe in cases farthest advanced in pregnancy.

While these cases naturally develop frequently among patients in

their own homes, if the patient is carefully watched there is always some warning before severe hemorrhage begins. In 34 cases carefully investigated, there was preliminary hemorrhage sufficiently severe to attract attention in all of them. Such warning should lead to the patient's transfer to a hospital as soon as possible. When cases are seen in the very early stages of labor in an aseptic condition, and mother and child are in good condition, Cesarean section in a hospital will undoubtedly give the best result. The prognosis of placenta prævia is becoming very much better, the maternal mortality dropping to 6 per cent., and lower in cases promptly treated.

Spontaneous Rupture of the Umbilical Cord during Labor.—Unterberger (*Zentralbl. f. Gyn.*, 1909, No. 14) reports two cases of spontaneous rupture of the umbilical cord during labor. In the first case the mother was a multipara, having a somewhat contracted pelvis. Labor pains were especially strong, the child being spontaneously expelled in the second position. The cord was about the neck, and the shoulders and body of the child were rapidly forced out after the birth of the head. The umbilical cord ruptured 22 cm. from the child's umbilicus, and the ends of the cord were immediately tied. The placenta was delivered spontaneously a little afterward. There was no hemorrhage from the uterus or from the cord. There was no laceration. The child was vigorous; the placenta contained many earthy deposits and a small infarct. There was no sign of syphilis. The cord was 47 cm. long. The second case was that of a primipara with slightly contracted pelvis, the child being expelled spontaneously with strong labor pains. The cord was also about the neck, and an effort to dislodge it was unsuccessful. The shoulders were born by a strong uterine contraction, and the cord ruptured 10 cm. from the child's umbilicus. The amnion sheath of the cord tore first, then both arteries, and finally the vein, the rupture of the cord permitting the expulsion of the shoulders. The tear across the cord was oblique with jagged edges. The ends were immediately tied, and hemorrhage did not occur. The cord was 50 cm. long, spiral, and with a great quantity of Wharton's jelly.

The Expulsion of the Uterine Decidua in Tubal Pregnancy.—Buché (*Zentralbl. f. Gyn.*, 1909, No. 13) reports the case of a multipara who had complained for four weeks of pain in the back and right lower abdomen, and had had four attacks of sharp pain in this vicinity. On examination a tumor the size of a goose-egg could be detected at the right side of the uterus. Douglas' cul-de-sac was filled by the tumor. At operation tubal pregnancy on the right side was found, with beginning hematocele surrounding the tube. On separating the tube the ovum was expelled into the abdominal cavity and removed practically entire. The uterine decidua was expelled in a complete cast of the uterus on the second day after operation. The shape of the sac was three-cornered, with prolongations which had evidently extended into the Fallopian tube. There were three openings to the sac, one to the cervix, the others at each tubal extremity. The lateral portions of the sac were thin and anemic, the middle more red and richly supplied with blood. Buché distinguishes three different ways in which the uterine

decidua is usually expelled: First, when the portions of the decidua in the tubes separate with difficulty, the mass is apt to come away entire. If this not the case, the decidua may be anteverted, the portion from the fundus being delivered first; or it may be delivered in situ, when the fundal decidua and that in the right uterine segment are loosened at the same time.

GYNECOLOGY.

UNDER THE CHARGE OF

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Primary Carcinoma of the Female Urethra.—L. S. McMurtry (*Trans. Amer. Surg. Assoc.*, 1908) reports two cases of primary carcinoma of the female urethra. He refers to the paucity of literature on the subject, and states that Percy's investigation of the 28 cases collected by Wassermann shows that but 3 of them were undoubtedly cancer developing primarily in the urethra. But Percy tabulated 16 genuine cases, including his one, that have been reported. The first was by Madame Boivin, in 1828, and the last in 1902. McMurtry adds 10 new ones, including his 2, that have since occurred. Caruncle is so similar to the early stage of cancer of the urethra that they are confounded. McMurtry advises very early excision.

Cyst of the Vagina.—Abadie and Range (*Ann. de gyn. et d'obst.*, 1909, xxxvi, 71) report two cases of cyst of the vagina, and hold to the opinion that such cysts are developed from the Wolffian ducts. In the first case the patient was thirty-eight years of age, and the cyst, the size of a walnut, was located in the roof of the vagina on the under surface of the right broad ligament. It was successfully removed by one operation. The second specimen was removed from a multipara aged twenty-two years. The tumor was in the right vaginal wall and developed into the labium majus, and had attained a size about equal to that of the first case. The microscopic findings in these two cases furnish a basis for argument favorable to the congenital origin of these cysts—that is, their origin from the ducts of Gaertner.

Posterior Sagittal Incision of the Cervix Uteri.—L. Herzl (*Zeit. f. Geb. u. Gyn.*, 1908, lxii, 465) describes his technique for posterior sagittal incision of the cervix uteri and recommends this operation in cases of stenosis of the cervical canal when the latter causes dysmenorrhœa and sterility, and particularly in acute anteflexions of the uterus. The operation is contra-indicated in any case in which disease of the uterus and surrounding tissues exists which may in itself serve as a causal factor in the production of dysmenorrhœa and sterility. This operation was employed at the Vienna General Polyclinic only after all conservative measures had been tried and had failed. Twenty-seven cases were re-

examined. Complete cure had been obtained in 16, partial cure in 8, and total failure in 2. Herzl draws in addition to his own on Lott's private practice, and reports cure of sterility after this operation in 6 out of 40 cases operated upon.

A New Technique in Perineorrhaphy.—Charles Jewett (*Long Island Med. Jour.*, August, 1908) modifies G. R. Holden's perineorrhaphy, employing it in nearly all forms of incomplete lacerations of the perineum. The modification consists of a few figure-of-eight interrupted sutures placed in the vaginal wall, including the levator ani on both sides, and a few passed deeply through the skin near the vertical wound. Chromicized catgut is used for sutures.

The Treatment of Gonorrhœa in the Female.—Samuels (*New York Med. Jour.*, March 20, 1909) says the important points in the treatment of gonorrhœa in women are cleanliness, intra-urethral injections, and constant observation of details. No active treatment during the early stages of the disease is advised. After the acute stage has passed, daily intra-urethral injections of 1 per cent. solution of protargol are to be used and the strength of the solution gradually increased. When the gonococci have disappeared, this remedy is stopped and a mild solution of sulphate of zinc substituted. In the chronic cases Samuels advises local applications of nitrate of silver, 20 grains to the ounce. His conclusions are that: (1) All cases with a history of burning and scalding urination should be thoroughly examined for an existing urethritis. (2) If a urethritis is found, presume it is of a gonorrhœal origin if no foreign body is present. (3) Institute a thorough treatment in all cases, for if a cure cannot be effected complications may be prevented. (4) Employ the vaginal douche only after all traces of the primary infection have disappeared and never in the early stages.

Extensive Lacerations of the Cervix Uteri, Involving the Parametrium: Their Significance and Treatment.—George Tucker Harrison (*Virginia Med. Semi-Monthly*, 1909, xiv, 30) regards lacerations that include the cervix uteri and parametrium, extending in many instances through the peritoneum, as of great importance, primarily from the danger of infection of deep extraperitoneal structures and peritoneum, and secondarily from the troublesome cicatrices resulting. In the production of these extensive lesions Harrison believes the Bossi dilator will in the future play an important role. In the treatment of the severe hemorrhage that often attends such severe laceration Harrison recommends the double compression plan of Fritsch, the details of which are: The placenta is, first of all, removed; this is easily done, as the inner os uteri is either wide or lacerated. The uterus now contracts readily. The physician then, with the right hand, presses the anteflexed uterus as deep as possible into the pelvis, and in that way all coagula are forced out. The operator, standing on the left side of the bed, seizes the vulva with the left hand, pressing the *labia majora* together, and forces them above into the angle of the arch of the pubes, as if he would shove the entire pelvic floor into the pelvic cavity above. Now, with the hand above coöperating with the hand below, the parts are forcibly compressed. The lower arm is not so readily paralyzed, as its elbow rests

on the bed; when the upper arm is paralyzed, an assistant is instructed to press on the upper hand with his or her two hands—to lean on it, as it were. It matters not whether the laceration is on the one side or the other, pressure is made directly downward. The hands are held in this way for half an hour, three-quarters, or even longer. A bag of sand or shot may substitute the upper hand. Harrison claims for it the following advantages: (1) The entire treatment is extravaginal; danger of infection by the procedure is consequently excluded. (2) The blood also, which would be certainly lost with suture or tampon, remains in the body. (3) The hemorrhage is controlled in the most rational manner from the fact that the bleeding surfaces are pressed together so firmly that there is no longer a space in which bleeding could take place. Provision is consequently made for adhesion and healing from the fact that the raw surfaces are on and into each other. (4) No after-treatment is necessary; when the hands are released, if no bleeding takes place, further manipulation is superfluous.

The Best Fixation Method for the Treatment of Fixed Retroversion with Flexion.—Kuestner (*Zentralbl. f. Gyn.*, 1909, xxxiii, 41) states that reëxamination of the material at his clinic by Hannes showed that the Alexander-Adams operation gave most excellent results in those cases only in which the uterus was found freely movable. The Alexander-Adams operation should not be undertaken when any doubt of mobility of the uterus exists. Kuestner recommends the combination of the Alexander-Adams operation with laparotomy when adhesions are present. The former may be done unilaterally if desired. In cases in which careful examination fails to establish positively the presence of adhesions a suprasymphyseal transverse incision through the skin between the two external inguinal rings down to the fascia is made. The fascia is then divided longitudinally within the linea alba and the peritoneum opened. If no adhesions are found on exploratory incision of this kind one is justified in concluding the operation by the Alexander-Adams method.

Suprapubic Hysterotomy as a Means of Diagnosis and Treatment of the Uterus.—W. W. Russell (*Ann. Gyn. and Ped.*, 1909, xxii, 90) advocates suprapubic hysterotomy for the following conditions: (1) It is of conspicuous service in cases of persistent uterine hemorrhage which is not controlled by any form of treatment and in which bimanual palpation of the pelvic organs has given an absolutely negative diagnosis. (2) In polypoid change of the mucosa in which frequent curettage has failed to stop the hemorrhage. (3) Small, submucous and pediculated myomas may be removed by this operation. (4) In cases in which the abdomen has been opened for some other purpose, and no explanation has been found. Russell reports 32 successful cases in which this procedure has been used by six surgeons. The technique consists of bringing the uterus out of the peritoneal cavity and, supported by gauze packed about it, a longitudinal incision into the uterus is made in the median line on the anterior wall half way down to the cervix, as well as through the fundus to the posterior surface. The entire uterus is split for a distance of two or more centimeters down into the cavity. In closing the uterine wall catgut or fine silk is placed through it about 0.5 cm. apart and a fine catgut closes the peritoneum.

DERMATOLOGY.

UNDER THE CHARGE OF
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Lichen Ruber Planus as a Family Affection.—Veiel (*Archiv f. Dermat. und Syph.*, 1909, xciii, Heft 3) has observed a family of four members in which three, the father and two children, suffered from typical lichen ruber planus, the father and one daughter several times. Veiel concludes from this observation and a number of similar ones in literature that there are without doubt families in which a certain hereditary predisposition to lichen ruber planus may be demonstrated, the term "disposition" being here employed in its widest sense.

Septicemia and Chronic Pemphigus Due to Bacillus Pyocyaneus.—Petges and Bichelonne (*Ann. de dermat. et de syphil.*, 1908, No. 7) report a striking example of chronic pemphigus associated with septicemia due to infection by Bacillus pyocyaneus, it being the first case reported, according to the authors, in which this organism has been found in the blood of patients with this affection. The patient was a man, aged twenty-two years, who, during a period of nine months, suffered from a bullous eruption presenting all the features of pemphigus which terminated fatally with symptoms of septicemia. At various times the urine presented a bluish-green hue; and from abscesses which formed on the scalp, the thigh, and in the axilla blue pus was evacuated. Cultures made from the blood antemortem and postmortem revealed the presence of Bacillus pyocyaneus. Petges and Bichelonne conclude that Bacillus pyocyaneus may be pathogenic for man, producing general diseases with or without cutaneous lesions, and diseases of the skin of apparently external origin. It may play a role in the production of chronic bullous pemphigus and pemphigus vegetans. The case which they reported they regard as incontestably caused by pyocyanic septicemia.

Biological Investigations in Pemphigus Vulgaris.—Bruck (*Archiv f. Dermat. und Syph.*, 1909, Band xciii, Heft 3) who has recently employed biological methods in the study of two cases of pemphigus, having in mind the possibility of the presence of antigen in the fluid of the pemphigus blebs together with an antibody in the blood, tested this fluid with blood serum, but the results were completely negative. He found, however, that the fluid from the blebs of one of the patients contained a lysin which acted fairly strongly with human blood. Although the fluid was found sterile it contained a strong streptococcic virus. Inoculation of this sterile fluid into the skin of a pemphigus patient who had been free from lesions for fourteen days, produced after

some hours a hazelnut-sized bleb filled with a clear serous fluid, and in the course of the same and the next day a moderate number of blebs appeared on various parts of the body, which disappeared entirely in the next three days. A repetition of this experiment five days later was followed by a similar result. Bruck finds from this study that bacterial toxins may be demonstrated in the fluid of the bullæ present in pemphigus patients. He suggests the probable usefulness of antistreptococcic serum in the treatment of such cases as are accompanied by streptococcic infection, and thinks it might be worth while to attempt to influence the thermolabile toxins deposited in the skin through hot-air treatment.

Crude Coal Tar in the Treatment of Eczema.—Brocq (*Bull. de la soc. Francaise de dermat. et de syphil.*, 1908, No. 9) reports a number of patients with eczema who had been successfully treated with crude coal tar, which he regards as superior as a siccative and antipruritic to all other topical applications. He employs this agent in the following manner: The surface to be treated is first thoroughly freed from crusts and scales and then carefully washed with boiled water, and if not too sensitive it is afterward soaped and wiped off with ether. The tar is then applied in a thick layer and allowed to dry for a considerable time, the longer the better, the drying being an important part of the technique. When it has dried as long as possible, not less than twenty minutes, several hours if possible, it is powdered with talc and enveloped in a soft cloth. If the skin is not too much inflamed nor the oozing too abundant, it is well not to touch the dressing for two days. If the inflammation and oozing are marked it is useful to dress the parts with a simple zinc paste the next day. After five to six days the application of the tar may be repeated. Usually three to five applications are sufficient for a cure. Exceptionally it may excite inflammation, but Brocq finds that it is better tolerated than almost any other local remedy.

The Diagnostic and Specific Significance of the von Pirquet Skin Reaction.—Meirowsky (*Archiv f. Derm. u. Syphilis*, 1909, xciv, Heft 2 und 3) finds that a large percentage of those who present none of the clinical signs of tuberculosis give a positive cutaneous reaction with undiluted A. T. A positive result is also obtained when the subject has had a tuberculosis and any dermatosis; in such cases the cutaneous reaction is to be referred to the preceding tuberculous infection and not to the skin affection. Of 105 cases of tuberculosis of the skin, 102 gave a positive cutaneous reaction with undiluted A. T. Of the three which showed no reaction, two had been given simultaneously a subcutaneous injection of tuberculin; and after the resultant reaction had subsided both of these gave a positive cutaneous reaction. In only one case of lupus was the cutaneous reaction permanently negative. Meirowsky concludes that almost all cases of tuberculosis of the skin give a positive cutaneous reaction; and he believes that in a patient with a doubtful dermatosis a negative result makes it highly probable that the affection is non-tuberculous. He also thinks that the fact that the cutaneous reaction runs parallel with the reaction of the organism after injection of old tuberculin, proves the specificity of this reaction.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF
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The Influence upon Intestinal Absorption of Exclusion of the Pancreatic Juice from the Intestines.—After discussing the results obtained by various observers upon the fat and nitrogen absorption by ligating the pancreatic duct in dogs, NIEMANN (*Ztschr. f. exp. Path. und Therap.*, 1909, xv, 466) describes two similar experiments which he performed. The main pancreatic duct together with the accessory duct was ligated, the dogs were allowed to recover, and later feeding experiments made. In both cases it was found that from 61.5 to 93.9 per cent. of the nitrogen of the food was absorbed, and from 91.2 to 98.7 per cent. of the fat. This seemed conclusive evidence that complete occlusion of the pancreatic ducts is compatible with excellent resorption of fat and proteid from the intestines. The effect on the pancreas of this operation is, however, very marked. When the dogs were killed, one or two months after operation, almost no normal pancreatic tissue was to be found. There occurs shortly after ligation of the ducts necrosis, first, of the central portion of the gland, and later, of the periphery. As the process extends toward the periphery the necrotic tissue is replaced by connective tissue.

Sclerosis of the Gastric Arteries.—The study of cases of arteriosclerosis in which the arteries supplying the stomach and intestines have been especially involved has led to the recognition of a more or less definite symptom complex spoken of as "Angina Abdominis." Perutz and Cheinisse have recently discussed this interesting question and have reviewed the literature extensively. The cases may be divided into three groups: (1) Those in which there are subjective and objective symptoms, pain and constipation during life, accompanied at autopsy by sclerosis of the abdominal aorta with thrombosis or stenosis of the opening of the cœliac axis or mesenteric arteries; (2) cases in which the same symptoms occur, with no other alterations than extensive sclerosis of the main branches or even finer twigs of the gastric and mesenteric arteries, and (3) those cases in which the disease is localized and manifests itself by ulceration of the gastro-intestinal mucous membrane or by hemorrhages. BUDAY (*Ziegler's Beit.*, 1908, xliv, 327) reports a case belonging to this third group. A man, aged forty-six years, had had, during the space of three years, several gastric hemorrhages. The last hemorrhage occurred five to six days before his illness, which was due apparently to collapse consequent upon the severe anemia. At autopsy old blood was found in the lower part of

the intestinal tract, but none was seen either in the duodenum or stomach. A careful examination of the entire gastro-intestinal tract failed to reveal any point of erosion or ulceration from which the bleeding might have arisen. There were, further, no changes in any of the other organs which could in any way explain the gastric hemorrhage. When, however, the gastric mucosa was searched again for possible bleeding points, it was found that the arteries in the wall of the stomach were the seat of an extreme grade of sclerosis, and in places groups of minute tortuous arteries could be felt as hard knots beneath the mucosa. Though there was a slight general sclerosis, there was in no organ, available for microscopic study, a sclerosis to compare with that of the gastric vessels. The arteries supplying the intestines were only moderately affected. Microscopic study of the gastric mucosa confirmed the findings at autopsy. The small arteries lying in the submucosa showed extensive alterations. There was great degeneration, sometimes calcification, and always thinning of the media with irregular dilatation of the vessel and thickening of the intima. The lumen of the vessels was rarely narrowed and never occluded. More often there was an aneurysmal dilatation. In places these diffuse aneurysms lay directly beneath the mucosa, and often the mucosa was much thinned above them. Buday concludes that the severe gastric hemorrhages in this case were due to rupture of these sclerotic vessels. In no place could an actual rupture be demonstrated, but since the last hemorrhage occurred over two weeks before death, and as there was no fresh blood in the stomach, it is fair to suppose that if a rupture had taken place it would have healed. Though Gallord, Sachs, and Hirschfeld have reported gastric hemorrhages from erosions of small arteries, very little attention has been called to this origin of the so-called idiopathic hemorrhages from the stomach, and Buday believes that a careful study of the arteries in such instances will lead to the finding of alterations such as he describes.

Experimental Heart Hypertrophy.—Following injections of adrenalin in rabbits, hypertrophy of the heart has been noted by certain observers, but so far the reason for this hypertrophy has not been satisfactorily explained. Miesoviez (*Wiener klin. Woch.*, 1909, xxii, 79) has compared the weights of the hearts of normal rabbits with the weights of the hearts from rabbits which had received repeated injections of adrenalin. Since the actual weights of the heart of rabbits varied within wide limits it was only possible to use the relative weight of the heart to the body weight. This figure was fairly constant. In all the rabbits inoculated with adrenalin, hypertrophy of the heart was found, and the hypertrophy affected almost exclusively the left ventricle. The importance of sclerosis of the splanchnic arteries in the causation of heart hypertrophy in man, as pointed out by Romberg and Hasenfeld, suggested that the change in the bloodvessels caused by the adrenalin injections might account for the cardiac hypertrophy; but on analysis it was found that in several cases in which there was well-marked hypertrophy arterial changes were entirely absent. It was also found that there was no constant relationship between the numbers of injections of adrenalin and the amount of cardiac hypertrophy; and well-marked hypertrophy occurred after comparatively few injections.

Bearing upon this question are the observations of Loeb and Fleischer (*Jour. Exp. Med.*, 1909, x) who found that there occurs regularly in rabbits after the injection of adrenalin, myocarditic lesions of the left ventricle. These lesions can be seen with the naked eye, occur early, and appear to be more constant and characteristic for the injections of adrenalin than the arterial lesions. They consist in an hypertrophy of the muscle fibers with increase in the number of muscle nuclei, and indistinctness of cross striæ, increase in the connective tissue, which occurs early, and degenerative processes affecting the muscle fibers which become especially marked at a somewhat later stage. The authors call attention to the analogy which exists between these lesions and the changes described in hypertrophied hearts in man.

The Multiplication of Diseased Lymph Nodes.—Rudolph Hammerschlag (*Virchow's Arch.*, 1908, cxciv, 320) studied, principally by means of serial sections, the form and arrangement of packets or groups of enlarged lymph nodes from such condition as tuberculosis, sarcoma, and carcinoma, and points out two methods, which, so far, seem to have escaped notice by which multiplication of lymph nodes may take place. He has noticed that many of the large lymph nodes are composed really of two or more lymphoid masses, separated, sometimes partially, sometimes almost completely, by a definite zone of connective tissue. These separate lymphoid masses may give the node an irregular, lobulated appearance. Such lobules arise from sprouts which grow from the original node and are gradually cut off by a band of connective tissue growing from the hilum of the node. These sprouts may become sessile and finally appear as new glands lying next to the original node. In another group of cases the actual number of lymph nodes is increased by the proliferation of the lymph follicles in the cortex of a node. The proliferated follicles become separated from the original node and form new glands.

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ORIGINAL ARTICLES.

INFECTION OF THE URINE AND THE URINARY TRACT BY BACILLUS COLI IN INFANCY.

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While infants do have ascending infections of the bladder and kidneys from urethritis of various sorts, severe inflammations of the bladder from stone with secondary pyelitis and pyonephrosis, tuberculosis of the bladder and kidneys, and all of the other diseases of these organs which occur in adults, they have them extremely rarely, so rarely, in fact, that they may almost be regarded as among the curiosities of medicine. Bacterial infection of the urine and urinary tract with the presence of pus in the urine, associated in some cases with epithelium of various sorts and occasionally with casts, is, however, not at all uncommon. It is usually not recognized, however, as most physicians are not familiar with the condition, or, if so, do not bear it in mind. In the vast majority of cases the infection is with the colon bacillus, although Bacillus lactis aërogenes, the typhoid bacillus, Bacillus proteus vulgaris, and several others have been found in rare instances. This condition has been described by various authors under various names, bacteriuria, pyelitis, cystopyelitis, colicystitis, pyelonephritis—according to the individual author's idea of the location of the infection, their opinions apparently having been based on the microscopic appearances of the urine. How difficult or even impossible it is to determine the exact seat of the lesion, and hence to give the condition a definite

name, will be evident later. It seems safer, therefore, to speak of this condition as infection of the urinary tract by Bacillus coli.

Mode of Infection. Theoretically, infection of the urine and the urinary tract by the colon bacillus can occur in three ways: (1) Through the blood—hematogenous or descending theory; (2) through the urethra—ascending theory; and (3) through the tissues between the intestines and bladder—transparietal theory.

1. *Descending Theory.* It is well known that a general bacterial invasion occurs in the course of many diseases. It was suggested by Cohnheim that in this event the body protected itself by the excretion of living organisms through the intact kidney. Other observers, notably Sherrington, have shown, however, that while organisms injected into the blood may escape in this way, the excretion is neither rapid nor immediate and may not occur at all. It is probable, therefore, that organisms pass through the kidneys only after the secreting tissues have been injured (Wyssokowicz[1]). The urine in these cases, however, does not, as a rule, show any evidences of injury to the renal tissue, containing only occasionally hyaline and fine granular casts, almost never blood, and seldom much albumin. The fact that pathological lesions of the pelvis of the kidney have been found at autopsy without any lesions of the ureters or bladder points very strongly, nevertheless, to a blood infection in some cases, at least (Knox,[2] Carpenter,[3] Finkelstein,[4] Baginsky[5]). So does the demonstration by cystoscopic examination of reddened ureteral orifices discharging pus into a normal bladder (Abt[6]).

Colon bacilli have been found in the blood of living infants ill with intestinal diseases (Czerny,[7] Moser,[8] Escherich[9]), and frequently in the blood and organs after death (Welch,[10] Marfan et Nanu,[11] Macaigne,[12] Finkelstein). On the other hand, they have not been found in the blood in some cases in which they were present in the urine (Moll,[13] Finkelstein), a fact pointing strongly, of course, to some other method of infection. Moreover, while the colon bacilli have frequently been found in the blood of boys, Bacillus coli infection of the urinary tract in them is rare (Finkelstein).

The fact that a large number of these cases are in girls points to some other method of infection, that is, through the urethra. Boys have this condition also, however, and in them an ascending infection is very improbable. In fact, almost 40 per cent. of my fifty patients were boys. In two of them, however, the infection was ascending as the result of circumcision.

[1] Zschr. f. Hygiene, 1908, lix, 1.
[2] Jour. Amer. Med. Assoc., 1907, ii, 1975.
[3] Ibid., 2004.
[4] Jahrb. f. Kinderheilk., 1896, xliii, 148.
[5] Deut. med. Woch., 1897, xxiii, 400.
[6] Jour. Amer. Med. Assoc., 1907, ii, 1972.
[7] Quoted by Cnopf.
[8] Quoted by Cnopf.
[9] Centralbl. f. Bact. u. Parasit., 1894, xv, 901.
[10] Quoted by Cnopf.
[11] Quoted by Cnopf.
[12] Quoted by Cnopf.
[13] Prag. med. Woch., 1907, xxxii, 501.

2. *Ascending Theory.* The chief arguments in favor of an ascending infection from the urethra are the much greater frequency of this condition in females, and the short, wide urethra which makes infection easy when the external genitals are soiled by feces. An argument against ascending infection is the fact that cystitis is a comparatively unusual complication of vulvovaginitis in infancy (Hutinel,[14] Baginsky, and others). Other arguments in favor of ascending infection as against descending infection are that in certain cases the urine is characteristic of cystitis, while in others the autopsy has shown lesions of the mucous membrane of the bladder while that of the ureters and pelves has been intact (Finkelstein).

The mere entrance of the colon bacillus into the bladder is not of itself sufficient to cause cystitis, as is shown by the fact that the injection of cultures of Bacillus coli into the bladder of puppies does not cause cystitis (Caccia[15]). In order that cystitis may develop there must be some abnormality of the bladder mucous membrane, diuresis must be interfered with, or the virulence of Bacillus coli exalted. The effect of hyperemia and lesions of the mucous membrane of the bladder or of stasis, produced by ligation of the penis, is proved by the experiments of Albaran and Halle,[16] who under these conditions were able to produce Bacillus coli cystitis by the injection of the colon bacillus through the urethra. The explanation given for the well-known fact that invasions of the urinary tract by the colon bacillus are especially common after or in connection with intestinal disturbances is that the inflammation of the colon causes tenesmus, the diarrhœa diminishes diuresis, and the indigestion or enteritis increases the virulence of the colon bacillus, while the general toxemia diminishes the resistance of the bladder mucous membrane by the cardiac weakness or degeneration of the epithelium which it produces. Trumpp,[17] however, found Bacillus coli in the urine of 14 of 17 infants—9 girls and 5 boys—ill with intestinal disorders without any other signs of infection, while Escherich's investigations have shown no increase in the virulence of the organism in cases of urinary infection with the colon bacillus.

3. *Transparietal Theory.* This conception presupposes some lesion of the intestinal mucous membrane, as various experimenters have shown that the colon bacillus cannot traverse the normal intestinal mucous membrane. Reymond[18] has shown that in adults Bacillus coli salpingitis and metritis are sometimes complicated by Bacillus coli cystitis. In these cases there are corresponding areas of inflammation in the bladder and in the organs outside, pointing to the direct passage of the organisms, and relief of the genital disease is followed by that of the cystitis. Wreden[19] has called attention to

[14] Presse méd., 1896, iv, 625.
[15] Riv. di Clinica Pediatrica, 1907, Abstract in Archives de méd. des enfants, 1907, x, 705.
[16] Quoted by Caccia.
[17] Jahrb. f. Kinderheilk, 1897, xliv, 268.
[18] Ann. des mal. des organes génito-urin., 1893, 253.
[19] Centralbl. f. Chirurgie, 1893, xx, 577.

the intimate anatomical relations between the colon and bladder in the male, and has shown by experiments on male rabbits that every injury of the mucous membrane of the colon at the level of the prostate or higher is followed by cystitis. The bacteria found in the urine in these cases were those found commonly in the intestines of rabbits. Vaseline and oil were found in the urine when these substances were injected into the bowel after injuring the intestinal mucosa. He concludes, therefore, that in males, at any rate, the colon bacillus can pass directly from the intestine to the bladder. Posner and Lewis,[20] however, state that bacteria have never been found in the tissues between the intestine and the bladder, while Posner and Cohen[21] have shown that when the renal artery is previously ligated the urine remains sterile. Consequently they believe that the infection is hematogenous.

Infection of the bladder, as already stated, is impossible while the mucous membrane is normal. Inflammation of the large intestine, because of the anatomical relations, produces congestion of the bladder mucous membrane and thus predisposes to infection, while in severe cases the rectal tenesmus modifies the function of the bladder and diminishes diuresis. The fact that urinary infection with the colon bacillus occurs most often in connection with or subsequent to intestinal disturbances is another argument in favor of the transparietal theory.

The examination of the urine does not aid a great deal in the determination of the method of infection. In many cases the urine contains only bacteria or bacteria and pus cells, a condition consistent with any method of infection. In some cases the urine also contains caudate cells and casts, suggesting that the severest process, and probably the earliest, is in the kidney and pelvis, and that the infection is therefore hematogenous or transparietal. In others the urine contains no casts or caudate cells, but considerable quantities of squamous cells, suggesting that the severest and presumably the earliest pathological process is in the bladder and that the infection is either through the urethra or transparietal.

It is evident from the consideration of these theories, it seems to me, that the method of infection is not always the same. It seems reasonable to conclude, however, that in the majority of the cases in girls the infection is through the urethra, while this route is very unusual in boys, and then only when there is some evident lesion, such as phimosis causing stasis, severe balanitis, or an infected wound from circumcision. In most cases in boys, and in a fair proportion of those in girls, the infection is probably transparietal, while in both sexes it is occasionally hematogenous.

Pathology. The pathological changes in these cases are slight, some reddening and swelling of the mucous membrane of a part

[20] Berl. klin. Woch., 1894, 742.
[21] Ibid., September 3, 1900. (Quoted by Ritchie.)

or of the whole of the urinary tract, some desquamation of the epithelium, and sometimes the evidences of degeneration of the lower tubules of the kidney.

Terminology. The confusion as to the terminology and the difficulty in the determination of the seat of the lesion or lesions in the infections of the urinary tract by the colon bacillus have already been mentioned. There can be little doubt, however, that the condition may vary from a simple bacteriuria without lesions of the pelvis of the kidney or of the bladder wall to one in which not only the bladder wall and the pelvis of the kidney, but also the renal tissue itself is involved. Judging from the urine and the pathological findings, the bladder is involved alone in some cases, the pelvis of the kidney alone in others, and both together in still others. It is presumable that the part first or most affected depends largely on the method of infection. In some cases the condition of the urine and the symptomatology make it evident where the chief seat of the disease, at any rate, is located. For example, the presence of considerable numbers of caudate cells indicates strongly involvement of the pelvis of the kidney; casts, an affection of the renal tissue itself; squamous cells, inflammation of the bladder wall. Tenderness in the lumbar region or in the hypogastrium points to involvement of the kidneys or the bladder, respectively. Frequent and painful micturition suggests that there is local trouble in the bladder. In many cases, however, there is nothing about either the symptomatology or the examination of the urine to aid in the localization of the process, the symptoms being entirely general and the urine containing merely bacteria and pus.

The general belief is that infection of the urinary tract with the colon bacillus in infancy is usually secondary to some disturbance of the intestinal tract, whether this be in the nature of constipation or diarrhœa. In many instances, however, no such connection can be made out, and in others it is very difficult to determine whether the intestinal disturbances are the result of the trouble in the urinary tract or the reverse. The disease was apparently primary in about two-thirds of my own cases. In two it directly followed circumcision, and in one girl the irritation from pin-worms apparently led to infection.

Bacteriuria. It must be remembered in this connection that the presence of bacteria in the urine as a secondary symptom of an infectious nephritis, or of a general infectious disease, does not constitute the disease, bacteriuria. This is a disease characterized by the presence of bacteria in exceedingly large numbers in the freshly passed urine and by the absence of marked symptoms of an inflammatory process in the mucous membrane of the urinary tract. Bacteriuria is most common in infancy and is almost always due to Bacillus coli. There are, as a rule, no severe general symptoms; at most, malaise and a slight elevation of temperature. Frequent

and painful micturition are not uncommon and older children often suffer from incontinence. The urine is uniformly cloudy, having the appearance of a bouillon culture of bacteria. The odor is unpleasant, the reaction acid. Animal experiments have shown that the virulence of the organisms in these cases is slight (Mellin[22]).

Urine. In most cases the urine is pale and uniformly cloudy or turbid. The turbidity is due in part to the bacteriuria and in part to the presence of large numbers of pus cells, chiefly to the latter. In rare instances it has a peculiar gelatinous appearance. The odor varies from normal to stale or very foul. The specific gravity is usually not increased. The reaction is almost invariably acid, not infrequently highly so. The acidity is due to the facts that Bacillus coli, although preferring an alkaline or neutral medium, can thrive in an acid medium, and that, as it does not decompose urea, it does not develop an alkaline reaction in the urine. This acidity is inimical to the growth of other organisms, so that Bacillus coli is most often found in pure culture. The urine usually contains less than 0.1 per cent. of albumin.

Microscopically, the sediment is composed almost entirely of pus cells, usually single, sometimes in clumps. Caudate, small round and squamous cells, in small numbers, are also present in many or most cases. Squamous cells are never present in large numbers, as they are in most cases of purulent inflammation, probably because of the absence of the ammoniacal products of the decomposition of urea which are the cause of the destruction and desquamation of the bladder epithelium in other infections. Hyaline and fine granular casts are occasionally seen, blood or blood elements almost never. The amount of urine is apparently uninfluenced by the disease, and varies directly with the amount of liquid ingested.

Symptomatology. In the majority of cases there is nothing whatever in the symptomatology to call attention to the urinary tract, the symptoms being merely an elevation of temperature and those common to all febrile disturbances in infancy, such as restlessness, drowsiness, fretfulness, and signs of indefinite discomfort. In some cases a yellow stain on the napkin first calls attention to the urinary tract. Symptoms of disturbance of the gastro-intestinal tract are especially common. Anorexia is the rule, and is often very marked. Vomiting is not unusual. The movements are usually abnormal, sometimes as the result of some disease to which the urinary infection is secondary, sometimes as the result of the infection itself.

The temperature (Chart I) is usually very irregular and in no way characteristic, suggesting confined pus more than anything else. In some cases it suggests an atypical malaria. In fact, the condition is for this reason often mistaken for malaria. This is especially

[22] Jahrb. f. Kinderheilk, 1903. lviii, 40.

likely to happen when, as is sometimes the case, there are also chills and sweating. In some cases, however, there is no fever. The following charts (2 to 5) are good examples of the irregularities in the temperature curve.

In some instances it is evident that the baby is suffering pain somewhere. It is very seldom, however, that there are any symptoms to suggest that the location of the pain is in the bladder or kidney. The pain is often paroxysmal. In one of my cases, a male,

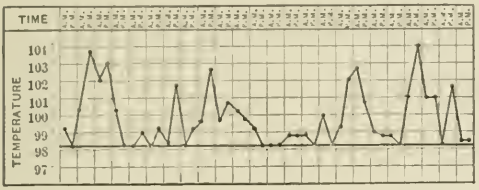

Chart I.—Irregular temperature of bacteriuria.

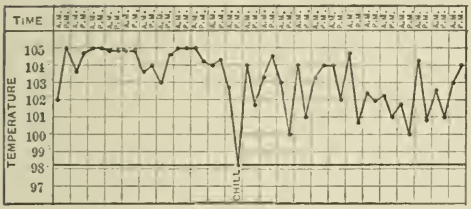

Chart II.

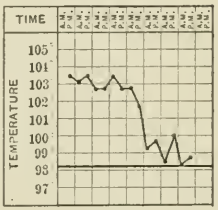

Chart III.

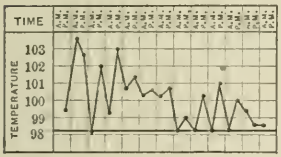

Chart IV.

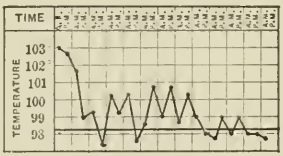

Chart V.

Charts II to V.—Irregular temperature of Bacillus coli infections of the urinary tract.

the attacks were apparently due to the blocking of the ureters by masses of pus. At any rate, the attacks were accompanied by swelling and tenderness of the kidney, and relief was followed by the passage of considerable amounts of urine containing macroscopic lumps of pus.

Tenderness or enlargement of the kidney is unusual. In one of my cases the enlargement was due to a non-suppurative perinephritic inflammation which required operative treatment. Tenderness over the bladder is somewhat more common. Frequent micturition is not common; sometimes there is partial retention of urine. Enuresis

may be the chief symptom in older children. Painful micturition is, in my experience, comparatively unusual. Loss of weight is usually rapid and marked. Anemia develops rapidly. Durante[23] attributes it to the specific hemolysins formed by Bacillus coli. There was always a leukocytosis in those of my cases in which the blood was examined, the highest count being 51,700.

To sum up: In the majority of cases of infection of the urinary tract in infancy with the colon bacillus there is nothing about the symptomatology to direct attention to the urinary tract. When such symptoms are present they are usually mild and are easily overlooked. Consequently the urine should be examined in all diseased conditions with indefinite symptoms in infancy, especially if febrile, and no physical examination should be considered complete or diagnosis satisfactory in obscure conditions in infancy unless the urine has been examined. In fact, in the vast majority of cases the diagnosis can only be made by the examination of the urine.

The following histories illustrate very well the various types of the symptomatology:

Case I.—A girl, aged thirteen months, was breast-fed for five months. She was then weaned gradually and put on a "hit-or-miss" mixture of top milk with Mellin's Food. on which she did very well. About three weeks before she was seen she began to be feverish, and was given calomel. The next day she was better, but two days later she had a chill. She had had no chills since then, but had sweat profusely at times and had lost much weight. The temperature had not been normal but once in the last two weeks, and had been very irregular. The food had been changed to a weak top-milk and barley-water mixture. She had not vomited, but had been constipated. The movements, however, were normal in character. The Widal reaction was negative. The diagnosis of malaria having been made, she had been given quinine in considerable doses during the last six days, without, however, any improvement in the symptoms. She was well developed and nourished and of fair color, but somewhat flabby. The physical examination, including the ears, was entirely negative. The white corpuscles numbered 37,600. The fresh urine was cloudy, pale, neutral in reaction, and contained a very slight trace of albumin. The centrifugalized sediment showed very many pus cells, free and in clumps, a few small, round, squamous, oval, and caudate cells, no casts, and many motile bacteria.

In this case, on account of the absence of urinary symptoms, the true condition had not even been suspected. Typhoid fever being excluded by the negative Widal reaction, an almost positive diagnosis of malaria had been made because of the fever, chills, and sweating. The failure of quinine to relieve the symptoms, however, then led to

[23] La Pediatria, 1903. (Quoted by Caccia.)

the suspicion that the diagnosis was wrong and that the baby had some other obscure disease.

Case II.—A girl, aged five and one half months, that had been perfectly well, suddenly became fussy and began to cry out and to have a high fever. Physical examination was entirely negative except for very questionable reddening of one drum. Paracentesis was followed by no discharge and no relief of the fever and restlessness. She took her food fairly well and digested it perfectly. The temperature ran between 103° and 105° F. for eleven days, when it became very irregular, ranging from normal to 105° F. On one occasion there was a chill. Examination of the urine on the seventh day showed only bacteria, but on the fourteenth day it was full of pus. This was complicated by a non-purulent perinephritic inflammation requiring operation.

In this case, also there was nothing whatever in the symptomatology to draw attention to the urinary tract. Looking backward, however, it is evident that the pain from which the baby suffered was caused by the inflammation about the kidneys. The redness of the ear drum led me astray for several days, and then I did not appreciate the importance of the bacteriuria, not realizing at that time that simple bacteriuria may be accompanied by severe symptoms.

Case III.—A girl, aged eight and one-half months, had been fed on home modified milk and had done perfectly well. Two weeks before she was seen the temperature suddenly rose to 104° F. without any apparent cause. The physical examination was negative. Her bowels were opened and she was given barley water and Mellin's Food. The temperature dropped to normal in about thirty-six hours, and remained there until the third day, when it rose again to 105° F. It then gradually fell to normal, went up again in four days, dropped to normal again in one day, stayed normal for two days, and went up to 104.8° F. again the morning she was seen. The physical examination had been entirely negative. She had refused her food on the days of fever, but had taken it well on the other days. She had not vomited. The movements were normal at first, but latterly had contained mucus. There had been a tendency to constipation. The bowels had been washed out almost every day. The washings apparently reduced the temperature. There had been no cough, no chills, and no cyanosis. She was well developed and nourished, but a little pale. The tonsils were a little large and the throat slightly reddened. The physical examination was otherwise entirely negative. The temperature was 102.8° F. The movements contained a good deal of mucus and were discolored with bismuth. The white blood count showed 51,700 leukocytes. The blood smears showed no malarial organisms and no changes in the red cells. The urine was cloudy and contained a large amount of pus.

In this case, too, there was nothing about the symptomatology to

direct attention to the urinary tract. The fever had been attributed to toxic absorption from the gastro-enteric tract because of the absence of physical signs, the mucus in the movements and the fall in temperature following irrigation of the bowels. The true condition had never been suspected.

Case IV.—A boy, aged eight months, ran a high temperature from December 23 to January 2 without any other symptoms, except that he had an attack of cyanosis daily when the temperature was highest. Puncture of the ear drums had no effect on the symptoms, which ceased as they had begun without any apparent reason. He was well from that time until January 13, except for some flatulence and constipation. The temperature from that time until he was seen, January 17, had been under 102° F. until that afternoon, when it went up to 105° F. He had apparently had some pain in the abdomen, had taken his food rather poorly, and had been troubled a good deal by gas. The movements were normal. There had been no trouble in passing water. The physical examination was absolutely negative in every way. The white corpuscles numbered 14,400. The blood showed no plasmodia. The urine, however, was found to be loaded with pus.

There was nothing in this case, any more than in the others, to direct attention to the urinary tract. Otitis media was at first supposed to be the cause of the fever, but this explanation had to be given up when there was no relief from the paracentesis. Typhoid fever and malaria were considered as possibilities, but an intestinal toxemia was considered most probable until the urine was examined.

Case V.—A girl, aged seven months, was taken suddenly sick with high fever the night of July 7. No cause could be made out. The temperature ran between 103° and 105° F. up to the time she was seen, July 14. The physical examination had always been negative. She had had a slight cough in the beginning. She had taken her food poorly, but had vomited but once. The bowels had moved regularly and the movements had been normal. She had always been conscious, but during the last two days had seemed tender all over and had held her head backward. During the last two or three days micturition had been painful and the urine had left greenish yellow spots on the diapers. She was well developed and nourished and of fair color. She was conscious, but irritable. She held her head backward, but the motions of the neck were not limited, and there was no tenderness of the neck. The physical examination was otherwise entirely negative. There was a small spot, looking like pus, on several of the diapers. The temperature was 103.6° F., the pulse and respiration were very rapid. The cerebrospinal fluid, obtained by lumbar puncture, was clear and under low pressure. Microscopic examination showed nothing abnormal. The urine contained much pus, an occasional cell, and many colon bacilli.

The importance of the painful micturition and of the greenish yellow spots on the napkins had been entirely overlooked, while meningitis was suspected because of the general tenderness and the retraction of the head.

Case VI.—A boy, aged twenty months and previously well, had a moderately severe attack of acute intestinal indigestion early in September, from which he recovered quickly. About the middle of September he had an infectious diarrhœa from which he had practically recovered, when, on October 4, his temperature suddenly rose to 103.5° F., and micturition became painful. He retained his urine as long as possible, and screamed when it was passed. The temperature ran between 99° and 103.5° F. The urine had stained the diapers yellow. His appetite was fair and the movements normal. Physical examination on October 6 showed nothing whatever abnormal until the urine was drawn and examined. It was found to be very turbid and to contain large shreds. It was full of pus cells, single and in clumps, but contained no casts or squamous cells.

The symptoms pointing to the urinary tract were unusually prominent in this case. Their importance had not been appreciated, however, and they had been attributed to the intestinal tract, probably because of the previous diarrhœa.

Case VII.—A boy, aged seven months, had always been breast-fed and had always been perfectly well. A month before a slight yellowish stain was noticed on a diaper wet with urine. The urine at that time was perfectly clear and the meatus clean. Two weeks later a similar stain was noticed on another napkin, and a week later the urine which was passed in the vessel contained a yellowish shred. This was examined microscopically and found to be composed of pus cells held together by a little mucus. From that time on the urine had contained pus. He had lost considerable color during the last two weeks and had gained little in weight; otherwise he had seemed well. There had been no increase in the frequency of micturition, no pain on micturition, no irritation about the meatus, and no urethral discharge. The physical examination was entirely negative, the temperature 99.4° F. An *x*-ray examination showed no stones in either kidney. The blood contained 8500 leukocytes. The urine contained a great deal of pus and many small round cells, but no tubercle bacilli.

In this case there was a deposit of pus on the napkins two weeks before the appearance of any constitutional symptoms. The importance of the yellow stain on the napkins was not appreciated, however, until the shred was noticed in the urine. It is noteworthy that there were no symptoms pointing to the urinary tract except the yellow stain on the napkins and the shreds in the urine. These would probably have been overlooked if the baby had not been the child of a physician.

Prognosis. The prognosis as to recovery is very good, a fatal termination being very unusual. But one of my 50 patients died. In most cases, however, the duration is long. Some patients recover entirely in two or three weeks, but more drag on for several months in spite of treatment. Exacerbations are common and relapses not rare. Complications are most unusual.

Treatment. Local treatment of the bladder is, as a rule, not indicated and is of comparatively little value. The drugs most commonly used may be divided into two groups—the alkalies and hexamethylenamine and its compounds. Theoretically the alkalies should do harm rather than good because the colon bacillus grows more luxuriantly in alkaline or neutral than in acid media. Moreover, there is more opportunity for a concurrent infection with other bacteria if the urine is alkaline. Clinically, however, many cases improve rapidly and recover when the alkalies are given. In fact, it seems to me that more cases do well on the alkaline treatment than on hexamethylenamine.

Hexamethylenamine is by far the most powerful drug of its class, and should be used in preference to any of the others. It liberates formaldehyde readily, but slowly and persistently, in the urine and has a strong antiseptic action. Unfortunately, although I am unable to find any experimental evidence on this point, it is a well-established clinical fact that the colon bacillus is, as a rule, but little and often not at all affected by hexamethylenamine. Consequently much less is to be expected from it in diseases of the urinary tract when the infecting organism is Bacillus coli than when other organisms cause the trouble. Nevertheless many authors feel that they have obtained good results with it and recommend it highly. I have myself seen cases improve and recover while taking it, but, as I have just said, it does not seem to me to act as well as the alkalies. Salol is also recommended. Its antiseptic action is, however, much less powerful than that of hexamethylenamine. Abt has seen guiacol, in one drop doses, apparently help in obstinate cases.

Various attempts have been made to treat this condition with serums of one sort or another. Combe[24] gave 10 c.c. of the Celli-Valenti anticoccal serum on two successive days in one case, and thought that he got favorable results. Dudgeon[25] has used an anti-colon bacillus serum in 50 cases in adults, giving 25 c.c. on three successive days. He found it most useful in acute cases, not so useful in subacute, and useless in chronic cases.

Another method of treatment, which seems most rational, and which has recently come into use, is the subcutaneous injection of dead cultures of the colon bacillus, regulated or not by the opsonic

[24] Riv. di Clinica Pediatrica, April, 1904. Abstract in Brit. Jour. Child. Dis., 1904, i, 331.
[25] Lancet, 1908, i, 615.

index. Caccia in his experiments on agglutination in Bacillus coli infections never got heterogenous agglutination, but did get homologous agglutination in high dilutions, suggesting strongly that, on account of the various strains of Bacillus coli, little can be expected from any but autogenous vaccines. These only, therefore, should be used. I feel sure, from the very general use of vaccine therapy during the last two years, that many cases must have been treated in this way. I have not, however, been able to run across them in my search of the literature. Dudgeon, speaking from his experience in adults, says that they occasionally do good in chronic cases, that they make the patient feel better and raise the opsonic index, but do not influence the bacilluria. Reasoning from the action of vaccine therapy in other infections, more should be expected from it in chronic than in acute cases of urinary infection with Bacillus coli. My own experience with the vaccine treatment has been rather limited and, on the whole, rather disappointing. In some instances it has seemed to do good, in others not. In no case, however, has it done any harm, although in one the first injection apparently caused considerable depression. The following cases are examples of the treatment by this method.

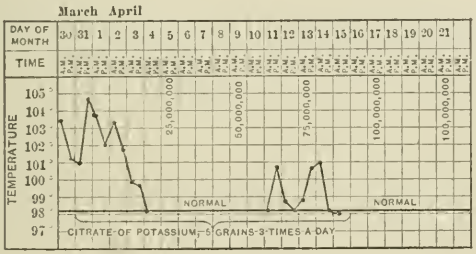

Chart VI.—Case VIII.

Case VIII. No. 3148.—Female, aged sixteen months, began to be out of sorts March 26. March 29 she was feverish, drowsy, and had a cough. She was sent to the Infant's Hospital with the diagnosis of pneumonia. The physical examination on March 30 was essentially negative, except for a slight rhinitis and a mild catarrhal otitis media. These were quickly relieved by treatment. The urine was cloudy, acid, and contained a slight trace of albumin. The sediment was composed of pus with an occasional squamous cell. Citrate of potassium, 5 grains, three times daily, was begun on March 31. (Chart VI.)

April 1. The urine contained many motile bacteria, many pus cells, singly and in clumps, and an occasional small round and caudate cell. There was no pain on micturition. The temperature dropped to normal by a rapid lysis after the citrate of potassium was begun, reaching normal on April 4.

April 5. 25,000,000 dead bacteria from an autogenous culture of colon bacillus were injected.

April 7. Urine: pale, slightly cloudy, faintly acid, 1016, slightest possible trace of albumin. Sediment: a few small round cells, and an occasional motile bacillus but no pus. The bacterial injections were repeated at intervals of about four days. In spite of them, however, there were slight exacerbations of temperature on April 11, 13, and 14, at which time the citrate of potassium was stopped.

April 15. Urine: the sediment contained bacteria and an occasional squamous cell.

April 22. Urine: normal color, clear, acid, slightest possible trace of albumin, an occasional squamous cell.

The injections were controlled by the opsonic index in this case. It is very difficult to decide whether or not they did any good, because the temperature had fallen to normal before they were begun and because the citrate of potassium was continued afterward. It seems reasonable to suppose that the improvement would have continued as it had begun if they had not been given. Whether it would have been as rapid, it is impossible to state.

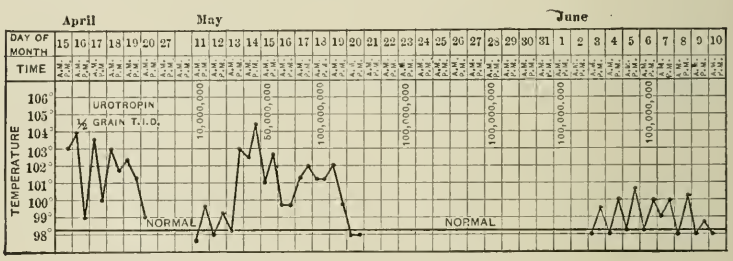

Chart VII.—Case IX.

Case IX.—No. 2883. Female, aged eleven months, had a temperature between 104° and 105° F., was restless and cried a good deal April 4, 5, and 6. During the next week she refused her food and slept but little. She did not vomit and the movements from the bowels were normal. During the last three or four days micturition had been accompanied by pain and straining, and the napkins had at times been stained brown and had a foul odor. She was admitted to the Infants' Hospital April 15. The physical examination was essentially negative except for moderate pallor. (Chart VII.)

Urine: the odor was foul; it did not contain albumin. There was much sediment which was composed of pus, free and in clumps, an occasional caudate cell, and many bacteria.

The next day hexamethylenamine, one-half grain, three times daily, was begun. The temperature rapidly dropped, reaching normal April 20. The urine on this day was foul, turbid, 1010,

slightly acid. The sediment was much less and was composed of pus, with many small round cells and many bacteria.

April 27. Urine: slightly foul odor, much less turbid, 1010, acid, sediment slight. It contained a moderate number of leukocytes and many highly refractive small round cells.

The hexamethylenamine was then omitted. As the urine did not clear up entirely, an autogenous vaccine of an unusual variety of the colon bacillus was prepared.

May 11. Injection of 10,000,000 dead bacteria.

May 13. Urine: slightly foul, slightly acid, 1010, slightest possible trace of albumin. The sediment was composed of broken-up cells, pus, and bacteria. The temperature on the 13th rose to 103° F., and continued more or less elevated in spite of injections of 50,000,000 bacteria on May 15, and of 100,000,000 May 18. After this it dropped to normal, where it remained for some time.

May 17. Urine: foul, turbid, amphoteric, slightest possible trace of albumin, much degenerated epithelium, very few casts, many bacteria.

May 18: Urine: turbid and foul, slightly acid, slightest possible trace of albumin. Sediment composed of pus, broken up-cells and many bacteria, but no casts. It will be noticed that the temperature rose and the condition of the urine became worse after the injections of bacteria were begun. On the other hand, however, the temperature dropped to normal and the urine began to improve while the injections were continued.

May 22. The turbidity of the urine was less.

May 28. The urine was slightly turbid, 1015, acid, no albumin, slightly foul odor. The sediment contained many less cells, fewer bacteria, and no casts. The injections were continued, but either in spite of, or on account of, the injections, the temperature was somewhat elevated between June 3 and 9.

June 9. Urine: pale, acid, 1010, no albumin, no sediment. In the centrifugalized sediment microscopic examination showed nothing except an occasional leukocyte.

The temperature did not rise again, and there was no return of the urinary condition. The injections in this case were not controlled by the opsonic index. The temperature had fallen to normal and the character of the urine was improving when the injections were begun. The temperature rose again and the character of the urine become worse after the first injections. These were continued, however, and the urine finally cleared and the patient recovered, whether as the result of or in spite of the injections it is not possible to say.

Case X.—No. 3232. Female, aged five months, was taken ill May 18 with a "cold on the chest." Since then she had been fretful and feverish and had had a slight cough, but had taken her food well. She had vomited once on May 23. The movements from

the bowels had at first been normal. She was said to have had a tender swelling on the right side of the abdomen May 22. She had strained and cried when the bowels moved during the last two or three days. The movements had been green and had contained curds and mucus. Micturition had been frequent and a yellowish stain had been noticed on the napkins. She was sent to the Infants' Hospital, May 25th, with the diagnosis of appendicitis or ileocolitis. The physical examination was negative. Nothing abnormal was detected in the abdomen. There was no tenderness over the kidneys. Urine: cloudy, acid, large trace of albumin, much sediment, consisting mostly of pus. It also contained an occasional red corpuscle, numerous caudate cells, a few small round cells, and much mucus. (Chart VIII.)

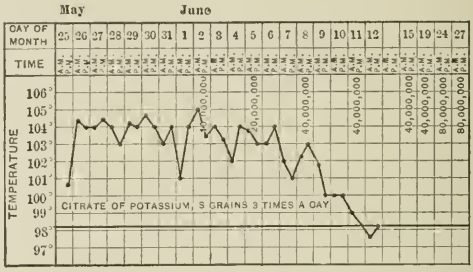

Chart VIII.—Case X.

May 28 she was given 5 grains of citrate of potassium three times daily; this was continued throughout her stay in the hospital.

June 1. Urine: acid, slightest possible trace of albumin, very many small round and pus cells, the pus being both free and in clumps, many motile bacteria.

June 2 she was given an injection of 10,000,000 dead colon bacilli, the vaccine being prepared from the organisms in her own urine.

June 5. Injection of 20,000,000 dead bacteria. Urine: high color, acid, very slight trace of albumin, very many pus cells free and in clumps, few small round cells, many bacteria.

June 7. The temperature dropped and there seemed to be some slight improvement in the general condition which, up to this time, had been poor.

June 8. Injection of 40,000,000 dead bacteria. This was followed by a further drop in the temperature and a rather rapid improvement in the general condition.

June 11. Injection of 40,000,000 dead bacteria. A drop of colon filtrate placed in the eye caused a purulent discharge.

June 12. The temperature reached normal the day before, and continued normal during the rest of her stay in the hospital. The urine contained much less sediment. This consisted of pus and

small round cells. The injections were continued, the amounts and dates of the injections being shown on the accompanying chart.

June 19 the urine showed about half as much sediment. This contained a very few small clumps of pus and an occasional scattered pus cell and small round cells with a few oval cells. There were many clumps of bacteria and a few motile organisms.

June 27. Urine much less turbid. The sediment contained many small, round cells, free and in clumps, an occasional leukocyte, many motile bacteria, and many clumps of dead bacteria.

June 28 she contracted measles and passed from observation.

In this case there was no improvement until the injections were given, although citrate of potassium was being taken. In fact, the general condition had grown steadily worse. Improvement was almost immediate after the injections were begun, and was continuous as long as the baby was under observation. It is almost certain that they did good in this instance. They were not controlled by the opsonic index.

My feeling at present as to the treatment is this: Alkalies are most likely to do good and should therefore be tried first. If there is no improvement while they are being given, hexamethylenamine should be used. If there is still no, or very little, improvement and the case is becoming chronic, autogenous vaccines should be tried. Circumcision should not be performed in these cases, as the resulting local reaction is liable to cause stasis and favor further infection (Rey[26]). In fact, two of the worst cases I have seen were the result of circumcision done while the infant was well.

THE DIAGNOSIS AND TREATMENT OF BILATERAL CYSTIC KIDNEYS, WITH SPECIAL REFERENCE TO THE DETERMINATION OF THE RENAL FUNCTION.

By Martin Krotoszyner, M.D.,
of San Francisco.

Prior to 1901, when James Israel's *Chirurgische Klinik der Nierenkrankheiten* appeared, only four authentic cases of bilateral polycystic degeneration of the kidneys in adults, correctly diagnosticated prior to autopsy or operation, had been recorded in the literature (Dugut, Verneuil, Stiller, and Lanenstein).[1] To this number Israel added four more cases, which he, too, was able to diagnosticate without resorting to the aid furnished by operative ocular inspec-

[26] Jahrb. f. Kinderheilk, 1901, liii, 648.

[1] Quoted from James Israel's Chir. Klin. d. Nierenkr., p. 542.

tion. The main reason for the failure to establish a correct diagnosis seems to be the absence of characteristic symptoms in many cases, until suddenly severe uremic symptoms set in and are quickly followed by the patient's death. I have seen a case of this type:

Case I.—A woman, aged sixty years, was treated in the medical service of the German Hospital for gastric disturbances (frequent vomiting) which apparently did not yield to symptomatic treatment. Quite suddenly she became comatose. On the right side of the abdomen a large mass was palpable, which was thought to belong to the right kidney. I was asked to effect, if possible, cystoscopic separation of both renal secretions with a view to determine the kidney function, prior to the intended exposure of the right kidney. Upon examination I found the patient in deep coma. The very scanty urine gave a heavy cloud of albumin and contained numerous granular casts and degenerated epithelial cells. On the right side of the abdomen an irregularly shaped mass was palpable; it was movable with respiration, of hard consistency, had a nodular surface, and apparently represented the enlarged right kidney. The left kidney could not be palpated. While preparing the patient for cystoscopic examination, she had a violent uremic convulsion, owing to which the examination was deferred. A few hours later she died. The autopsy showed the typical picture of polycystic degeneration of both kidneys, hardly any normal kidney tissue being noticeable. The right kidney was considerably larger than the left, but that organ, too, was at least three times the size of a normal kidney.

Although, on account of abdominal tympanites, palpation was difficult, nevertheless, a mass the size of the left kidney should have been detected, if efforts at palpation had been made as persistently and systematically as on the right side, and if the probability of the affection being bilateral had been thought of at that time.

Unfortunately the clinical charts and records of this case, which was observed at the time of the great San Francisco earthquake and fire, were lost. But the lesson I learned from my brief observation at the bedside, and especially from the autopsy findings, were not forgotten, and aided me in the diagnosis of the following case:

Case II.—A teamster, aged forty-one years, with a family history and a previous history of no importance, had at first, four years ago, experienced a rather sharp pain on both sides in the kidney region. The pain set in rather suddenly, was very severe, and at that time confined the patient to his bed for five days. After a year of apparently good health, during which time he followed his usual occupation, similar pain suddenly set in again. This time the patient was treated at a hospital for three weeks without much benefit, for he was still suffering from pain in both loins when he left the hospital. Gradually the pain decreased in intensity and the patient's condition improved to such an extent that he was able to resume his usual occupation for another year, when the pain set in again and

grew so unbearable that in December, 1907, he entered St. Mary's Hospital. Through Dr. T. E. Bailly, the attending surgeon's, kindness I was enabled to examine the patient, who at the time complained of continual pain in his back, general malaise, and great thirst. Repeated examinations of the urine revealed about the same findings as are generally present in chronic interstitial nephritis: large daily quantity, low specific gravity, traces of albumin, and microscopically small round epithelial cells and a few hyalin and granular casts. On both sides of the abdomen large irregularly shaped tumors of hard consistency were palpable; they moved with respiration, and apparently represented enlarged kidneys. Cystoscopy showed nothing of note in the bladder; ureteral catheterization had at that time to be deferred owing to the patient's extreme sensitiveness. The patient left the hospital a few days later, and at his home was seized with hematuria, which lasted several weeks. On January 9, 1908, he entered the German Hospital, where a systematic study of his case was made and where he remained until his death.

A careful general examination did not reveal anything of note, except an anemic murmur at the apex of the heart. The temperature ranged between 97° and 99° during the whole period of observation; the pulse varied between 80 and 100, except that toward the end of life it went gradually above 100. The urine analysis gave always the chemical and microscopic features generally observed in chronic interstitial nephritis. There were always a few red cells present in the microscopic slides examined. During the patient's stay at the hospital a marked hematuria of ten days' duration was observed; afterward the urine gradually cleared up again. Owing to the patient's great nervousness and his objections to cystoscopic examination, ureteral catheterization was performed on only two occasions. The first time the ureteral catheter entered the right ureter without difficulty, while the urine of the left kidney had to be taken from the bladder. Since, for obvious reasons, this method of separation of renal secretions is not absolutely reliable, only the urea content and the microscopic findings were determined at that time:

	Right kidney.	Left kidney.
	Per cent.	Per cent.
Urea	0.6	0.7
	Microscopically.	
	Many small round cells, blood shades, many bacteria.	Many red cells, pus cells, bacteria.

On February 17 both ureters were catheterized, but although the catheters were permitted to remain in situ for several hours, only a few cubic centimeters of urine was obtained, an amount too small to permit the performance of more than one functional test.

	Right kidney. Per cent.	Left kidney. Per cent.
Sugar (after phloridzin injection)	0.0	0.0
Urea	0.8	0.7

Microscopically.

Blood cells, blood shades, urates.	Blood cells.

When, a few days later, the same amount of phloridzin (1 c.c. of a 0.5 per cent. solution) was injected a very slight sugar reaction with Fehling's test was obtained in the bladder urine ninety minutes after the injection. (1 c.c. of the same phloridzin solution injected at the same time in a man, aged seventy-one years, suffering from a papilloma of the bladder showed a very strong sugar reaction thirty minutes after injection.) The blood cryoscopy was —0.60.

In order to gain more reliable data upon absolute functional renal capacity, the daily amount of excreted urea was measured over a certain period, during which the patient was kept on practically the same diet. Below is given a table showing the daily amount of urine and the urea excretion for fifteen consecutive days:

1908	Urine quantity in ounces.	Urea per cent.	Total urea.
February 5	60	0.7	12.6
6	60	0.7	12.6
7	50	0.7	10.5
8	48	0.8	11.5
9	40	0.7	8.4
10	30	0.8	7.2
11	28	0.8	6.7
12	28	0.8	6.7
13	28	0.9	7.3
14	52	0.8	12.4
15	28	0.8	6.7
16	28	0.8	6.7
17	32	0.8	7.68
18	28	0.9	7.56
19	16	0.9	4.32
Average	37	0.8	8.64

Various blood examinations made during the time of continuous observation gave the following result: The hemoglobin decreased from 70 per cent. to 55 per cent. The color index varied between 0.74 and 0.88; the red cells decreased from 3,088,000 to 1,920,000; the white cells increased from 12,400 to 17,000; the various differential counts gave an average of about 75 per cent. of polymorphonuclears, about 10 per cent. of large mononuclears, 4 per cent. of small mononuclears, and 7 per cent. of eosinophiles.

The tumors on both sides of the abdomen grew rapidly in size. On February 2, 1908, the following notes were made: In the left hypochondrium a resistance is felt extending down to the line of the navel. On the right side a tumor is palpable, the lower pole of which

extends to 5 cm. above the umbilical line. Both tumors are of hard consistency and are nodular.

On February 15 the following note was made: On ordinary examination the mass on the left side is palpable at a line about 4 cm. to the left of the navel and extends downward to 1.5 cm. below the umbilical line. The tumor on the right side is smaller and extends to about 3 cm. above the navel. The surface of both tumors is irregular, protuberances the size of a walnut being felt. These protuberances are particularly marked on the right side. Both tumors move downward with respiration about 2 or 3 cm.

The patient became perceptibly drowsy the beginning of February, and drifted into a comatose state about the middle of the month. He vomited frequently. On February 19 he was restless and complained of severe pain in the region of both kidneys. On February 20

FIG. 1.—The enlarged left kidney, one-fourth actual size, of Case II.

he was in deep coma; did not urinate spontaneously; although 300 c.c. of urine was evacuated by catheterization. Marked symptoms of uremia (coma, vomiting, convulsions, almost complete anuria) developed, and with these symptoms he died February 26.

Of the autopsy, which was performed by Dr. Ophüls, only those notes are given which pertain to the kidneys and liver (otherwise nothing of pathological note was found):

The left kidney (Fig. 1) is tremendously enlarged (more than 12 times the normal size), and consists practically entirely of smaller and larger cysts. It weighs 4 pounds 2 ounces, and measures 23 x 13 cm. The right kidney (Fig. 2) is almost as large as the left, and presents the same picture. It weighs 2 pounds, and measures 20 x 10 cm. The cysts are partly filled with clear watery fluid, partly with a brown liquid, and partly with blood clots. The liver is of normal size, and

shows quite a number of small cysts, from 2 to 4 mm. in diameter, filled with a clear fluid.

A careful ocular inspection of both kidneys fails to detect any perceptible areas of renal parenchyma which might be supposed to be of appreciable secreting activity. It appears, therefore, almost inconceivable that the patient, without grave symptoms of disturbed metabolism, could carry on life until the rather sudden onset of renal insufficiency two or three weeks prior to death. Such an observation, though, proves once more the clinically important fact that infinitesimal areas of normally secreting kidney tissue, not noticeable to the naked eye, may suffice to maintain the renal equilibrium under normal conditions. Equally surprising is the fact that a man

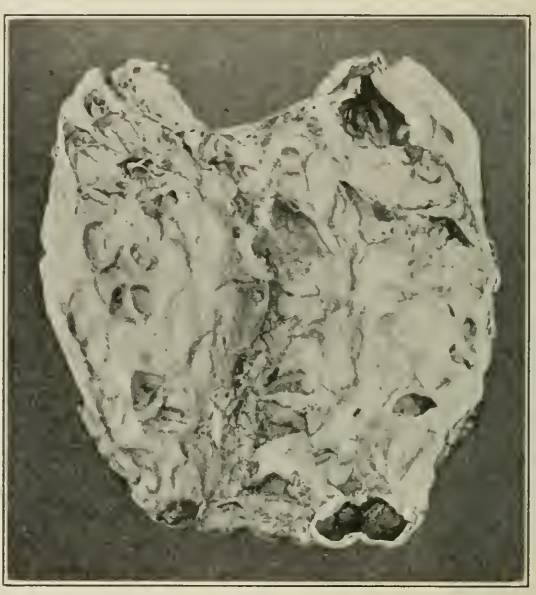

Fig. 2.—The enlarged right kidney, one-fourth actual size, of Case II.

with both kidneys anatomically diseased and functionally incapacitated could for a long time do hard physical work with comparatively little disturbance of his metabolism. Although this patient's first symptoms date back four years, he was able to attend to his occupation as a teamster almost continuously until a few months prior to death. He had probably large cystic kidneys for many years before their presence was ascertained by palpation, a presumption which is based upon the knowledge that cystic degeneration of the kidneys is in most instances a congenital affection. Israel has also observed several cases which, from the time that the presence of bilateral cystic kidneys could be ascertained by palpation, went on for ten

years and more with comparatively few symptoms caused by disturbance of renal metabolism.

The literature contains but scant references upon absolute and relative functional capacity of bilateral cystic kidney. Casper[2] expresses the hope that most valuable diagnostic data would be obtained by systematic investigations in this direction. Kapsammer[3] considers our newer methods of testing renal function most valuable for those cases of suspected bilateral cystic kidney in which enlargement of both kidneys cannot be ascertained by palpation, and in which bilateral functional renal depreciation points to the probable diagnosis of cystic kidney. Steiner[4] applied in one case in Israel's clinic the test of determining absolute urea excretion, and found abnormally large amounts (over 36 grams in twenty-four hours). In my case the daily average output of urea (8.6 grams) was far below that generally considered normal. These findings are the more significant, since they coincide with the low point of blood cryoscopy (—0.60) and especially because they are in accord with the comparative values of urea excretion found in the separated renal secretions (0.8 on the right, and 0.7 on the left side).

Since in cystic kidney, as a rule, we have to deal wth a bilateral affection, it stands to reason, that the determination of absolute or total renal function is as valuable if not more important than that of relative or comparative renal capacity. The determination of the twenty-four-hours urea quantity was always considered a more or less reliable index of absolute renal function. It is generally believed that normally functionating kidneys excrete a certain daily amount of urea (20 to 30 grams). We are, therefore, justified in assuming a lessened renal function from the inability of the kidneys to excrete waste products of metabolism in sufficiently large quantities. It must be admitted, though, that the determination of the twenty-four-hour urea, done but once, is more or less valueless, since urea excretion varies considerably under normal conditions, and the test itself is not scientifically accurate unless exact quantitative chemical determinations of ingesta and excretions over a certain period of time are carried out, which, for obvious reasons, is not a feasible routine bedside procedure. Nevertheless, if the test is carried out over a certain period of time, during which the patient is kept under strict observation in bed and on the same diet, the test for practical purposes is sufficiently accurate to permit reliable conclusions. Innumerable observations have proved that, other things being equal, 20 to 25 grams represent the average daily urea excretion of healthy kidneys. A decrease to over half the average normal quantity, therefore, would point to renal insufficiency, and in this case, was regarded as a contra-indication to operative interference.

[2] Lehrbuch der Urologie.
[3] Nierendiagnostik und Nierenchirurgie, ii, 301.
[4] Loc. cit., p. 545.

I am glad to see that Kümmell[5] too, is in the habit of applying the absolute urea test in a similar manner upon his large material of surgical kidneys prior to every intended operation. If the urea test applied over several days in the average coincides with a low point of blood cryoscopy, an operation, in Kümmell's opinion, is contra-indicated. An average daily urea excretion below 15 grams, according to Kümmell's experience, points toward a grave renal functional incapacity and forms as strong a contra-indication against an operative procedure as a low point of the more exact method of blood cryoscopy. Personally, I think that the urea test as carried out in this case is, as an indication of absolute normal function, more reliable than blood cryoscopy, which has disappointed most observers except Kümmell. I will concede to Kümmell, though, that the urea test gains in significance if it coincides with that of blood cryoscopy.

The third absolute functional test, the determination of phloridzin glycosuria in the mixed or bladder urine, was applicable in this case on account of the complete absence of sugar from both renal urines. It is well known that occasionally absence of phloridzin glycosuria in otherwise functionally normal renal urines occurs, and in the majority of instances is due to technical errors (old phloridzin solution, failure of heating the phloridzin properly, etc.), although some failures of obtaining positive results after injection of phloridzin as recorded in the literature (Israel and others) are not explained. These objections, though, are obviated by the positive result of the control test, and, therefore, the test as carried out in this case proves a marked depreciation of renal function by the belated appearance of a very faint positive reaction.

Since I was not able to obtain sufficient amounts of separated renal secretions for urine cryoscopy (10 c.c.), I had to content myself with the comparative phloridzin and urea tests. Both of them pointed likewise to an advanced renal insufficiency. The absence of phloridzin glycosuria alone, according to the experience of many observers, points to a functional renal insufficiency of such an advanced degree as to designate any operative interference a fatally hazardous risk. It is important, though, in order to gain a clear picture of the renal function in a given case, to perform several tests for absolute as well as relative renal capacity, since in the coincidence of these tests lies the reliability of the method. If the absolute tests, as in this case, are equally high or low among themselves, and if, as a whole, they correspond with the relative tests, we are able to draw fairly accurate conclusions, not only upon absolute renal function, but also upon the functional value of each kidney separately—for the real value of all functional tests, when combined, lies in their enabling us to formulate a reliable vital prognosis, and especially in offering us strict indications and contra-indications for treatment.

[5] Zeitschr. f. Urologie, ii, 3 and 4.

Whether to operate at all in a case of bilateral cystic kidney, which of the two diseased organs to expose first for relief of pressure and other distressing symptoms caused by large cysts, or whether nephrectomy, if otherwise indicated, should be attempted—all these questions can be answered with more or less accuracy by a careful study of the various functional tests in combination with the microscopic examination of the separated renal secretions.

In this case all functional tests pointed to such an advanced bilateral renal insufficiency, that an operative interference appeared to be contra-indicated. According to Kümmell and Graef,[6] Lund,[7] and others, excruciating pain, profuse hematuria, and very distressing symptoms caused by the presence of enormously large tumors would in exceptional cases be considered as indications for operative procedures (nephrotomy; puncture of the cysts, and packing of the larger cyst walls with gauze; nephropexy). None of these symptoms existed in this case to make an operation desirable, and, considering my patient's low renal function, I doubt whether the presence of one or more of these symptoms would have induced me to perform a hopeless operation. From this standpoint the value of our newer methods of renal diagnosis (ureteral catheterization and application of functional tests) as regards prognosis and treatment cannot be overestimated.

A word must be added in regard to the differential diagnosis. Malignant tumors, hydronephrosis, and echinococcic cysts of both kidneys had to be considered. Bilateral malignant kidney tumors (hypernephromas) are extremely rare. Malignancy does not lead to as enormous enlargement of both kidneys as does cystic kidneys, because in the rare instances in which malignancy on both sides has been observed, the second kidney became involved only a long time after the first. Palpation of hydronephrotic sacs gives characteristically smooth tumors, or, if they are irregular, equally large and flat protuberances can be felt, which are distinctly different from the unequal gibbus-like protuberances of cystic kidneys. Bilateral echinococcic cysts are extremely rare (only one such case having been observed) and give about the same palpatory results as hydronephrosis.

If, therefore, the symptoms and course of this case permitted from the start an exact diagnosis, and did not leave much doubt as regards the rational treatment, a third case, on the other hand, offered many unusually perplexing features, which made a correct diagnosis well nigh impossible and presented interesting problems as regards treatment.

Case III.—In April, 1906, I was asked by Dr. T. E. Bailly to examine a young woman, aged twenty-eight years, with the following

[6] Handbuch d. prakt. Chir., 1907, iv.
[7] Jour. Amer. Med. Assoc., August 18, 1906.

history: Her father died at the age of forty-three years from inflammation of the bowels; her mother is sixty-two years old and healthy; one brother is alive and healthy. The patient menstruated first at the age of fifteen years; menstruation has always been painful. Since early childhood she has had to urinate very frequently. About ten years ago she had painless hematuria, which lasted three days and has never returned. She has been married six years, and has no children. Of late she has suffered from severe dysmenorrhœa, for which curettage was performed about two years ago. Two months after this operation the patient was seized one night with very severe pains in the left loin, followed by high fever and general malaise. Her physician at that time informed her of the presence of a large floating kidney on the right side. The patient did not improve under symptomatic treatment, and was, therefore, sent to St. Mary's Hospital, where Dr. Bailly observed an irregular septic temperature, cloudy urine containing albumin and pus in abundance, but no tubercle bacilli. On the left side a mass was palpable, the exact outlines of which could not be made out satisfactorily owing to the patient's extreme sensitiveness. The right kidney was not palpable. There was frequent and very painful micturition. I was asked to perform cystoscopy, which revealed a severe cystitis and many ulcerated areas on the bladder wall. Catheterization of the left ureter was very difficult, as the ureteral os on that side was embedded in ulcerated tissue; the ureteral catheter readily entered the right ureteral orifice, which appeared to be normal. The separated kidney secretions showed macroscopically on the right side clear, and on the left side cloudy urine. The functional tests showed on both sides good values comparatively and absolutely, and the microscopic examination revealed on the left side the presence of pus cells in abundance and many blood cells, while the findings on the right side did not present anything of pathological note. This examination took place on April 16, 1906, and a report upon the findings was sent Dr. Bailly on the evening of April 17, the night before the great San Francisco earthquake. My own notes on this case were destroyed by the fire following the earthquake, and the report sent to Dr. Bailly by mail was likewise lost. This explains the failure of presenting exact data upon the functional and microscopic findings. In my opinion, communicated to Dr. Bailly, the patient was suffering either from an infected hydronephrosis or pyonephrosis on the left side. I advised, on account of the ascertained good function of the left kidney, against nephrectomy, which, as was pointed out, would only be indicated in case the inoculation test (guinea-pig) with the urine of the left kidney should reveal tuberculosis of that organ.

On account of the patient's grave general symptoms, Dr. Bailly, a few days later, opened from the loin by a left-sided nephrotomy a large suppurative cyst and drained through the incision. On the surface of the kidney several smaller cysts were noticed, and

were punctured. The operation had to be performed rapidly and under very awkward conditions in an overcrowded Oakland Hospital (St. Mary's Hospital having been destroyed by fire). The patient after the operation was for many weeks in a very precarious condition, which Dr Bailly ascribed to his failure to remove the left kidney. A renal fistula persisted for several months, but, however, finally closed. Before leaving the hospital the patient underwent a laparotomy under general anesthesia for ventrofixation of a retroflexed uterus. She recovered rapidly from this operation and left the hospital in October, 1906, in a fairly good condition, her only complaint at that time being frequent micturition and, at times, urinary incontinence.

The patient was sent to me again for examination in December, 1907. She suffered from headache, severe backache, and general malaise during the last six months. She urinated every hour during the day and once or twice during the night. The kidney on the right side was enlarged and easily palpable, the kidney on the left side also appeared to be larger than normal upon palpation. Cystoscopically the bladder showed the picture of an old cystitis, areas of scar tissue here and there in the trigone, but nowhere in the bladder were ulcerations noticeable. Both ureteral orifices appeared normal and were easily catheterized. Clear, straw-colored urine was collected from both sides. The analysis was as follows:

	Right kidney. Per cent.	Left kidney. Per cent.
Urea	1.5	1.1
Δ	—1.8	—1.5
Sugar (after phloridzin)	0.28	0.18

Microscopically.

Small round cells (pear- and spindle-shaped prevalent); red cells (in moderate amounts); pus cells (frequently in groups).	Small round cells (often in groups); red cells (abundant); blood shades; no pus cells.

The daily amount of urine was about 2000 c.c. The patient noticed an increase in the daily amount after the ureteral catheterization. Her headache improved since that time. *X*-ray plates showed the outline of large kidneys on both sides, while no characteristic shadows of calculi were visible.

The patient was seen again on April 14, 1908. She stated that headache and backache had gradually increased in intensity, so that life appeared almost unbearable. There was general malaise, and she was unable to work. She urinated about 100 c.c. every two hours during the day and twice during the night, and suffered tenesmus in the bladder at the end of urination. On palpation the right kidney appeared greatly enlarged, its surface irregular and nodulated; its lower pole was palpable at the navel line on ordinary respiration, while it was felt 5 cm. below the umbilical line on deep

inspiration. The left kidney was palpable, but did not appear to be perceptibly enlarged nor tender to the touch.

Cystoscopy: The second washing returned clear. The bladder wall and ureteral orifices were almost entirely normal. Both ureters were readily entered. The urine was clear on both sides. Both urines gave a positive sugar reaction after phloridzin injection. The urine in appreciable quantity was only obtained from the left side at this sitting, and all attempts to obtain sufficient urine for functional tests from the right side were futile. Therefore, cystoscopy was repeated two days later. Again, too little urine was obtained from the right side for the performance of more than one functional test and for comparative microscopic examination:

	Right kidney.	Left kidney.
	Per cent.	Per cent.
Urea	0.02	0.009
	Microscopically.	
	Blood cells; pear-shaped kidney cells; calcium oxalate crystals.	A few blood cells; a few pus cells; a few small round epithelial cells.

From the standpoint of differential diagnosis, renal neoplasms (hypernephroma), cystic kidney, and hydronephrosis on the right side had to be considered. The formation of a hydronephrotic sac in the dislocated right kidney seemed to be the most probable diagnosis, although the palpatory evidence (nodulation) did not support this presumption. Owing to the good functional capacity of the right kidney, which was also comparatively superior to that of the left organ, the removal of the right kidney was advised only as an extreme measure in case that organ should be found to be the seat of a malignant neoplasm.

FIG. 3.—Two cysts, two-thirds actual size, removed from the right kidney of Case III.

April 23, 1908. Operator, Dr. Bailly. The right kidney was exposed by a lumbar incision. The fatty capsule appeared to be very much thickened. The greatly enlarged organ was brought to the surface with great difficulty, and presented the typical picture of cystic kidney, the whole organ being studded with cysts of sizes from a walnut to a pinhead (Fig. 3). The largest cyst, which ex-

tended from the centre to the upper pole of the posterior surface and revealed a turbid, mucopurulent fluid, was evacuated. From all other cysts a clear watery fluid flowed upon puncture. When the kidney was divided, it presented on both cut surfaces numerous larger and smaller cysts, which were carefully evacuated. Very small islands of apparently normal kidney parenchyma seemed to be left after evacuation of all macroscopically noticeable cysts. All larger cystic cavities were packed with vioform gauze, and the kidney was sutured at both poles with a few deep catgut sutures. The final steps of the operation were decapsulation and nephrofixation, and drains to the kidney pelvis and from the upper and lower kidney poles through the incision.

The patient rallied comparatively quickly from the operation. On the second day after the operation the bandages were soaked with urine. A rather alarming hemorrhage occurred when the gauze drains from the cut surfaces of the kidney were removed. The patient urinated spontaneously about 1200 c.c. of urine daily, and nineteen days after the operation had an almost normal temperature and a strong pulse of about 100. The wound was practically closed about one month after the operation, while a small fistula in the centre of the incision was closed about a month later.

September 16, 1908. The patient appears again for examination. She has gained in weight and strength, and is able to be about. Her only complaint is a rather severe and annoying backache. The urine is clear, of amber color, 1012 specific gravity, of acid reaction; it contains microscopically many bladder cells, a few round epithelial (kidney) cells, a few pus cells, and some uric acid crystals. The kidneys are not palpable. Cystoscopy: The bladder wall is entirely normal except the trigone mucosa, which is slightly congested. Both ureters being catheterized, sugar appears on both sides almost simultaneously twenty-six minutes after the injection of 2 c.c. of phloridzin solution. Blue colored urine appears almost simultaneously from both sides fourteen minutes after the injection of 4 c.c. of a 4 per cent. indigo-carmine solution. The comparative functional tests give the following results:

	Right kidney.	Left kidney.
Albumin	Very slight trace	Present
Urea	0.01	0.008
Δ	—1.0	—1.1
Sugar	0.2	0.23

March 3, 1909. The patient's general appearance is excellent; but she complains of lack of strength and backache; she is not able to do her housework. The lower pole of both kidneys is palpable. She urinates every four hours during the day and once or twice during the night. The daily amount of urine is about 1500 c.c.; it is clear, straw color, has a specific gravity of 1016, and contains a faint trace

of albumin and a few leukocytes, flat and round epithelial cells, and many bacteria. The patient cannot be persuaded to submit to cystoscopy and ureteral catheterization for determination of the renal function.

This case represents the not very rare type of bilateral cystic kidney, in which the cystic degeneration of one kidney is followed after a long time by involvement of its sister organ, so that a tumor was palpable on one side (left), while palpation on the other (right) side gave a negative result. This phenomenon alone made the diagnosis a very difficult task. The evacuation of a large suppurative cyst on the left side reduced that organ to almost normal size, while the increase in size of the right kidney made its palpation possible only during the few months prior to operation. The case further proves the value of determining comparative kidney function. If the left kidney had been removed—and its removal was only prevented by the fact that its functional value was found to be good and comparatively even better than the right organ—the operation which later had to be done on the right side would have been a more hazardous if not a prohibitive procedure.

Jändler[8] describes the case of a woman, aged forty-four years, in whom a cystic kidney on the right side, erroneously diagnosticated as a malignant tumor, was removed. Shortly after the patient had recovered from her nephrectomy, uremic symptoms set in and were followed by complete anuria. The left kidney, at that time, proved upon palpation to be as large as a child's head; its exposure revealed a typical cystic kidney, which was divided down to the pelvis; all cysts were evacuated and the patient made a good recovery for the time being. Bardenheur[9] successfully removed a cystic kidney which was the seat of a large pus cavity, although the other kidney was known to be probably cystic.

The mortality of nephrectomy for cystic kidney is very high. Sieber[10] has collected 213 cases of cystic kidney; in 62 nephrectomy being done, gave a mortality of 32.8 per cent. Küster[11] records a mortality of 21 per cent. among his nephrectomies for cystic kidney, while Newman's[12] statistics show a mortality as high as 37.5 per cent.

The majority of authors, therefore, advise against nephrectomy in cystic kidney. Paul Wagner[13] considers nephrectomy in apparently unilateral cystic kidney indicated only in those cases in which by the various functional tests, after bilateral ureteral catheterization, good function of the apparently normal organ has been proved, and in which, besides, by direct inspection and palpation of the exposed

[8] Würzburger Dissertation, 1894.
[9] Mittheilungen a. d. Grenzg. d. Med. u. Chir., 1900, vi.
[10] Deut. Zeitschr. f. Chir., 1905, lxxix.
[11] Nierenchirurgie, 1902.
[12] Glasgow Med. Jour., 1889, xxxi.
[13] Handbuch d. Urologie, 1905, ii.

and supposedly healthy organ, the absence of cystic degeneration has been demonstrated. On account of the ever-present danger of cystic degeneration of the other kidney, which may occur at a later period, Küster[14] considers the removal of a cystic kidney contra-indicated in every instance, even if the other kidney, for the time being, is found to be anatomically and functionally intact, a view which is also expressed by Garré and Ehrhardt.[15] Kapsammer[16] is almost the only authority who advises nephrectomy in unilateral cystic kidney, for it is possible, he reasons, that the removal of one cystically degenerated kidney may prevent a similar retrogression of its sister organ. This theoretical presumption should not militate against the more conservative course of nephrotomy which, as my last case demonstrates, proved to be the means of saving the patient's life.

On account of the frequently fatal outcome of nephrectomy, attempts have been made of late to relieve symptoms by less severe procedures. Tuffier[17] performed partial nephrectomy in one case with an apparently satisfactory result up to the time of publication (two years after operation). Repeatedly nephrotomy with drainage, in a similar manner as on the left side in my third case, has been done, and been followed by relief of symptoms. Edebohls[18] performed decapsulation in a case of bilateral cystic kidney complicated by chronic nephritis, which was followed by rapid improvement of symptoms.

In my third case the operation performed on the right side was that first practised in two cases by Curtis and Kammerer[19] and also successfully carried out in one case by F. B. Lund.[20] The kidney is incised, the walls of the larger cysts are broken down, and the depleted organ sutured to the lumbar muscles.

Küster[21] has proposed a similar procedure; he advises cutting away with scissors so much of the cystic walls as to prevent their being again filled with fluid. Paul Wagner[22] warns against this and Lund's or Kammerer's procedure on account of the inclination of cystic kidneys to septic processes. In my own case a septic temperature set in immediately after the last operation and necessitated the removal of the gauze pack much earlier than was intended; this procedure also was attended by an alarming hemorrhage.

From my own observations and after a careful perusal of the literature on the subject, I must endorse Sieber's[23] dictum: "If we consider everything that is said and written upon operative proced-

[14] Loc. cit. [15] Nierenchirurgie, 1907. [16] Loc. cit.
[17] Garré and Ehrhardt's Chirurgie, p. 303.
[18] Zentralbl. f. Chirurgie, 1903, xxx, 39.
[19] Annals of Surgery, 1901, xxxiv, 419.
[20] Loc. cit. [21] Loc cit.
[22] Die Fortschritte in d. Behandlg. d. soliden u. cystischen Nieren.- u. Nebennieren-tumoren, Folia Urologica, January, 1908.
[23] Loc. cit.

ures in cystic kidney and the results of these operations, we must again and again warn against unnecessary operations. Only strict indications which are a menace to life should force upon us the necessity of an operation, which ought to be as conservative as is consistent with the condition found at exposure of one or both kidneys."

Conclusions. 1. Polycystic degeneration of the kidneys is in almost all instances a bilateral affection. In cases in which, for the time being, one kidney appears to be anatomically and functionally healthy, an involvement of this organ may be expected to develop later.

2. Determination of the kidney function is indispensable prior to decision upon any operative procedure.

3. Nephrectomy in cystic kidney is always contra-indicated.

4. Excruciating pain, general sepsis caused by suppuration of cysts, profuse hematuria, and distressing symptoms due to the presence of enormously large cysts are, under favorable conditions (good renal function), indications for operative interference.

5. Nephrotomy with puncture of cysts, decapsulation, and nephrofixation are the operative procedures which in selected cases may give satisfactory (temporary) results.

In conclusion, I wish to thank Dr. T. E. Bailly for the opportunity to observe and the permission to report Cases II and III.

THE VALUE OF THE WASSERMANN REACTION IN CARDIAC AND VASCULAR DISEASE.

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The importance of syphilis as an etiological factor in the causation of arterial disense has been appreciated manv a year. Ambroise Paré, Morgagni, and Virchow insisted on this relationship. More recently Heller and Doehler have shown that the larger arteries, particularly the aorta, exhibit special changes due to syphilis. Heller thought these changes to be miliary gummas in the aorta, the changes occurring chiefly in the adventitia and media, and suggested that the process be designated as a mesaortitis syphilitica. Chiari preferred to speak of it as a mesaortitis productiva.

The same relation that exists between syphilis and aortic disease

has also been claimed for aneurysm. Many of the physicians of the seventeenth century were so much impressed with the importance of syphilis in the production of aneurysm that they described a special variety to which the name Venereal Aneurysm (Lancisi) was given. The later studies on mesaortitis syphilitica have substantiated these earlier views. But it has always been unfortunate that in encountering a case of aneurysm or of aortic disease occurring in middle or in late adult life, it was impossible to determine from a consideration of the clinical facts whether or not these conditions were of syphilitic origin, even though all other common determining causes of the disease were absent. If we conceive that endarteritis syphilitica is a common occurrence in the aorta, we may readily understand how gummatous changes in the tunica media may give rise by slow stages to the development of an aneurysm.

It has been customary, if not universally prevalent, to treat aneurysms by the administration of iodide of potassium. From a careful study of the later manifestations of constitutional syphilis, particularly as they affect the vascular and nervous systems, we are convinced that in these conditions the iodides are of very little avail, except that they may aid in the absorption of the gummatous thickenings. On the other hand, we are equally impressed with the fact, gained by experience, that for these late manifestations of syphilis, mercurial treatment is our chief reliance. Everything points, therefore, to the importance of finding corroborative evidence for the suspicion that in any given case of cardiac or vascular disease syphilis has been the most important factor. The uncertainty of former years has been replaced by the gratifying results obtained by recent serodiagnostic methods. The Wassermann reaction, if positive, proves with considerable accuracy that an active syphilitic poison is still circulating in the system. We were glad, therefore, to follow the lead of Julius Citron[1] in determining by this method the frequency of syphilis in cases of aortic insufficiency; but instead of limiting our inquiry to this one form of vascular disease, we have included aneurysms and other cardiac and arterial conditions. During the past winter such tests have been made upon cases that have come into the general medical service of one of us at the City Hospital, and into the neurological and medical service of the other at Mount Sinai Hospital.[2]

As for the method pursued in these tests, Dr. Sachs, in association with Dr. Castelli, has adhered to the classic form of the test as it is now practised in Berlin by the Wassermann school, the only variation from the original method consisting in the substitution of an alcoholic extract of the liver of a syphilitic fœtus for the aqueous

[1] Berl. klin. Woch., 1908, No. 48.

[2] Dr. Sachs wishes to express his indebtedness to Drs. Rudisch, Manges, and Brill for their courtesy in aiding him in this inquiry.

extract. All the precautions insisted upon by the Wassermann school were practised and the quality and strength of the reagents were tested in every series of examinations; control tests were instituted liberally, so that we are willing to vouch for the accuracy of the results. The Wassermann reaction, cumbersome as it may seem,

Number.	Initials.	Age.	Character of lesion.	Specific etiology known.	Wassermann reaction.	Remarks.
1	S. (male)	42	Aortic insufficiency	No	Negative	Two tests made
2	U. (female)	30	Aneurysm of aorta	No	Positive	
3	Bertha D.	18	Congenital heart disease, mitral stenosis, and insufficiency	No	Positive	Secondary chronic endocarditis
4	John S.	..	Aortic stenosis and insufficiency	No	Positive	
5	Jennie H.	20	Mitral stenosis	No	Positive	
6	Gussie N.	40	Chronic endomyocarditis	No	Negative[3]	
7	Nathan, E. C.	47	Angina pectoris	No	Weak positive	Noguchi test negative
8	Benjamin P.	..	Aortic stenosis and insufficiency	Yes	Positive	Tertiary lues; sudden death
9	Albert D.	41	Arteriosclerosis	Yes	Positive	
10	Esther G.	55	Diabetes and mitral stenosis	No	Negative	
11	Mary B.	..	Chronic mitral endocarditis	No	Negative	
12	S.	30	Aortic and mitral stenosis and insufficiency	No	Weak positive	In hemiplegia
13	Carrie B.	..	Mitral stenosis	No	Negative	
14	Robert J.	..	Mitral stenosis	No	Negative	
15	Mary F.	66	Aortic stenosis and insufficieny	No	Positive	
16	Robert T.	53	Aneurysm of aorta	Yes	Positive	Confirmed by autopsy
17	McG. (male)	40	Aortic insufficiency	No	Weak positive	
18	P. (male)	64	Mitral and aortic stenosis	No	Negative	
19	W. (male)	..	Tabes and aortic insufficiency	Yes	Positive	
20	Michael O.	37	Aortic insufficiency	Yes	Strong positive	
21	B. (male)	49	Aortic insufficiency; tabes dorsalis	No	Strong positive	
22	William C.	50	Aortic insufficiency	Yes	Weak positive	Confirmed by autopsy
23	John N.	70	Mitral insufficiency	No	Negative	Confirmed by autopsy
24	Mary C.	49	Aortic and mitral stenosis and insufficiency	No	Positive	
25	C. (male)	54	Tabes and aortic insufficiency	Yes	Positive	Second test after prolonged treatment; weak positive
26	L. (male)	24	Aortic insufficiency	Yes	Positive	Symptoms of cerebral lues
27	J. L. (male)	58	Aortic and mitral insufficiency	No	Weak positive	
28	M. (male)	42	Aortic insufficiency	Yes	Positive	Reaction negative after protracted treatment
29	Walter E.	24	Aortic insufficiency	No	Positive	Death; no autopsy
30	Philip L.	60	Aneurysm of aorta	Yes	Strong positive	
31	Sadie M.	45	Mitral stenosis	No	Negative	
32	Z. (male)	45	Aneurysm of innominate artery	No	Strong positive	
33	Mary N.	32	Mitral stenosis	No	Negative	
34	Adam Y.	32	Mitral stenosis and insufficiency	No	Negative	
35	Edward J.	55	Arteriosclerosis; aneurysm of aorta	Yes	Strong positive	
36	M. (male)	67	Aortic and mitral stenosis and insufficiency	No	Negative	

[3] Even at autopsy the syphilitic nature of disease was suspected, but could not be proved; the Noguchi test was also negative.

and demanding much time and patience on the part of the examiner, has, after all, stood the test of several years, and the results obtained by this method have been universally accepted. The few exceptional and contradictory findings, particularly in some acute infectious diseases, which have been published do not militate against the worth of the method in the vast majority of cases of syphilitic origin.[4]

We were interested chiefly, however, in the clinical facts, and in order to avoid criticism it seemed best in this study to adhere strictly to the original Wassermann technique. These cardiac cases constitute only a part of over 250 cases examined by us by the Wassermann method, and basing our opinion on this larger experience, we are persuaded that a positive Wassermann reaction implies active syphilis. Negative findings are not so significant. In spite of a negative reaction, active lues may exist, and yet we doubt whether this is frequently the case. When a negative reaction follows upon a positive reaction in the course of antisyphilitic treatment, a negative reaction signifies a gradual disappearance in the system of an active luetic poison. So much for the method and for the deductions to be made therefrom.

Returning to the consideration of cardiac and vascular conditions, we may note, first of all, that we have had five cases (II, XVI, XXX, XXXII, XXXV) of aneurysm, four of the cardiac aorta, and one of the innominate. The Wassermann reaction was positive in each of them. Two of these five patients admitted having had syphilis, two denied it, and one had had chancroid. If the test is reliable as indicating the existence of syphilis, it is seen how little importance is to be given to the patient's denial of the infection. This series is a relatively small one, and we are not willing to infer that a positive reaction will be found in all cases of aneurysm. On the other hand, there are cases in which the physical signs as well as symptoms indicate the existence of aneurysm, and in which the occurrence of a negative Wassermann reaction leads to some doubt as to the accuracy of the diagnosis. This was so in the case of a physician, aged fifty years, who, when he was thirty, contracted syphilis. He was treated by a prominent syphilologist in the city where he lived, and was pronounced cured. Every year after that time, until the onset of his present symptoms, he took iodide of potassium for some weeks. About ten years ago he began to have abdominal pain. Failing to get relief he had a laparotomy done. This revealed the existence of a kink of the colon. After the operation he was free from pain for some years. Two years ago he began to have pain in the back,

[4] At a later period one of us will have occasion to refer to some of the modifications of this method which have been proposed. Incidentally, however, we may refer to the fact that in a series of forty-five cases examined by Drs. Castelli and Sachs, according to the old method, and by Dr. Noguchi, according to his new method, the results were in almost entire agreement; so that there is little doubt that Dr. Noguchi's method will prove a most welcome addition to the serodiagnostic procedures.

which was often so intolerable that morphine had to be given. A diagnosis of aneurysm had been made, but the negative reactions to the Wassermann test had much weight in deciding against that diagnosis.

It is generally admitted that incompetency of the aortic valves is sometimes, perhaps frequently, due to syphilis, that is, to a syphilitic endocarditis, and there is no doubt that degeneration of the myocardium is likewise the result of the same infection. Adler[5] has insisted that syphilis should be considered a possible cause in every case of heart disease, and has also uttered the warning that in every case of syphilitic infection the heart should be carefully investigated. Realizing the rarity of gumma of the myocardium and of sclero-gummatous arteritis, he has dwelt upon the necessity of recognizing syphilitic myocarditis, that is, a primary interstitial myocarditis associated with panarteritis. He has cited the histories of several patients who recovered under rigorous antisyphilitic medication.[6]

The question that confronts us as practising physicians is, Can we distinguish cases of endocarditis or of myocarditis of syphilitic origin from those due to other causes? Until the advent of the Wassermann test the only way of determining this was by the results of treatment. From empiric knowledge the syphilitic cases could be suspected; neither from symptoms nor physical signs could it be said that the heart lesion was of syphilitic origin. A case of aortic incompetency of rheumatic origin presents the same clinical signs as does one of syphilitic. We are therefore unable to determine from a consideration of the clinical facts alone whether or not specific medication should be employed. When such doubt exists the Wassermann test will be of greater value to us, for although the absence of the Wassermann reaction does not disprove the existence of syphilis, its presence certainly justifies antisyphilitic medication.

Among the cases studied by us there were thirteen (I, IV, VIII, XV, XVII, XIX, XX, XXI, XXII, XXV, XXVI, XVIII, XXIX) in which the disease was limited almost entirely to the aortic valve. In 92 per cent. of cases of aortic disease a positive reaction was obtained; in 77 per cent. it was strongly positive. Citron found this to be the case in 62.6 per cent. of his cases. In his and in our own series the percentages are so high that one cannot doubt the importance of syphilis as an etiological factor in the causation of this condition. Of these 13 subjects, 8 acknowledged a previous specific infection; in the other 5 it was either unknown or denied. We have had 5 cases (Nos. XII, XVIII, XXIV, XXVII, XXXVI) in which there was both aortic and mitral disease. In this series there was one positive, 2 weakly positive, and 2 negative reactions. This proves that

[5] New York Med. Jour., October 22, 1898; Medical Record, February 20, 1904.

[6] Wright and Richardson (Boston Med. and Surg. Jour., 1909, clx, 539) have found structures identical with Spirochæta pallida in five cases of aortitis.

when there is a combined lesion of the mitral and aortic valves, rheumatism and other forms of endocarditis vie in etiological importance with those due to syphilis. In 11 cases of mitral disease alone the Wassermann test was made, and of these, 2 were positive and 9 were negative. In all of these specific infection was either unknown or denied.

Although it was not our purpose to include cases of arteriosclerosis or cases of dilated aorta accompanied by generalized arterial sclerosis, there were two such cases (VII and IX) in our series, one of which gave a positive and the other a negative Wassermann reaction.

In a case seen by one of us in the neurological wards of Mt. Sinai Hospital, a curious light was thrown by the Wassermann test on a condition that seemed unusually puzzling. The patient, a young man, aged thirty years, in good general physical condition, had sustained an apoplectic seizure followed by a left hemiplegia. The hemiplegic attack came on gradually without loss of consciousness, after the fashion of a hemiplegia due to thrombosis and not to embolism. His heart exhibited all the signs of aortic and mitral insufficiency, from which the occurrence of embolism could easily have been explained. In this case the Wassermann reaction was positive, and in view of such positive reaction it seems to us fair to infer that the resulting hemiplegia was not directly due to the cardiac condition, but that there was unquestionably a widespread specific endarteritis, which, as in so many other cases, was responsible for a hemiplegia due to thrombosis.

The value of this test, from the point of view of therapeutics, was well demonstrated by the following cases: A young man, aged thirty-seven years, was admitted to the City Hospital with symptoms which suggested aneurysm, but the preponderance of evidence was finally in favor of incompetency of the aortic valves. The diastolic murmur over the entire aortic area, which was propagated down the sternum, the Flint murmur, the forcible apex beat, and the hypertrophy, despite the apparent difference in the radial pulses, indicated aortic regurgitation. The patient made marked improvement under rest and mercurial medication, which was pushed to the extreme as a result of a positive Wassermann finding.

A positive finding helped to confirm the diagnosis of aneurysm in the following case: A negro, aged fifty-three years, who had had a chancre at twenty, complained of dyspnœa one year previous to his admission to the City Hospital. Six months later he began to cough and to have trouble in swallowing, and about this time he complained also of pain over the sternum. Physical examination revealed a tracheal tug, no pulsation of the neck, a soft, blowing, systolic murmur over the entire upper part of the sternum, profound dulness over the transverse arch of the aorta, and a distinct difference in the volume of the radial pulses. He had likewise a chronic interstitial nephritis. The aneurysm ruptured into the left pleural cavity.

It was large and sacculated, about 15 cm. in diameter, and has its opening into the aorta in the lower part of the transverse arch; anteriorly it pressed against the sternum. The left lung was adherent to the aneurysmal sac. There was a rupture of the aneurysm about the centre of the anterior wall along the line of adhesion of the lung. The left recurrent laryngeal nerve was involved in the adhesions.

In the accompanying table the main facts are stated in condensed fashion upon which our conclusions are based. These are: (1) From a study of 36 cases of cardiac and vascular disease the importance of syphilis as a causative factor is established in a very large percentage of all the cases. (2) The highest percentage was obtained in cases of aneurysm, in which a positive Wassermann reaction was obtained in all of five cases. In the cases of aortic disease 10 out of 13 cases gave a strongly positive reaction; 2 gave a weak positive reaction. (3) In marked contrast to this are the cases of diffuse chronic endocarditis affecting the mitral valve, in which only 1 of the 7 cases gave a positive reaction. The inference is justified that rheumatic and other infectious agencies are a much more potent etiological factor in diffuse chronic endocarditis than is syphilis. Taking all these facts into consideration, there can be little doubt that when we have pronounced symptoms of aneurysm or of aortic disease it is well to suspect syphilis, even though the manifestations may be latent. Citron is no doubt right in believing that syphilis is by far a commoner cause of aortic insufficiency than the history and the clinical findings would seem to suggest. Even in those cases in which articular rheumatism and other infectious agents may have played some sort of role, and in which metallic poison or abuse of alcohol or of nicotine be admitted, if a Wassermann reaction gives positive results, antisyphilitic treatment should be instituted, whatever else may have been discovered regarding the antecedent history of the patient. No doubt articular rheumatism and syphilis occur often enough in one and the same individual, and if the course of antisyphilitic treatment has been decided upon, we may at least ask that it be properly administered. Without entering upon the many moot points in this therapeutic controversy we are in favor of the routine treatment by hypodermic injection of mercury. We leave the choice of a soluble or insoluble salt to the experiences or the prejudices of the individual practitioner. Our best results have been obtained by the use of deep sublimate injections. It may be argued that little is to be expected from antisyphilitic treatment in an advanced case of aortic insufficiency or of aneurysm. In some of our cases marked improvement in the cardiac and vascular symptoms has followed upon such treatment, but even if the vascular system should not improve upon the mercurial treatment, a positive Wassermann reaction would justify vigorous specific treatment for the purpose of preventing serious syphilitic involvement of other organs and systems of the body.

THE VARIOUS TYPES OF PLAGUE AND THEIR CLINICAL MANIFESTATIONS.[1]

By Khan Bahadur N. H. Choksy, M.D.,
SPECIAL ASSISTANT HEALTH OFFICER TO THE BOMBAY MUNICIPALITY, IN CHARGE OF THE ARTHUR ROAD INFECTIOUS DISEASE HOSPITAL AND THE MARATHA PLAGUE HOSPITAL, BOMBAY, INDIA.

The invasion of the human organism by Bacillus pestis manifests itself in certain well-defined and clinically recognizable forms. Their development is determined solely by the path of entrance of the virus into the system: (*a*) If the infection takes place through the skin, by the bite of a rat-flea, or any other agency, and is conveyed into the lymph glands by the lymph stream, the *bubonic* type is the result; (*b*) if, under identical conditions, the infection becomes localized in the skin and subcutaneous cellular tissue, with or without secondary involvement of the lymph glands, the *cellulocutaneous* type ensues; (*c*) if, again under the same circumstances, the blood stream becomes directly infected, the *septicemic* type develops; and (*d*) if the lung-tissue becomes primarily infected, through inhalation or other causes via the air passages, the *pneumonic* type ensues.

Four clinical types are, thus, capable of differentiation at the bedside, each having special characteristics of its own, and each with an important bearing upon the prognosis. The almost classical and synonymous association of the word buboes—*emerods* in Biblical language—with plague, absorbed the whole attention of the observers to the exclusion of the other types, when the disease first broke out at Bombay. Clinical observation, however, soon revealed the presence, as Childe[2] was able to demonstrate, of a few cases of the pneumonic type in 1897. Subsequently the septicemic type was encountered and described by several observers, including myself; and finally, after careful study of a number of cases, extending over three epidemics, I was enabled to verify and describe the cellulocutaneous type in 1900.[3]

All these types do not prevail to the same extent. Hospital experience at the Arthur Road and Maratha Hospitals at Bombay,[4] throughout twelve epidemics comprising over 13,600 cases, shows that the *bubonic* type contributes 92.8 per cent. of all cases; the *cellulocutaneous* type, 3.7 per cent.; the *septicemic* type, 2.4 per

[1] Read by title at the sixth annual meeting of the American Society of Tropical Medicine, Washington, D. C., April 10, 1909.

[2] Transactions of the Medical and Physical Society of Bombay, 1897.

[3] Some Observations on Plague and its Treatment with Lustig's Serum, Transactions of the Medical and Physical Society of Bombay, 1900.

[4] N. H. Choksy. An address on the General Pathology and Serum Treatment of Plague, 1908.

cent.; the *pneumonic* type[5] 1.0 per cent.; and *pestis ambulans,* 0.1 per cent. These ratios, however, are not constant even for all of India; and in other parts of the world many variations have been recorded. At Hongkong, for instance, Koch[6] has reported the following incidence: The bubonic type, 63.6 per cent.; the septicemic type, 31.4 per cent.; and the pneumonic type, 5 per cent., in 510 patients observed during 1904.

I. The Bubonic Type. Within three days after infection and usually without any prodromes,[7] the patient becomes suddenly ill, sometimes in the midst of his work, has a rigor, feels giddy, and may even fall in the act of walking. The temperature begins to rise almost immediately and simultaneously with it, pain and tenderness in the infected glands are complained of. Such glands may be situated in different parts of the body, the order of frequency being the femoral, inguinal, and iliac glands, separately but more often in continuation, the axillary, and the brachial chain with the epitrochlear, the cervical, parotid, and submaxillary glands, and but rarely those in other positions, such as the femoral chain, the popliteal, in the calf, at the ankle, at the bend of the elbow, pre-auricular, or even occipital. The eyes become suffused, the mind somewhat dull, the speech faltering, and the expression either apathetic or anxious and frightened The patient is generally averse to acknowledge that he is at all ill, and often refuses to take to his bed. Even if convinced that he is afflicted with plague—for there exist but few who do not realize it—he persists in ascribing his symptoms to a chill or exposure, and the swelling and pain in the bubo to a sudden strain and more often to the initial fall. On the other hand, it often happens that the patient not only apparently deludes himself, but succeeds in deceiving those around him, by denying the presence of pain or tenderness in the bubo, and if the medical attendant is not confident or resolute in his diagnosis, he shares the same fate, to the ultimate ruin of the patient. As the temperature rises, the pulse becomes quickened, usually out of all proportion to the normal ratio, is found to be of low tension and easily compressible, even within a few hours. There may be nausea or vomiting, mostly of bile, and by the evening the temperature becomes elevated to 104° to 105°; there is restlessness and insomnia, if not actual delirium. Pain and swelling in the bubo are more noticeable, and periglandular œdema may appear. The patient thus presents within a few hours all the cardinal symptoms of plague.

[5] Major G. Lamb, I.M.S. The Etiology and Epidemiology of Plague: A Summary of the Work of the Plague Commission, 1908. The Plague Research Commission states that the proportion is about 2.5 per cent., and that is probably correct, inasmuch as many cases go unrecognized and others succumb at home.

[6] Colonial Medical Report for Hong Kong for the Year 1904, Journal of Tropical Medicine, August 1, 1905.

[7] No local lesion is noticeable at the seat of infection except among 8 to 10 per cent. of cases.

The following morning a slight remission in the temperature may or may not be observed, but without any amelioration; all the symptoms, general and local, become more intensified; the tongue is now dry and presents the characteristic appearance of wash leather, but with a red tip and margins; the weakness of the circulation is more pronounced, and the extremities are cold. The patient does not like to be disturbed out of the position he may have assumed, resents all or any interference, is obstinate, and refuses nourishment and medicines; there is extreme irritability, coupled with greater dulness of the mental faculties; the speech is thicker, the syllables being drawn out, or there may be aphasia; delirium may exist; and the temperature rises to the same or even a higher range than the first day. After a restless night and the lapse of forty-eight hours, there appears a distinct and perceptible remission in the temperature, varying from 1° to 4° or more, with or without apparent amelioration. If the former, it is extremely deceptive and of short duration, inasmuch as within a few hours there is a complete change for the worse in every respect. The usual course however, is for the remission to be unaccompanied by any temporary improvement. As all the symptoms become more and more aggravated, the buboes are larger and more prominent, the local œdema greatly increased, the pulse feebler. By evening the temperature again becomes elevated to the same or even a higher level than on the previous days. With these symptoms the patient may be said to enter the most critical phase of the disease, inasmuch as the following two days decide his fate—the average duration of fatal cases being but five and one-half days. Here we are practically at the parting of the ways the future depending upon two factors: (1) It depends upon the extent of the cardiovascular paresis, as also upon the acute infective degeneration of the myocardium brought about by the toxemia. These lead to sudden cardiac syncope or gradual enfeeblement of the circulation, with eventual heart failure, preceded by a slowly progressive œdema of the lungs, owing to the vicious circle set up between the circulatory and the respiratory apparatus. Should this occur, death usually takes place between the third and fifth days of illness. Fully 82 per cent. of all deaths among these cases occur within this period, and 18 per cent. thereafter. The average rate of recovery among them is about 55 per cent. (2) It depends upon the presence of septicemia, for it is about this period, that is, the third day—though in a few cases even earlier—that this primary dominant factor in plague shows itself. Instead of the infection remaining strictly localized within the lymphatic system, it becomes generalized through infection of the blood stream, owing to numerous direct communications that naturally exist between the lymph channels, on the one hand, and arteries and veins on the other, in the axilla, the side of the thorax, in the groin and other situations where buboes mostly predominate. In the case of deep abdominal

buboes the veins become adherent to them, and thus direct infection of the blood is brought about. Once Bacillus pestis gains entrance into the circulation, it multiplies enormously, and rapidly becomes distributed throughout the internal organs, notably in the spleen and the liver. Masses of bacilli cause embolic infarcts in the organs, besides inducing terminal pneumonia. And as disintegration goes on simultaneously with multiplication, the liberated endotoxin simply floods the tissues, produces necrotic degeneration of the organs, and sets up hemorrhages from almost all hollow and solid viscera; profound changes in the finer elements of the nervous system occur from the toxin combining with the tissue cells, disorganizing their functions and laying in train the seeds of future mischief, the effects of which time and patience alone can eradicate.

The septicemia is generally of a progressive type, being most intense just prior to death; the number of organisms present in the blood may vary from 10 to 1,000,000 per cubic centimeter, and the growth of colonies on sloped agar from 0.1 c.c. of the blood may be few, fairly numerous, numerous, or very numerous, as classified by the Plague Research Commission.[8]

The development of septicemia can only be positively confirmed by bacteriological examination of the blood by culture, as the microscope fails to give any positive indication except in about 17 per cent. of such cases. The clinical signs, however, in about 72 per cent. of these cases raise a strong presumption of its presence; these consist in a quick, thready, compressible or almost imperceptible pulse, accompanied by intense nervous prostration, jaundice, and rapid wasting of the muscular and adipose tissues of the face, the features becoming pinched and hollowed out, even in the course of a single night, together with hemorrhages from the stomach, kidneys, or intestines. *Per contra,* the condition of the patient in about 28 per cent. of such cases, even in spite of a profound septicemia, is apparently so favourable that the physician is often deceived; the prognosis suddenly and, indeed, without any warning becomes falsified within a few hours, as death takes place by rapid cardiac failure. About 85.5 per cent. of all deaths among septicemic cases occur within six to seven days of illness, and 14.5 per cent. thereafter. The average recovery rate among them varies from 3 to 4 per cent. only, as compared with 55 per cent. among the non-septicemic.

If the ultimate fate of the patient is to be decided by the first factor—cardiac failure through toxemia—his general condition becomes gradually worse after the evening of the third day, the temperature oscillates until death between 102° to 104°, the delirium becomes fierce or maniacal, requiring physical restraint; he refuses

[8] Journal of Hygiene, Extra Nos. 1 and 4, being Reports on Plague Investigations in India, September, 1906, and May, 1908.

medicines and nourishment, and each act of struggle or restlessness adds to the efforts of the greatly embarrassed heart, until it eventually ceases to beat, the patient sometimes expiring in the course of or immediately after a violent attempt at exertion, engendered presumably through deficient oxygenation or air-hunger. On the other hand, if a septicemia is to be the determining factor, the struggle between its steady, onward progress and the defensive processes of the system is no less arduous or prolonged. The clinical features above described supervene, the temperature, however, affording no clue; terminal pneumonia, internal and external hemorrhages, a semicomatose condition or actual coma, end the scene within the first week. Whenever a septicemia is limited in extent or is controlled by the action of the antipest serum, and the life of the patient thus prolonged, he drifts into marasmus, which I have described fully in a paper read at the Bombay Medical Congress of 1909.[9]

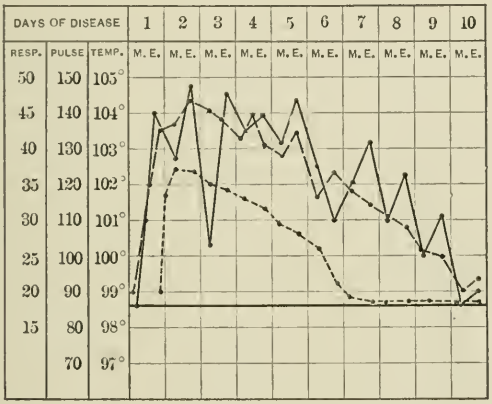

FIG. 1.—Chart of a normal case of plague of the bubonic type. (Solid line = temperature; dash line = pulse; dotted line = respiration. Same device in the other charts.)

If the patient is to recover, the further progress of the case is demonstrated by the accompanying chart (Fig. 1). After the lapse of forty-eight to sixty hours or longer since the evening of the third day, that is, by the sixth or seventh day, a gradual but perceptible change for the better becomes evident. The temperature slowly declines, there is amelioration in the general condition, the pulse improves in volume and tension, and the bubo or buboes become more circumscribed and prominent from commencing necrosis and suppuration. The pyrexia terminates by lysis, by short elevations and remissions, until it reaches normal on the morning of the ninth

[9] N. H. Choksy. On the Clinical Significance of Septicemia in Human Plague, Bombay Medical Congress, 1909.

or tenth day, the tongue gradually clears, the appetite returns, the patient's appearance is brighter, and a very hopeful sign—he commences to grumble about something or other, or everything imaginable. Should the buboes suppurate, incision is required. The curve of temperature in a normal and uncomplicated case is thus limited to about ten days; occasionally it is shorter, but never less than eight days. It is usually divisible into three curves, the first terminating on the morning of the third day, the second on that of the sixth or seventh, and the third on or about the tenth day. This indicates that the disease is self-limited and bound to run a definite course, and that no abortive treatment can possibly deflect or modify its course except the early and liberal use of the antipest serum. Variations in the above curve are numerous: there may be but two only, or they may merge into each other; and, besides, complications, deep-seated buboes or their extension, or a succession of buboes in different positions, each ushered in by a well-marked elevation—all these render the curve unrecognizable except to an expert.

II. The Cellulocutaneous Type. The development of the cellulocutaneous type is dependent upon the peculiar property of Bacillus pestis whereby its protoplasm or endotoxin is capable of setting up inflammatory and retrogressive changes of tissue elements, leading to progressive local necrosis. Klein[10] says, in reference to other organisms (but equally applicable to the bacillus plague), that the necrosis following typical symptoms of inflammation is the result of the local action of the bacilli, and so long as they are in sufficient numbers and of sufficient virulence, and so long as their multiplication proceeds, the necrosis of the tissues spreads into larger and larger areas. That is well exemplified in the case of the large necrosis often observed in plague.

Definite lesions at the point of infection exist in about 8 to 10 per cent. of cases, consisting in the development of phlyctenules, pustules, bullæ, and umbilicated bullæ. It is from the latter that this type originates. If the bulla is cut open or bursts of itself and the central core is removed, its point of attachment to the base appears as a small dark spot. It gradually increases in size until the whole of its base is encroached upon, and obliterated by a dark eschar. This continues to spread at the circumference from day to day until a period is reached when the medium suitable for the further multiplication of Bacillus pestis becoming exhausted, a line of demarcation appears. The eschar assumes a dark greenish hue, feels cold to the touch, is almost hard and leathery, and depressed in the centre, as if firmly bound down to the tissues underneath. Its spreading margin, somewhat higher, is conterminous with the surrounding skin, which becomes converted into a hard, angry, in-

[10] Brit. Med. Jour., August 4, 1900.

flamed areola studded with minute vesicles; and often small, secondary necrosis is noticeable at some distance from the periphery through infection from the parent necrosis when it hapens to be extensive. Multiple umbilicated bullæ are also occasionally met with on different parts of the body surface, each giving rise to a small necrosis. If a little fluid be examined from the margin, it invariably shows a pure culture of Bacillus pestis. The destructive process, if limited, becomes a comparatively benign form of plague, to which I have applied the term "Cellulocutaneous Plague."[11] The buboes, if present—they are not invariably so—are secondary; and although, owing to the slowness of the process, the course of the disease is prolonged from two to three weeks instead of the normal period of eight to ten days, the recovery rate is higher. It is probable that recovery is determined, as Pescarolo and Quadrone[12] say in relation to Eberth's bacillus in typhoid fever, by the behavior of Bacillus pestis in the tissues in relation to the processes of infection and immunization, which differs, as we have already seen, from what it is when confined to the circulation; and in its conflict with the cellular elements of the tissues, immunizing bodies are produced which bring the infective process to a rapid termination. Should the necrosis become extensive, as in some cases it does, involving as much as sixty to ninety square inches of the body surface, the infection becomes generalized and the patient succumbs through septicemia.[13] Once the line of demarcation has formed, it is not difficult to remove the entire slough en masse, leaving behind a saucer-shaped depression in the subcutaneous tissue (often exposing the deep fascia and muscles) of gradually increasing depth from the periphery to the centre, filled with shreds of necrosed tissue, pus, and blood; it gradually assumes a healthy aspect under appropriate treatment. Such necroses have been observed on the scalp, neck, face, back, chest, arm and forearm, the abdominal wall, lumbar and gluteal regions, over the mammary gland, the vulva, and on the scrotum, the thigh, leg, and foot; in fact, wherever such umbilicated blisters might be situated The destruction of the soft tissues is often enormous, and large cavernous cavities exposing the bones of the face and head, involving exfoliation of the periosteum, and so also the cartilages of the ear, the eyelids, and the nasal bones. The upper and the lower jaws may be exposed when the process is confined to the head, neck, and face; the ribs may become exposed on the side of the thorax, and the testes if the scrotum is involved.

The clinical features are confined mainly to the local manifestation, its progress being marked by a slow and steady advance from

[11] Otherwise called carbuncles, and hitherto assumed to be a complication of plague; also known as pest necrosis.

[12] Zentralb. für inn. Med.; Brit. Med. Jour. (Epitome No. 292), November 14, 1908.

[13] It is also possible for septicemia to occur through the original bulla, before necrosis develops, as in Case No. 129, of 1908, at the Maratha Hospital.

day to day until the line of demarcation forms. The presence of buboes in direct lymphatic communication with the necrosis is dependent upon its extent. If the necrosis is small and less than a dollar in size, the buboes are more pronounced and the clinical phenomena almost identical with those observed in the bubonic type. Should it be larger—four to six inches in diameter—the buboes are of subordinate significance, inasmuch as the invasion being strictly localized, the general disturbance of the system is not so great and the result favorable. When the primary necrosis is at all extensive and oversteps the above limit, there are more chances of a septicemia developing, or, again, the patient may succumb through secondary infection or prostration, even after its excision.[14] The recovery rate is about 36 per cent., as compared with 25 per cent. in the bubonic type, and thus it constitutes the most benign type of plague.

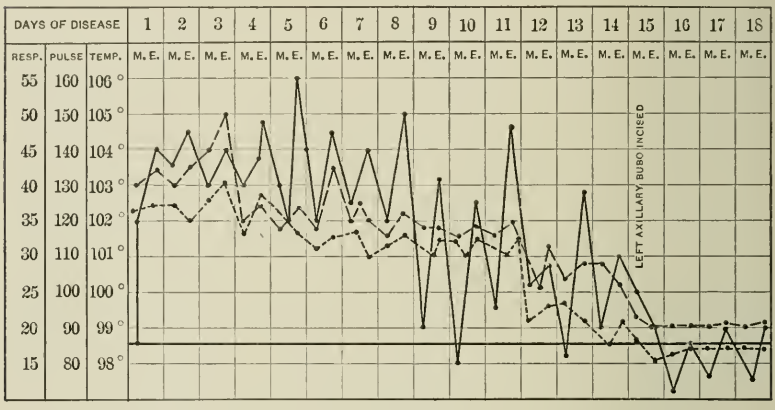

Fig. 2.—Chart of a normal case of plague of the cellulocutaneous type.

The accompanying chart (Fig. 2) illustrates the temperature curve of a case in which two necroses existed under the left costal margin, together with a bubo in the left axilla; the latter suppurated and was incised on the fifteenth day of illness. The course of the affection is seen to be prolonged to a fortnight, and the temperature curve is quite dissimilar to that in the bubonic type.

The accompanying photograghs (Figs. 3 to 8) demonstrate the varying extent of the necrosis in different cases.

III. The Septicemic Type. The septicemic type owes its origin to primary infection of the blood, Bacillus pestis gaining direct entrance into the circulation.[16] The course of the affection is vari-

[14] N. H. Choksy. The Treatment of Plague with Prof. Lustig's Serum, Bombay, 1903.

[16] Malignant infection, with tiny deep-seated buboes and early septicemia, is liable to be mistaken for septicemic plague, the true nature of such cases being revealed only at the autopsy.

able, extending from five to ten days. As in secondary septicemia in the bubonic type, a certain proportion of cases do not exhibit

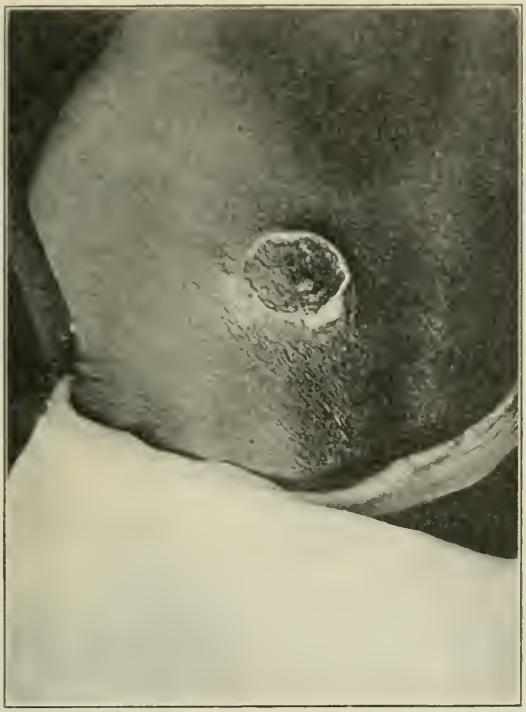

FIG. 3.—A small necrosis on the back in the lumbar region with secondary buboes in Scarpa's triangle; recovery.

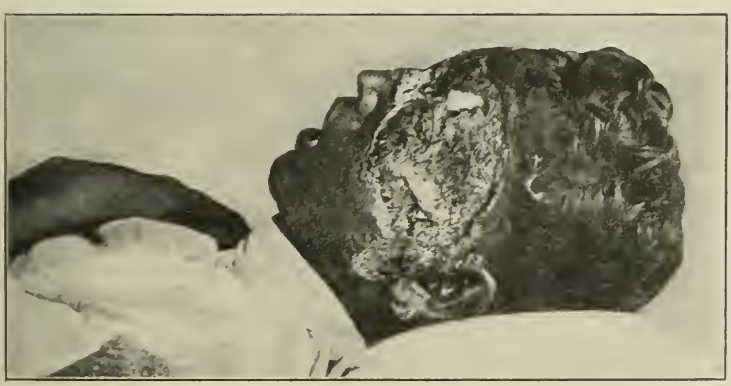

FIG. 4.—A large necrosis involving the forehead and side of the face over the temporal, zygomatic, malar, and nasal regions, with destruction of the upper and lower eyelids; recovery.

any clinical signs of grave systemic infection; so also there exist similar cases in the primary infection, in which, beyond a persistent high temperature with short morning remissions and evening exacerbations and a weak and frequent pulse, there is total absence of all grave symptoms: no jaundice, no wasting, no prostration, no delirium

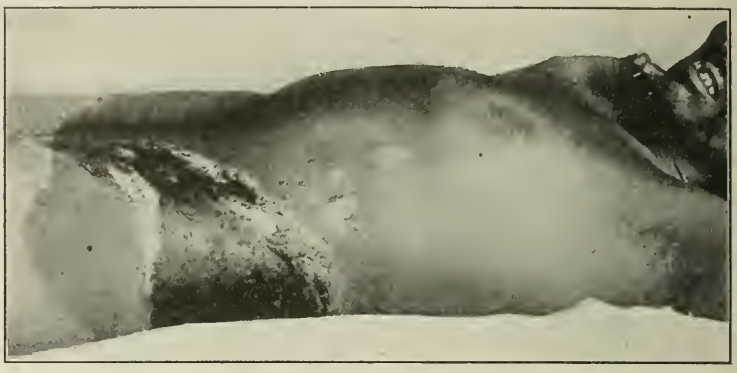

Fig. 5

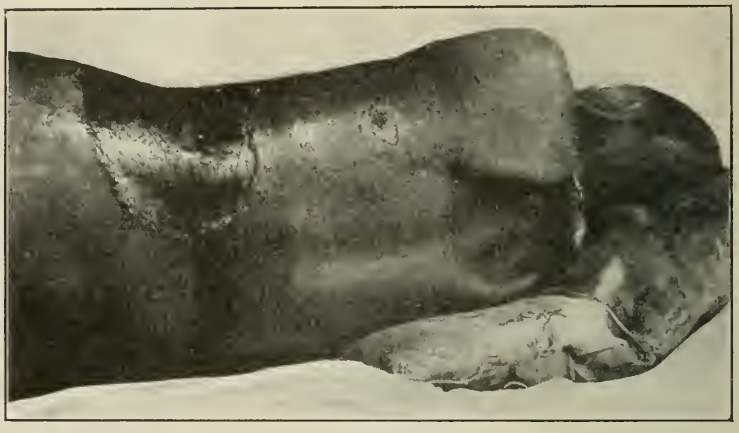

Fig. 6

Figs. 5 and 6.—Front and back views of an extensive necrosis, extending from the fold of the groin to the iliac crest, over the hip, and to the lumbar region, with small secondary necroses; fatal.

the mental faculties perfectly clear, and to all intents and purposes nothing apparently serious to bespeak a fatal termination within three to six days. And yet, such is the way in which they invariably terminate. The only possible means of diagnosis is by culture of the blood, which is not often undertaken until too late, and by the time it becomes confirmed the patient has succumbed through cardiac failure.

The accompanying chart (Fig. 9) illustrates the course of such a case with all the above characteristics.

Fig. 7.—Secondary necrosis from the bubo infecting the subcutaneous cellular tissue in Scarpa's triangle; recovery.[15]

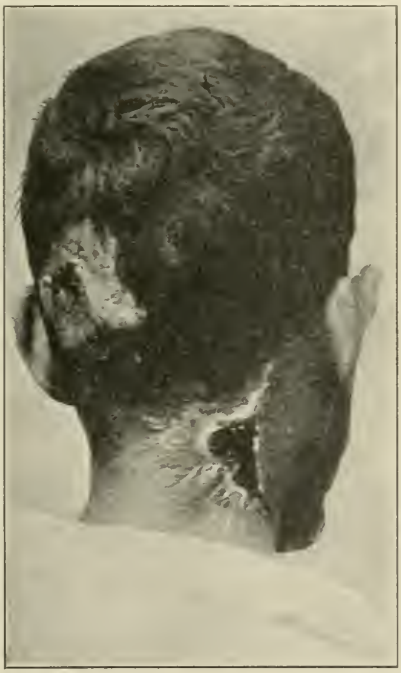

Fig. 8.—Wounds from two necroses, one on the scalp and the other on the side of the neck, in the process of cicatrization.

[15] Secondary necrosis from infection of the skin and subcutaneous cellular tissue by the bubo is occasionally met with, when the latter rapidly enlarges, comes up to the surface, and forms adhesions. It spreads similarly as the primary, and when the eschar is removed, the bubo is found to be firmly attached to its under surface.

By far the more common, however, is the type in which the symptoms are well pronounced at the very outset, the temperature usually following until the fourth day the ordinary course, as in the bubonic type, but variable thereafter. An extremely weak and thready pulse, intense and grave jaundice, profound nervous prostration, the limbs heavy and almost leaden, stupor or coma, the tongue brown, hemorrhages from the stomach, kidneys, or intestines, or subconjunctival, coldness of the extremities, and a few hours before death the appearance of one or more buboes in different parts of the body—such is the clinical picture exhibited. The only affection for which such cases are likely to be mistaken is grave spirillar infection in relapsing fever, but the absence of spirilla in the blood and the culture test confirmed the diagnosis. The following charts illustrate the course of such cases: Case No. 1721, of 1905 (Fig. 10), indicates the presence of severe melena causing

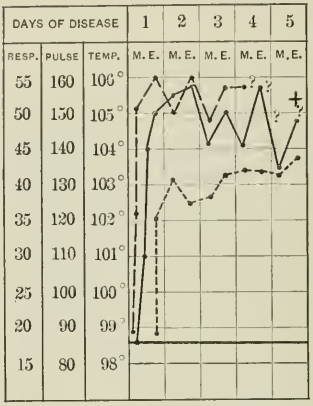

Fig. 9.—Chart of the septicemic type of plague.

collapse with subsequent reaction, and the development of femoral, inguinal, and iliac buboes on the morning of the day the patient succumbed. The other two cases, No. 838, of 1905 (Fig. 11), and No. 65, of 1904 (Fig. 12), show a somewhat more prolonged course, extending between nine to ten days, with the development of buboes on the seventh and ninth and tenth days respectively. In a few septicemia cases, however, the buboes appear on the third day of illness, the affection becomes partially localized within the lymphatic system, and recovery is just possible. But, on the whole, the fatality of these cases is so great that not more than 2 per cent. recover.

IV. The Pneumonic Type. The most insidious and virulent and at the same time highly infective is the pneumonic type. The infection is usually contracted by inhalation of the fine spray of the infective sputum teeming with the bacilli. It is just possible that

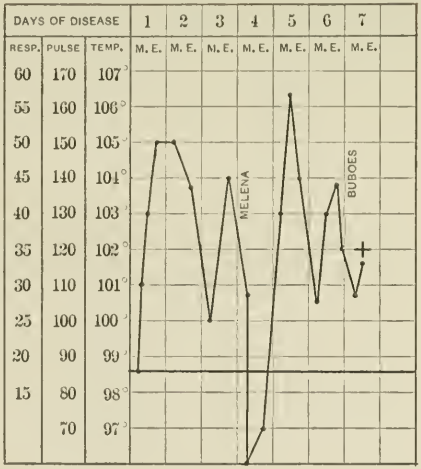

Fig. 10

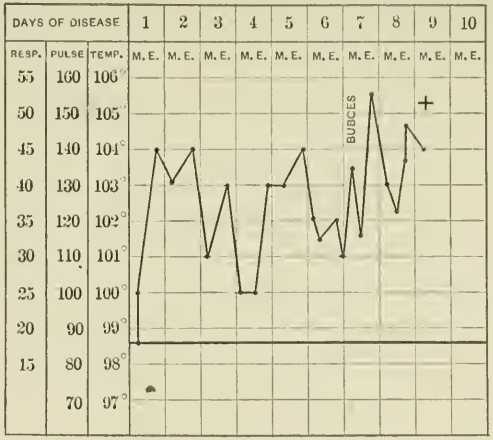

Fig. 11

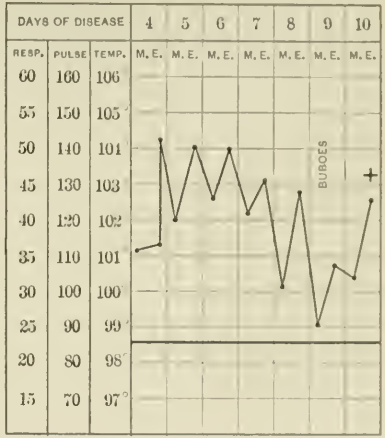

Fig. 12

Figs. 10, 11, and 12 —Charts of the septicemic type of plague to illustrate the presence of internal hemorrhage and the late development of buboes just before death

it may be conveyed by the infected finger to the pharynx or tonsils, where a focus is formed, whence the lungs become infected. Or, again, the apparent primary pneumonia may be not really so, but a secondary development of malignant infection with deep-seated buboes, not felt during life-time, and early septicemia. The clinical picture of the type is masked in the early stages, and does not exhibit its full development until late. The course varies from three to five days, the termination being invariably fatal (Fig. 13).

Moderate fever, neither the pulse nor the respiratory ratio correspondingly higher; no physical signs, and yet marked prostration and aspect of grave illness; then, within a few hours, some scanty pellets of hemorrhagic sputum, followed by death within twenty-four hours, characterize the disease. The apparent initial mildness rapidly assuming gravity even without the appearance of the char-

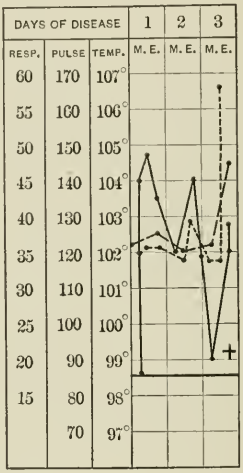

Fig. 13.—Chart of the pneumonic type of plague.

acteristic sputum is a pecular feature of the type. The sputum may be thick, viscid, almost or even purely hemorrhagic or thin and mucoid, tinged bright red; it may be profuse or extremely scanty, and brought up at long intervals. Simple microscopic examination of the sputum is extremely deceptive for purposes of diagnosis, as other organisms simulating the morphological characters of Bacillus pestis are not of infrequent occurrence. Many cases so diagnosticated have eventually proved not to be pneumonic plague. It is, therefore, absolutely necessary to make a culture and inoculate animals with it or with the sputum. If the animal responds to the usual tests, and plague bacilli are recovered from it, then only can the diagnosis be positive.

The rarity of this type and its great infectivity claim special

notice. One single case is often the focus of a whole series of cases. The case of the late Major Manser, I.M.S., and his attending nurses; that of one of the former house surgeons of the Maratha Hospital, with its series of sixteen cases; that of one of the nurses at the Bai Motlabai Hospital, and of the student, both attending upon a case of the pneumonic type, who had miscarried, one and all terminating fatally, are sufficiently appalling. So far as regards Bombay. On the other side of India, Major Green and Dr. Justice[17] have recorded the history of the Baker-gunge epidemic of pneumonic plague, in which one case originating at Calcutta, in the house of Kaviraj Dwarkanath, led to a series of nineteen other cases, all of which proved fatal. Lieut.-Col. Crawford[18] has recorded another similar outbreak, involving twenty persons at Hughli, in January, 1905. In all the above instances the infective agent was the sputum of the patient, which induced a similar affection among others. Under other circumstances, however, especially if it happens to fall upon an exposed and inflamed mucous surface like the conjunctiva, it gives rise to bubonic plague. The saddest case of the kind that has come under my observation was that of one of the English nurses, at the Arthur Road Hospital. She had a severe catarrh with injected conjunctiva at the time. While bending over a patient to feed him, he happened to cough, and a drop of the infected sputum fell into the eye; three days later she developed plague with preauricular, parotid, and cervical buboes, with infiltration on the side of the infected eye, and eventally succumbed from œdema of the glottis.

No recovery from pneumonic plague in which the diagnosis has been confirmed as above has been hitherto recorded in Bombay. I have seen none in the course of twelve epidemics. I am, however, able to report one instance in which there was double infection of the lungs by Bacillus pestis and Bacillus pyocyaneus. The patient was admitted on the third day of illness—July 1, 1907—with moderate fever, 101.8°; pulse, 101, of fair volume; and respirations, 30; no physical signs and no sputum to indicate the nature of the case, except marked prostration. On the night of the sixth day, however, the temperature rose to 104.4°, the pulse to 130 and respirations to 35, and he developed symptoms of pain, tightness and constriction in the chest, dyspnœa, and cough, with scanty sputum, the naked eye appearance of which was characteristic neither of pneumonic plague nor of croupous pneumonia. It appeared to be somewhat dark and rusty, with a layer of greenish mucus, almost purulent. Physical examination revealed isolated patches of dulness in the middle region on both sides. A culture of the sputum was then made and sent to Dr. Ghadially, the Municipal Analyst, who reported thrc

[17] Report of the Indian Plague Commission, 1902, p. 91, par. 203.
[18] Indian Med. Gaz., October, 1905.

days later the presence of Bacillus pestis and Bacillus pyocyaneus in the culture, a little scraping from which was inoculated subcutaneously into a white mouse, and it succumbed within forty-eight hours and plague bacilli were recovered from it. There was thus no room for doubt as to the nature of the case. The patient remained in a rather precarious condition for three days, but improved thereafter, the temperature, pulse, and respirations gradually became lower, and when the sputum, which had in the meantime altered its character, was reëxamined on the eleventh idem, the thirteenth day of illness, there were no plague bacilli, but Staphylococcus aureus only; some of the culture from the sputum inoculated into a mouse did not kill it. In the meanwhile infection from the bronchial glands had spread to the deep abdominal and mesenteric glands, and pain, fixation of the abdominal wall, and tympanites developed, the course being subacute, without much rise of temperature after the seventeenth day of illness. Recovery, however, was gradual. In another instance there was double infection from Bacillus pestis and the pneumococcus, and it ended in recovery. Recovery is thus possible in cases of very moderate mixed infection.

V. The Ambulatory Type. The mild type of plague recognized as *Pestis ambulans* is characterized by moderate fever lasting for a few hours to a day or two, one or more small and tender glands, and hardly any constitutional disturbance. The patient may or may not rest; he usually works; and if the buboes get gradually absorbed, there is no other trouble. Occasionally suppuration takes place, whereupon he resorts to the hospital for relief.

Such, in brief, are the various types of plague and their principle clinical features as observed at Bombay. Whether the affection presents essential differences in its types and symptoms in different parts of the world owing to climatic, racial, or other causes, I must leave to others better qualified than myself to say. In so far as Bombay or Western India is concerned, those types prevail, but with a distinct racial incidence and fatality, depending to a great extent upon social conditions and environments. The greatest sufferers with the heaviest mortality rate (76.57 per cent.) are the Hindoos; the Mahomedans come next (68.52 per cent.), closely followed by the native Christians (65.80 per cent.); and lastly the Parsees (50 per cent.)[19] Europeans and Eurasians suffer so little in comparison that their statistics are scarcely worth comparing. The racial incidence of attacks also presents corresponding differences.

[19] These figures relate to the actual case mortality rate recorded among 136,000 patients treated at the Arthur Road and Maratha Plague Hospitals during the twelve epidemics.

LEPROSY IN THE PHILIPPINE ISLANDS AND ITS TREATMENT.[1]

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Notwithstanding the comprehensive title of this paper, it is not my intention to discuss the history of leprosy in the Philippine Islands any farther back than the beginning of American occupation, because that is not essential to the practical questions under consideration, and it has already been done in one of my previous papers.[2]

At the beginning of the period with which this paper deals there were in the Philippines, according to the best record obtainable, between 3500 and 4000 lepers. While it is true that many of the reported cases were, on the application of more accurate diagnostic methods than had at first been employed, found not to be cases of leprosy, there were enough obscure cases overlooked to contraindicate any revision of the figures. There were never any grounds for the estimates ranging from 10,000 to 30,000, which were given circulation in the earlier days of the new regime.

The attempt at partial segregation of the worst cases, which in Spanish times had been begun and carried on by the church authorities as a matter of charity rather than legal proscription, was continued by the Americans, both before and after the establishment of civil government; but a systematic plan which had for its object the reduction of the number of new infections and the ultimate eradication of the disease from the Islands was not begun until 1905, although the island of Culion was selected for this purpose as early as 1901.

This island is well isolated, is roughly 20 by 40 miles in size, and is located about 240 miles from Manila in a southwesterly direction. The lepers are permitted to establish themselves at any place on the island, and if they desire to follow agricultural pursuits, the necessary animals and implements are provided at Government expense. In actual practice, however, it is found that they practically all live in the town of Culion, where everything is furnished for them by the Government. A large modern hospital has been established for those who are bedfast or those who desire to undergo special treatment. The town is located on an elevated site, has a modern water and sewage system, and is practically controlled by regulations and ordinances which the lepers themselves make. If

[1] Read by title at the sixth annual meeting of the American Society of Tropical Medicine, Washington, D. C., April 10, 1909.

[2] Leprosy in the Philippine Islands and the Present Methods of Combating the Disease, Medical Record, June 8, 1907.

the work of collecting lepers could have been rushed through with military rigidity, the problem would have been very much simplified, but it was deemed advisable to precede the collection of the lepers by a campaign of education and thereby secure the coöperation of the public rather than its opposition.

After this preliminary work, the plan adopted and still followed, which the geography of the country so eminently favored, consisted in removing all leprous patients from the well-isolated islands which contained only a few victims, and subsequently recanvassing the territory two or more times for cases which might have escaped, been overlooked, or which subsequently developed. By the method pursued the greatest amount of territory was freed in the shortest possible time. In military phraseology, the outposts were captured first and the lines gradually moved forward to the strongholds.

Experience has shown that when all of the native lepers have been removed from an island, the people of that island may be safely trusted with the duty of keeping foreign lepers from taking refuge there. The better points of human nature are being utilized in the campaign and often times prove valuable weapons.

Commitment to the Colony does not mean permanent separation from friends and relatives, since provision has been made for restricted visiting under proper safeguards from a neighboring island, on which the non-infected are permitted to camp or to live permanently if they desire, in order that they may be permitted to visit their friends and relatives. The law under which the policy of segregation is carried out probably vests more absolute power in the director of health than the laws of other states or countries. Briefly, he has authority to cause to be apprehended every person who is believed to be a leper, and to detain them if the bacillus of the disease can be demonstrated. Furthermore, it is the duty of all officials and others to report to the director of health any case of leprosy that comes to their knowledge. Failure to do so is punishable by fine.

The results of the segregation policy so far have been very encouraging. On February 1, 1909, according to official figures, there were only 2291 lepers in the Philippine Islands, which fact demonstrated that since May, 1905, when effective segregation was begun, the number of deaths has exceeded the number of new cases by 1409. The falling off in the number of new cases is one of the most encouraging evidences of success. It is estimated from the data on hand that under the old system there were about seven hundred new infections annually. During the past year there have not been more than 300 new cases, a difference of about 57 per cent. If these results can be brought about by incomplete segregation, it can readily be seen that the prospect of ultimately reducing the number to insignificant proportions is very encouraging.

In an address before the Philippine Islands Medical Association, more than two years ago, I held that whatever may be the views of

well-informed persons with regard to the communicability of leprosy, and however widely eminent medical men may differ upon this question, the incontrovertible fact remains that every leper who is capable of giving off lepra bacilli is at least one centre of infection, and that it is an utterly hopeless and useless undertaking to attempt to eradicate the disease without removing the source from which it is transmitted; and I asked that prophylactic medicine should not be turned aside by a few sentimentalists from its march to a goal which offered the magnificant hope of the complete eradication of this plague from the face of the earth and the saving of many innocent human beings who are now sacrificed annually to this most loathesome disease through sheer sentimentality. As more and more experience becomes available, these views are constantly strengthening, and it is believed that the cases which cannot be traced to another case are very few.

The most important factor now in connection with the work in the Philippines is early diagnosis. The earliest and most constant symptom observed is a nasal ulcer which is situated on the nasal septum at the junction of the cartilaginous and bony portion. Among 1200 cases taken in regular order at Culion, these ulcers were noted in 799. It is my opinion that if the remaining septums had been examined microscopically, scar tissue would have been found which would be strongly presumptive evidence that ulcers had existed previously. My experience in the examination of more than two thousand Philippine lepers and of those of Hawaii and of the Louisana colony near Bruns, confirms the importance and constancy of this sign, which may often be found long before there are any other objective or conscious subjective symptoms. Writers on leprosy give the incubation stage as from three weeks to twenty-seven years. No reliable data have become available here to prove or disprove this theory. Several Americans, whom it is fair to presume were not exposed to the disease in the United States, were found to have the disease within two years of their arrival in the Philippines.

Its extent and effect in different individuals or in the same patient at different times is very variable. I have seen a number of microscopically positive cases in which the only clinical symptom was a nasal ulcer, perhaps very obscure and innocent looking. Such cases usually come from houses in which advanced cases have existed. For this reason it is customary to have those who have been in contact with bad cases examined for this sign and scrapings submitted to a microscopic examination. The precautions enjoined at the leper institutions under the bureau of health is to protect the nasal mucous membrane as a first consideration, giving second place to, but not under-rating the old-time precautions. The membrane of the nose is easily protected by nasal plugs of sterilized absorbent cotton.

With regard to the types of the disease in the Philippine Islands, a fair estimate may be found from the fact that of 1200 cases studied

in the order of admission, 374 were of the anesthetic type, 157 of the tubercular type, and 650 partaking of the character of both, or mixed cases. It was observed that in a majority of cases the integrity of the eyebrows was disturbed. Among the 1200, the eyebrows had been entirely destroyed in 264 cases, and in 599 cases they were half gone. In the remainder of the cases there was generally some impairment of the appendages sufficient to constitute some degree of expressional deformity.

Treatment. As health officers, we are naturally more interested in preventing the disease and in the protection of the public than in the care or relief of the individual, yet no remedy within the realm of reason, whether physiological or empirical, has been neglected. Potassium iodide, mercury, creosote, salicylic acid, chaulmoogra oil, gurgon oil, leprol, nastin "B," experimental sera, and the *x*-rays have all been tried without curative effect, except the *x*-rays, which have proved successful in a few cases, but from our present experience can by no means be regarded as being suitable as a routine treatment. The cases in which they are apparently of value is in the early stage of the tubercular type in young and otherwise healthy subjects. The breaking down by the *x*-rays of one large, leprous granuloma of an individual usually results in marked coincident improvement in the granulomas situated in parts of the body far removed, and to which the *x*-rays have not been applied. One of the cases successfully treated by this method was recently reported.[3]

San Lazaro Hospital at Manila was the first institution in the world to use the *x*-ray treatment for leprosy with any degree of success, but this was probably due to the fact that there were a larger number of cases to choose from and the particular type which yielded to this form of treatment came under observation early. Chaulmoogra oil so far has not proved of value, but owing to the excellent results obtained in Louisana by Dyer with the crude oil, another thorough test of this treatment is being made.

Conclusions. 1. The experience in the Philipinnes during the past three years demonstrates that segregation has decreased the incidence of leprosy by over 50 per cent.

2. Of all the treatments tried, the *x*-rays is the only one which produced a cure, but as yet it is suitable only for specially selected cases.

[3] Preliminary Notes upon a Case of Leprosy apparently Cured with the *x*-Rays, Medical Record, October 31, 1908.

LUETIC BURSOPATHY OF VERNEUIL.

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Thirty-three years have elapsed since the elder Keyes[1] published in this journal one of the earliest articles on "Syphilis as Affecting the Bursæ." The affection had been first described by Verneuil,[2] who, in 1868, had reported four cases of "hydropisie des gaînes tendineuses des extenseurs des doigts dans la syphilis secondaire," and shortly after the appearance of this article Fournier had published a fairly complete account of the condition, stating that its occurrence was much more frequent than one would suppose from the silence of the literature on the subject. Verneuil's observation had also stimulated opposition; and in the same journal in which his publication appeared, Hérard[3] and Dechambre had written a reply, citing cases of "rheumatism," with the same tendinous lesions, but without the history or signs of syphilis. Still the condition had been soon pretty generally accepted as luetic in origin; and in a few years following Verneuil's second publication, several theses on the subject (Moreau[4] Chouet,[5] Voisin[6]) had appeared, in which a few additional cases were reported, and the new observations were coördinated with those of the early observers. In 1876, however, Keyes was able to collect only fourteen cases, including his own; and he wrote: "The literature of the subject seems wonderfully meagre in facts, so far as I have had access to it. It is to be hoped that further contributions may be forthcoming to make our knowledge more complete." This hope can hardly be said to have been realized. Finger,[7] in 1884, in an extensive article on the general subject of syphilis of the joints, tendons, etc., gave some attention to syphilitic bursitis; Buechler,[8] in 1889, reported four cases, two of them by no means convincing; and reports of occasional cases are found elsewhere. But, on the whole, the subject has received little attention; and it is striking that even in extensive articles on bursal affections—

[1] Syphilis as Affecting the Bursæ, Amer. Jour. Med. Sci., 1876, p. 349.

[2] L'hydropisie des gaînes tendineuses des extenseurs des doigts dans la syphilis secondaire, Gaz. hebd. de méd., 1868, No. 39, p. 609.

[3] Hydropisie des gaînes des tendons extenseurs, Gaz. hebd. de méd., Paris, 1868, No. 42, p. 662.

[4] Les affections syphilitiques tertiares des bourses séreuses, Paris Thesis, 1873.

[5] De la syphilis dans les bourses séreuses, articulaires, sous-cutanées et tendineuses, Paris Thesis, 1874.

[6] Contribution à l'étude des arthropathies syphilitiques, Paris Thesis, 1875.

[7] Zur Kenntnis der syphilitischen Gelenks, Sehnen, u.s.w., Wien. med. Presse, 1884, xxxiv, 864.

[8] Ueber Bursitis Luetica, Med. Monatschr., N. Y., 1889, 1, 393.

like those of Sonneborn[9] Schuchardt,[10] Schoenborn,[11] Tilger,[12] Unger,[13] and Graffelder[14]—no mention at all is made of luetic bursitis.

Quite as interesting is the silence of earlier writers—even of the acute British syphilographers—on the subject. Richet's classical monograph,[15] the first complete and successful description of the clinical features of syphilitic arthropathies, appeared in 1853; but in this work no separate account was taken of the bursæ. In his first case there was a small fluctuating tumor at the internal tuberosity of the tibia, not connecting with the joint, which was probably due to inflammation of the anserine bursa, though, in the presence of the overshadowing arthritis, it did not attract Richet's attention. Lancereaux,[16] who was the first to attempt to give to Richet's work a pathological basis, made no mention of the bursæ except in Observation II (a case of general syphilis and double arthritis of the knees), in which the "bursa of the rectus," which did not communicate with the joint, was found unaltered. In his text-book he described syphilis of the tendons and aponeuroses, but made no mention of the bursæ. The English surgeons interested in syphilis either overlooked syphilitic bursitis entirely or mentioned it in quite an offhand way, as an incident of the arthritis. Syme,[17] in a chapter on the bursæ mucosæ, said nothing of luetic effusions. Colles,[18] mentioned effusions into bursæ of the large joints in connection with the joint-effusions themselves, but did not treat the affections separately, and thought the bursal effusions due to the injudicious use of mercury. Abernethy[19] was similarly silent, though his Case XIV—in which a painful affection of the upper part of the tibia developed, attended with a collection of fluid under the fascia, the disease having started with indolent buboes—may possibly have been a case in point. Hunter[20] wrote: "We know also that the tendinous parts, when inflamed, give in some cases very considerable pain and that of the heavy kind, while in others they will swell considerably without giving any pain." Swellings of the tendons and fasciæ he treated incidentally with those of bones, but in the chapter, "Of Nodes on Tendons, Ligaments, and Fasciæ," he mentioned "obstinate and disagreeable swellings which neither gave way to medicine or

[9] Ueber Schleimbeutel Erkrankungen, Marburg Thesis, 1894.
[10] Tuberculose u. Syphilis der Sehnenscheiden, Archiv f. path. Anat., 1894, cxxxv, 3, 394.
[11] Ueber Bursitis u. Peribursitis, Würzburg Thesis, 1885.
[12] Zur Pathologie der chronischen Schleimbeutel Erkrankungen, Würzburg Thesis, 1890.
[13] Ueber Bursitis prepatellaris, Halle Thesis, 1887.
[14] Anatomie, Pathologie u. Therapie der Knieschleimbeutel, Würzburg Thesis, 1877.
[15] Mémoire sur les tumeurs blanches, Mém. Acad. de méd., 1853, xvii.
[16] Union méd., 1873, pp. 153 and 182; Traité de syphilis, 1866, p. 264.
[17] The Principles of Surgery, 1832.
[18] Practical Observations on the Venereal Disease and on the Use of Mercury, 1837.
[19] Surgical Observations on Diseases Resembling Syphilis, and on Diseases of the Urethra, 1822.
[20] Of the Lues Venerea.

time." Velpeau, in his *Lectures on Clinical Surgery*, recognized an extracapsular arthropathy for which he recommended inunctions with potassium iodide or even mercury; and for "white swelling" he used O'Brien's mercurial treatment, giving very large doses of calomel with opium. These were his results: "In arthropathies with hydrarthroma, without changes of the hard parts, and without spongy degeneration of the capsule, the disease improved rapidly and the exudate was entirely absorbed in from eight to fourteen days." It is not at all impossible that he was unknowingly treating lues.

The meagre state of the literature of syphilitic bursitis, both before and since Verneuil's time, gives to the recent occurrence of a case in the service of Dr. Halsted at the Johns Hopkins Hospital a peculiar interest. This is the only instance of the condition that, so far as I can find, has been observed in this hospital, and no similar case has since occurred in the skin and genito-urinary dispensaries, though we have been on the lookout for the condition.

The patient was a negress, aged twenty-nine years. She had been married four and one-half years, and had had no pregnancies. Her family and personal history were negative for tuberculosis. Her occupation was housework. She denied venereal infection. She had worked on her knees a good deal as a scrub-woman. About two and one-half years before admission she noticed a lump below the left patella, which caused her a good deal of discomfort when on her knees. About one and a half years later a similar swelling appeared on the right knee, but this was never painful. Two years before admission painless nodules appeared on the back of each olecranon, and about sixteen months later she first noticed a circinate eruption on the right forearm, soon followed by a similar lesion on the left arm. She presented, on admission, subcutaneous gummas of the forearms and lower legs, roughened tibiæ, the pigmented scars of old luetic lesions on the legs, and a circinate eruption of the forearms and thighs. The prepatellar bursa on each side was enlarged and contained fluid not under tension. The swellings measured about 4 x 4 cm., and their summits were elevated about 2 cm. above the skin surface. There was no sign of skin involvement, no tenderness, and the joints were quite normal. On the posterior surface of the right olecranon, about 1 cm. from its tip, there was a hard, not tender, movable nodule about 2.5 cm. in diameter, and a similar one 7 cm. below the tip of the olecranon on the left side. In the presence of the signs of tertiary syphilis, and of the symmetrical and indolent character of the bursal involvement, a diagnosis of syphilitic bursitis was made. The patient was put on bichloride of mercury ($\frac{1}{16}$ grain three times a day) and increasing doses of potassium iodide. In about one week the eruption had partly disappeared and the prepatellar bursæ were smaller. The patient left the hospital against advice; but continued her treat-

ment for about three weeks. She was next seen sixteen months later. The skin was quite free from the eruption, the subcutaneous gummas were gone, there were no signs of the retroölecranal swellings, and the prepatellar effusions had entirely disappeared. On the left side slight thickening of the tissues superficial to the patellar tendon remained, but there was absolutely no fluid present. The patient is still under observation and specific treatment is being continued. At present (nearly nineteen months after admission to the hospital) there is no sign of recurrence. The patient could not say when the swelling of the prepatellar bursæ entirely disappeared, but she stated that it was not later than four weeks after she left the hospital.

The clinical features of this case were similar to those in the original description given by Verneuil. Of his four cases (the first of which was seen in 1863), only one was reported in detail, but the others were said to be quite like it. In all of his patients the bursa involved was the one about the extensor tendons on the dorsal surface of the hand, distal to the annular ligament; and he remarked that the condition had, until then, been observed only in the hand, though "it must be sought for elsewhere." All his patients were women and all presented signs of secondary syphilis. In three of his cases the condition was symmetrical, in two there was a coincident luetic arthralgia. The effusion appeared suddenly, and formed a circumscribed tumor with no skin involvement. Tenderness was very slight and disability practically absent; the cure was rapid and easy. Not only the clinical course, but the response to treatment proved the luetic nature of the disease.

Keyes added to what Verneuil[21] had written, the clinical description of the bursitis of tertiary syphilis, and was able to gather twelve fairly well reported cases from the literature. His search was not perfectly thorough (the case of Labarthé,[22] for instance, he overlooked), and a few scattered cases have been reported since; so that I have collected all the cases published[23] and have tabulated their features.

Before discussing the clinical picture of the condition it is interesting to inquire into the structure and development of the bursæ mucosæ, particularly as light is thus thrown on their relation to the joints and incidentally on the pathology of bursitis. For the condition of syphilitic bursitis is, it is to be remembered, an affection of the bursæ mucosæ, *lying in the tela*, for the most part unconnected with the joints and, as we shall subsequently see, constituting, so

[21] Lésions syphilitiques tertiares des bourses séreuses sous-cutanées et tendineuses, Gaz. hebd. de méd., Paris, January 10, 1873; Gommes peri-articulaires, Jour. de méd. et de chir. prat., 1876, p. 398.

[22] Gomme syphilitique de la bourse séreuse, etc., France, méd., Paris, 1873, xx, p. 804.

[23] Two references—one to an article by Ingold and the other to a Munich thesis by Specht—I have been unable to find; it is possible that original cases are there reported, though there is no reference to them in the subsequent literature.

far as syphilis is concerned, organs quite independent of them. The analogy between the joints and the subcutaneous bursæ has probably been too readily assumed; and the pathological conditions which affect the one have been taken for granted for the other in a way which the facts do not warrant.

Anatomical Considerations. The bursæ were rather late to be discovered. The work of Monro,[24] published in 1788, was one of the early complete descriptions; but Albinus, in 1734, described sixteen pairs of shut sacs interposed between the tendons and bones, to which he gave the name of bursæ mucosæ.[25] Winslow had previously observed the bursal sheaths about the tendons at the wrist and the ankle; but the subject had been pretty generally overlooked by anatomists up to this time.

Once the bursæ were observed, an attempt was made to account for them; and in the absence of careful microscopic and embryological studies the attempts were not successful. Monro, on the basis of numerous gross anatomical and pathological analogies, regarded the bursæ and the joints as identical in structure; but most observers inclined to the view of Virchow that "the bursæ are in no sense true serous sacs, . . . but are rather places where the connective tissue, originally present *in continuo*, forms spaces by a process of atrophy and where these spaces come, in time, to be independent cavities."

An extensive investigation of the embryology of the bursæ mucosæ has been made by Retterer.[26] This observer recognizes five stages in the development of connective tissue: (1) In the earliest stage the primordial connective tissue consists of homogeneous protoplasm containing nuclei but no cellular units. (2) The first change is an appearance of fibrillæ in the ground substance. (3) Then begins an active mitosis; cells are formed and processes are sent out, constituting a network which encloses "hyaloplasm" in its meshes (mailles pleines). (4) The fibrillæ then become finer and smaller in number, the cells disappear, the hyaloplasm liquifies, and spaces result (mailles vides). (5) Finally, atrophy of the fibrillæ occurs and cavities are thus formed. The "serous" spaces are, therefore, *not* intercellular lymphatic spaces; they arise by evolution and solution from *all* the connective tissue elements. The connective tissue is, in the early state of rest, homogeneous; in the succeeding states the fibrillæ give it first a fibrillary, then an alveolar, and finally a reticulated structure.

Originally, the tendons are covered with connective tissue arranged in parallel layers, the homogeneous protoplasm lying about

[24] A Description of all the Bursæ Mucosæ of the Human Body, Edinburgh, 1788.

[25] This name—though manifestly inaccurate—has persisted, and is here used on account of its general acceptance.

[26] Sur la developpement morphologique et histologique des bourses muqueuses et des cavités péritendineuses, Jour. de l'anat. et physiol., 1896, xxxii, 256.

the parallel oval nuclei. This undergoes solution and intraprotoplasmic (*not* intercellular) spaces are formed. Next the tendon, the connective tissue sheath is continuous with the intratendinous connective tissue. The superficial lining "endothelial" cells of the bursæ are, therefore, of connective tissue origin; the cavity, which is formed by solution of the connective tissue, continues to be lined by persisting nuclei with their perinuclear protoplasmic zones; these "cells" continue to be united by fibrillary, protoplasmic prolongations, and so simulate a membrane.

The bursæ mucosæ are present in fœtal life and at a period when the muscles are still incapable of causing, at least marked, motion; for this reason they must not be regarded as of mechanical origin, though it is not improbable that motion plays some part in their later development. The time at which they appear varies with their site; in general, the deeper bursæ appear first. At the sixth month of fœtal life the subcutaneous bursæ are not yet present; but the skin at the site of the future bursa consists, at this stage, of a superficial firm layer containing hair follicles, sweat-glands, etc., and a deep layer of looser structure in which the bursæ develop. The retroölecranal subcutaneous bursa appears about the second month of life, but the bursæ of the lower extremity appear earlier; the sub-Achilles bursa, for example, in about the fourth or fifth fœtal month. Of the subcutaneous bursæ, those of the knee (and the subfascial prepatellar bursa is the earliest of these) appear first, the bursa of the calcaneus next, and the olecranal bursæ last (Dömény).[27]

The origin of the accidental or professional bursæ has always been an interesting question. In a vague sort of way they have been thought of as the result of repeated movement or insult. But the immediate result of such traumatism is, according to Retterer, *not* cavity formation, but cellular proliferation. The cavity is formed later by liquefaction, that is, cellular activity. A hygroma is therefore originally a nodule of connective tissue, and the cavity, in both hereditary and accidental bursæ, results from protoplasmic activity.

The chief point of dispute as regards the structure of the bursæ mucosæ concerns the existence of an epithelial lining; and it seems quite probable that the contradictory observations of various authors have resulted from the fact that the bursæ mucosæ vary greatly in their make-up with age, sex, habits, profession, previous diseases, etc. Their walls, at any rate, consist largely of connective tissue, in which three layers may be recognized (Delord).[28] The external layer, which is made up of interlaced fibers, is not sharply limited externally, is continuous with the surrounding connective tissue, and contains elastic fibers and fat cells. The middle layer is denser than

[27] Entwickelung u. Bau der Bursæ Mucosæ, Archiv f. Anat. u. Entwickgesch., 1897, p. 295.

[28] De la bourse séreuse rétro-olécrânienne sous-cutanée et de ses maladies. Paris Thesis, 1893.

the external, and the internal layer is densest of all, elastic fibers and fat being here absent. An apparent endothelial lining is certainly sometimes present, but it is by no means constantly seen; and the preponderance of evidence seems to favor the view that the nuclei present belong to the original connective tissue fibers. Within the bursal cavity connective tissue processes are often found normally, and this is to be regarded as a sign of their origin through lacunization, rather than by an orderly development.

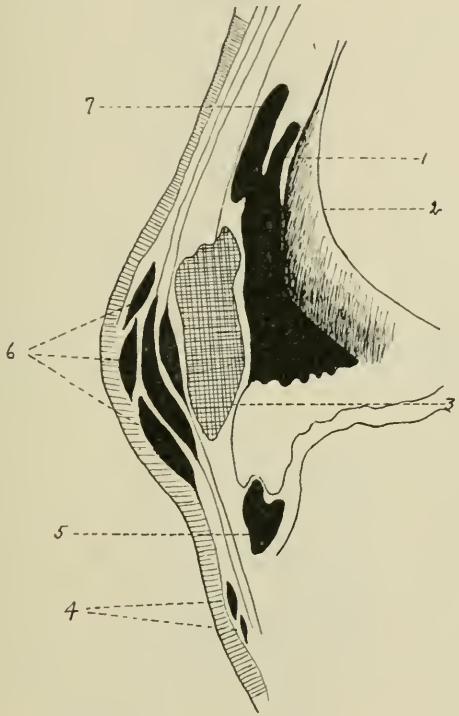

FIG. 1.—The bursæ mucosæ about the anterior aspect of the knee. (After Poirier.) 1, synovial prolongation; 2, femur; 3, patella; 4, pretibial bursa; 5, deep infrapatellar bursa; 6, superficial prepatellar bursæ (between these and the patella lie the median and deep prepatellar bursæ); 7, subtricipital bursa (in the diagram shown connecting with the joint cavity).

The chief bursæ of interest in connection with syphilitic bursopathies are those at the elbow, the wrist, the knee, and the ankle. The region of the knee is richly supplied with these structures, and their sites are illustrated in the accompanying sketches (Figs. 1, 2, and 3). Those of particular interest in connection with syphilis deserve special menton.

The Prepatellar Bursæ. Of these, there are three—the superficial or subcutaneous, the median or subaponeurotic, and the deep

or subfascial. Often only one of these is present, and when there is more than one they usually intercommunicate. The subcutaneous prepatellar bursæ (Fig. 1, 6), which were found by Bize in 88 per cent. of thirty-five cases, lie between the superficial fascia and the expansion of the fascia lata. Frequently they are multiple;

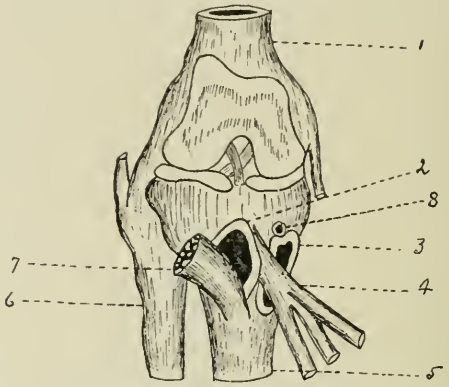

Fig. 2.—Bursæ about the knee. (After Debierre.) 1, femur; 2, pretibial bursa, 3, anserine bursa; 4, common tendon sartorius, gracilis, and semitendinosus; 5, tibia; 6, fibula; 7, ligamentum patellæ; 8, sartorial bursa.

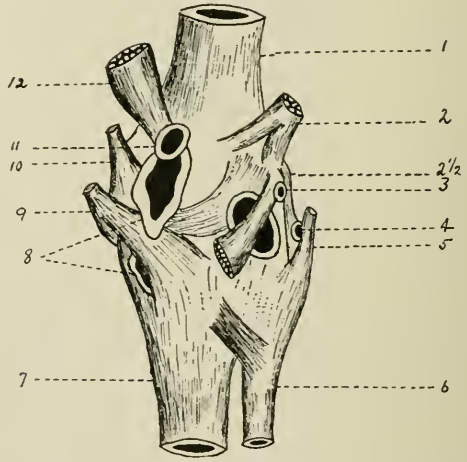

Fig. 3.—Bursæ about the posterior aspect of the knee. (After Debierre.) 1, femur; 2, gastrocnemius; 2½, external lateral ligament; 3, bursa of external lateral ligament; 4, bursa of biceps; 5, biceps; 6, fibula; 7, tibia; S, bursa of semimembranosus; 9, semimembranosus; 10, gracilis; 11, bursa of gastrocnemius; 12, gastrocnemius.

they often communicate with other bursæ but never with a joint. Their variations in number and dimensions often bear some relation to the profession of the subjects. Sometimes they contain the fatty foreign bodies observed by Luschka. The subaponeurotic (or

median) prepatellar bursa lies between the fascia lata and the fibrous expansion of the two vasti muscles. It is present in from 75 to 95 per cent. of the cases (Poirier[29] and Bize), and is always single. Sometimes it communicates with the superficial bursa, and occasionally with the deep. The subtendinous (or deep) prepatellar bursa is small, often absent, and lies between the lateral

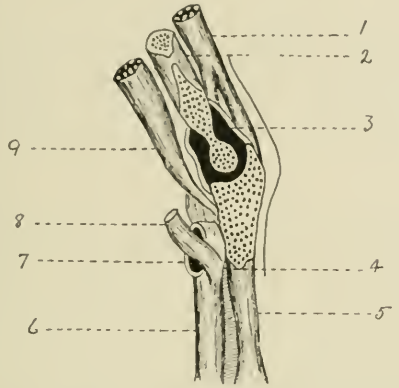

FIG. 4.—Bursæ about the elbow. (After Debierre.) 1, triceps; 2, humerus; 3, joint cavity; 4, bursa cubitalis interossea is frequently found at this site; 5, ulna; 6, radius; 7, bursa bicipitoradialis; 8, biceps; 9; brachialis anticus.

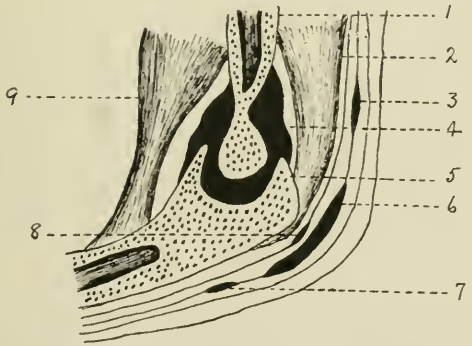

FIG. 5.—Bursæ about the elbow. (After Delord.) 1, humerus; 2, triceps; 3, supraolecranal bursa; 4, joint cavity; 5, ulna; 6, subcutaneous retroölcranal bursa; 7, infraolecranal bursa; 8, subaponeurotic retroölecranal bursa; 9, brachialis anticus.

muscular expansions of the quadriceps and the patella. The superficial subpatellar or pretibial bursa (Fig. 1, *4*) lies in front of the insertion of the patellar ligament into the tuberosity of the tibia. Its dimensions are variable and in about half of the cases it is replaced by a loose, areolar tissue. The deep subpatellar

[29] Bourses séreuses du genou, Arch. gén. de méd., 1886, pp. 539 and 694.

bursa, or bursa of the patellar ligament (Fig. 1, *5*) lies just above the insertion of this ligament, between its posterior surface and the bone. It is constant and its superior wall is usually in contact with a lobule of perisynovial fat. According to Poirier, it never communicates with the joint. The bursæ of the posterior and lateral aspects of the knee all lie in intimate relation with ligaments or tendons, and their sites are shown in the accompanying sketches (Figs. 2 and 3). Two almost constant bursæ, not represented in these drawings, are the inferior and superior bursæ of the internal lateral ligament, which lie between that ligament and the bone. The upper of these rarely, the lower never, communicates with the joint cavity. One of the lateral and posterior bursæ requires separate mention as of importance in connection with syphilitic bursopathies.

The Anserine Bursa (Fig. 2, *3*). This is the largest of the bursæ mucosæ at the internal lateral region of the knee, and has been, in two cases in which the site of the affection is carefully mentioned, involved in syphilis. The bursa was first described by Albinus. Its presence is constant, but it varies in size and form. It lies between the tibia (covered with the expansions of the internal lateral ligament) and the common tendon of sartorius, gracilis and semitendinosus. Within its cavity fibrous lamellæ are often found, dividing it into still smaller cavities; these indicate (Poirier) the origin of the anserine bursa by fusion of several bursæ belonging to each one of the three members of the common tendon.

Bursæ of the Elbow. These are shown in Figs. 4 and 5, and they all lie, as will be seen, in connection with the biceps and triceps muscles. Those at the posterior aspect of the elbow are of more importance in connection with syphilis than the well-known bicipitoradial bursa. They lie on two planes (Delord); that is, between the superficial fascia and the aponeurosis of the triceps (subcutaneous retroölecranal bursa, Fig. 5, *6*), or under this aponeurosis (subaponeurotic retroölecranal bursa, Fig. 5, *8*). The superficial bursæ lie behind, above, and below the olecranon (see Fig. 5), and may communicate with one another.

At the wrist and the ankle the chief bursæ of interest as regards the occurrence of syphilis are those in connection with the dorsal tendons. It was, indeed, a bursopathy in this site to which Verneuil first called attention.

Clinical Features. A thorough search of the literature reveals the reports of twenty-eight cases of syphilitic bursitis. The clinical features of these I have tabulated (see Table), and it is unnecessary to give their histories further. In two of these cases (Case XIII and Case XXII) there is room for considerable doubt as to the nature of the condition present. Moreau's patient (Case XIII) had a purulent bursitis (which is in itself strongly against its syphilitic origin); it was incised and cauterized, so that there is no evidence

that it yielded to the specific treatment also given; and it lasted four and one-half months, which is not the usual course of well-treated syphilitic bursitis. In Finger's patient (Case XXII) the history is not very complete, and it is not definitely stated that the disease originated in the bursæ. These two cases have been disregarded in the subsequent analysis. Several of the other reports are by no means complete; in two of them (Cases XXIII and XXIV) the patients passed out of observation before cure had occurred. These cases are, however, included in the analysis, as their syphilitic origin seems pretty well established. There are also in the literature other cases reported as luetic bursitis, the nature of which is too doubtful to warrant their inclusion in this table, but of which some mention should be made.

Verneuil reported a man with a syphilitic history who showed a symmetrical gummatous bursitis "in the neighborhood of the knee." There was no joint involvement. The lesion ulcerated. Neopolitan ointment was used locally and potassium iodide internally. The result is not stated. His third case, in the *Gazette hebdomadaire*, January, 1873, is also too briefly reported to pass muster. The patient was a woman with an ulcerating gumma at the internal condyle of one humerus, who showed the scars of an old, severe, ulcerating syphilide. There was no joint involvement. Nothing is said about the treatment or outcome of the case.

Keyes' Case VI (a woman, aged twenty-eight years, with an ulcerating syphilide on both knees and wrists, showing a fluctuating enlargement of both prepatellar bursæ which decreased in size under potassium iodide internally) may well have been one of bursal involvement by extension from the cutaneous lesions. In his Case VII treatment was not followed long enough to demonstrate the luetic origin of the condition. (The patient was a woman, aged fifty-two years, with a tubercular syphilide and an indolent swelling of both prepatellar bursæ.) The cases of Briddon and of Briddon and Taylor (cited by Keyes) are too doubtful to warrant consideration.

In the first case of Mauriac,[30] a man, aged thirty years, forty-two days after the appearance of a chancre, developed an acute and very painful fluctuating tenosynovitis of the extensor tendons of the hand. The case was regarded by Mauriac as one of syphilis, but all the signs and symptoms were those of a gonorrhœal tenosynovitis; and an adhesive involvement of the tendons persisted after the acute inflammation had subsided. This does not occur in syphilis. His second case was also undoubtedly one of gonorrhœa, though the possibility of syphilis was discussed.

[30] On Synovitis of Tendons Symptomatic of Syphilis and Gonorrhœa (translated by Cadell), Edinburgh Med. Jour., xxi, pp. 270 and 448.

Case reported by	Sex, age, occupation.	Evidences of syphilis.	Bursa involved. Form of bursitis.	Pain and other inflammatory signs	Joint involved. Functional disturbance.	Treatment.	Duration.		Remarks.
							Before treatment.	After treatment.	
1. Verneuil	Female	Secondaries present	Extensor com. dig. right wrist; hygroma	Slight pain and tenderness	Simultaneous arthritis shoulders and elbows; slight functional disturbance	Specific; kind and duration not stated	Not stated	Prompt cure	
2. Verneuil	Female; 34; dressmaker	Alopecia; mucous patches on vulva	Extensor com. dig. both wrists; hygroma; symmetrical	Slight pain; no others	Simultaneous pain in right arm and left foot; slight functional disturbance	Proto-iodide for 15 days	5 days	18 days	
3. Verneuil	Female	Definite secondaries	Extensor com. dig. both wrists; symmetrical; hygroma	Slight pain; no others	Simultaneous arthralgia (site not stated)	Specific; kind and duration not stated	Not stated	Prompt cure	
4. Verneuil	Female	Definite secondaries	Extensor com. dig. both wrists; hygroma; symmetrical	Slight pain	No joint involvement or functional disturbance	Specific; kind and duration not stated	Not stated	Prompt cure	
5. Verneuil	Male; 48; typesetter	Definite secondaries; bursitis; several mos. after chancre	Left retro-olecranal; hygroma	No pain or other signs	No joint involvement or functional disturbance	Mercury internally	1 week	1 month	
6. Finger	Male; physician	History of infection; secondaries; bursitis 4 months after chancre	Between rectus and suberurens, right side; acute inflammation with effusion	Pain and tenderness	No joint involvement or functional disturbance	Sodium iodide internally	Several days	About 10 days	
7. Buechler	Female; 20; housemaid	Mucous patches and secondary rash	Left prepatellar; "fluid tumor without pain"	No pain; slight thickening of skin	No joint involvement or functional disturbance	Biniodide of mercury and potassium iodide	4 days	22 days	
8. Lang	Male; 56	Chancre and roseola	Right semimembranosus; hygroma	No pain; slight tenderness	No joint involvement or functional disturbance	Specific; kind not stated	Not stated	4 weeks	
9. Chouet	Female; 18	Mucous patches and other secondaries	Left prepatellar; crepitating hyperæmia without effusion	Pain, tenderness and crepitati n	Similar condition in knee-joint	Mercurial inunctions; Gibert's syrup	15 days	18 days	An acute crepitating bursitis during syphilis; the only case of this kind in the series.

10. Verneuil	Male; 32; locksmith	Clinical features of tertiary syphilis 8½ years after chancre	Left prepatellar; gummatous bursitis	Slight pain; skin discolored, adherent, thickened, and ulcerated	No joint involvement; discomfort on hyperextension of knee and on kneeling	Local specific; mixed internally	4 months	3 weeks	
11. Verneuil	Male; law-student	Chancre 5 years previously; rode horseback a good deal	Under tendon of semitendinosus, both sides; "fungous" bursitis	Slight pain; no other signs of inflammation	No joint involvement; slight embarrassment in walking.	Mercurial inunctions; potassium iodide internally	Not stated	6 weeks	
12. Moreau	Female; 34	History of syphilis; fell on knee	Anserine bursa; gummatous bursitis	None	No joint involvement; slight functional disturbance	Mercurial inunctions; potassium iodide and proto-iodide internally	6 weeks	4 weeks	
*13. Moreau	Male; 64; baker	Luetic history; chancre 40 years previously; rupia present	Over left internal malleolus; purulent; later "fungous"	Tenderness and reddening of skin	.....	Incision and cauterization; mixed treatment internally	Not stated	4½ months	
14. Moreau	Male; 33	Chancre 6½ yrs. previously	Dorsal surface of little toe; metatarsophalangeal joint great toe; left side; ulcerating	Tenderness and ulceration	No joint involvement; functional disturbance	Potass. iodide; proto-iodide; sparadrap of Vigo	About 12 months	3½ weeks	Simulated perforating ulcer.
15. Moreau	Male; 33; laborer	History of lues; luetic periostitis present	Palm of hand, level second metacarpal gummatous	Skin reddened; no pain	No joint involvement or functional disturbance	Potass. iodide internally; Neapolitan ointment	Not stated	3 weeks	
16. Moreau	Male; 34	Chancre 7 years previously	Right retroölecranal; gummatous, then purulent, then fungating	Skin ulcerated after incision	No joint involvement; marked functional disturbance	Potass. iodide internally; Vigo plaster	8 years	Few weeks	
17. Keyes	Female; 48; housewife	Chancre 2½ yrs. previously	Both prepatellar; gummatous	No pain; slight adherence of skin to old scars of eruption	No joint involvement or functional disturbance	Potass. iodide	8 years	5 weeks	
18. Keyes	Female; 30; scrub-woman	Chancre 5 years previously	Both prepatellar; gummatous and ulcerating	No pain; reddening and ulceration of skin	No joint involvement or functional disturbance	"Antisyphilitic"	Not stated	4 months	
19. Keyes	Female; 33;	Iritis; mucous patches	Prepatellar (side?); gummatous and ulcerating	Slight pain; ulceration of skin	Slight stiffening of joint; osteoscopic pains	Not stated	Not stated	Not stated	These bursopathies had been present before and had disappeared under treatment.

Case reported by	Sex, age, occupation.	Evidences of syphilis.	Bursa involved. Form of bursitis.	Pain and other inflammatory signs.	Joint involved. Functional disturbance.	Treatment.	Duration.		Remarks.
							Before treatment.	After treatment.	
20. Keyes	Female; 40; housewife	Luetic history; miscarriages; nodules on shins	Both prepatellar; bursæ "thickened"	Slight pain; no other signs	Some interference with walking; no joint involvement	Not stated	Not stated	Not stated	These bursopathics had been present before and had disappeared under treatment.
21. Keyes	Female; 24; housewife	Chancre 18 mos. previously; secondaries present	Right prepatellar; acute inflammation with effusion	Pain; skin tender, red, and hot	Interference with walking; joint not involved	Potass. iodide	6 weeks	Prompt improvement	An acute inflammation which promptly cleared up under specific treatment.
*22. Finger	Female; 41	Signs of lues, but no history of it	Left prepatellar and over left coracoid; gummatous, ulcerating bursitis	No pain; skin discolored and ulcerated	No joint involvement	Kind not stated	3 years	2 months	
*23. Buechler	Female; 28; housewife	History of lues; gummata ecthyma; chancre 3 years before	Both pre- and infrapatellars; "hard, board-like tumor"	None	None	Potass. iodide	.....	Case not followed to cure	
*24. Buechler	Female; 35; housewife	Gummata present	Right prepatellar; "hard fibrous"	Pain; no others	None	Potass. iodide	Not stated	Not stated	Case passed out of observation; ulcerated under treatment.
25. Buechler	Male; 34; merchant	Chancre 31 mos. previously; syphilides (tertiary)	Right subpatellar; "elastic pseudofluctuating"	None	Slight stiffness of joint	Potass. iodide	Not stated	4 weeks	Chancre (as in other cases reported by Buechler) had been treated. The bursa healed readily, but the other syphilitic manifestations were most obstinate.
26. Labarthé	Male; 27; joiner	Chancre 7 years previously	Radial border, first phalanx fore-finger right hand; gummatous	No pain; skin slightly discolored	None	Mercurial inunctions; potass. iodide internally	3½ months	About 1 month	A bursa often exists at this site in joiners, but Labarthé excluded professional hygroma on the absence of pain, tenderness, and fluctuation.
27. Churchman	Female; 29; housewife	Gummata and circinate syphilide	Both prepatellar (hygromata) right retroöle-cranal (gummatous)	Slight pain in left knee when kneeling; no other signs	None	Bichloride of mercury and potass. iodide internally	2½ years	About 1 month	The exact date of disappearance of the bursitis patient could not state.

NOTE.—The asterisk indicates a doubt as to the syphilitic nature of the bursitis. Cases 13 and 22, though reported as syphilitic, can not be so regarded with any certainty. Cases 23 and 24 were probably syphilitic, though they were not followed until cure had taken place.

Voisin (Observation II) mentions a case of right tibiotarsal subacute syphilitic arthritis with tenosynovitis "des orteils" on the same side. No further data are given.

Gosselin[31] reported a case (Observation II) of an hysterical girl with a luetic history who developed a flexion of the knee and tenderness at the internal tuberosity. There was a fine crepitation present in the prepatellar bursa, which could be elicited by causing the skin to slide over the patella with the palm of the hand. The possibility of its luetic nature was discussed, but no conclusion was reached, and the ultimate fate of the patient is not stated. Volkman[32] describes a case of bursitis which he treated by a Schmierkur, but says nothing of its luetic nature. Zeissl mentions a case of bursitis of the left knee with a large effusion, which disappeared under antiluetic treatment.

The clinical features of the twenty-six certain cases here tabulated may be analyzed as follows:

Stage of the Syphilis. Nine of the cases occurred in the secondary, seventeen in the tertiary stage. The date of the occurrence of the lesion after the chancre varied from four months to eight and a half years.

Sex. Sixteen of the patients were women, ten were men. In twelve of the females the bursæ about the knee were involved—the anserine bursa once, the prepatellar bursæ eleven times. In the remaining four (the original cases of Verneuil) the extensors of the fingers were the seat of the trouble. In the males there was no such preponderance of prepatellar involvement. The anserine bursa was involved once, the semimembranous bursa once, the subpatellar once, the prepatellar once, and the bursa between the rectus and subcrureus once.

Age. The patients varied between eighteen and sixty-four years of age. The average age was about thirty-four years.

Profession of the Patient and History of Trauma. In 17 cases either there is no mention of trauma or it is distinctly stated that none had occurred. Of the 11 prepatellar cases in women, those who reported the cases could cite trauma as the exciting cause of the bursitis in only 3 patients. Of these 11 women, 9 did housework; the occupation of 2 is not stated. In some of the cases connection with trauma as an exciting cause was definite. Case XI, for instance, developed a luetic bursitis in the anserine bursa after excessive horseback riding; Case XXVI developed the condition in one of the professional bursæ of joiners. It is to be remembered that the affection is essentially indolent and that the patients would therefore be unlikely to connect it with previous insult, which might account for the absence of traumatic history in some of the cases. It is certain that the disease develops most often in bursæ most

[31] Archives générales de médecine.
[32] Berlin klin. Woch., 1876, No. 8.

exposed to trauma—one of its features which corresponds with the general habits of syphilis.

Pain was present in 16 cases, absent in 10, great in only 1 (Case IX). In the great majority of the cases it was an almost negligible feature. Functional disturbance was never marked, and in many cases was entirely absent.

Signs of Inflammation. In 15 cases there were none. Tenderness and discoloration of the skin were occasionally observed, and ulceration was present in six cases. Three cases showed definite acute inflammatory signs. In Case XVIII the skin over the left prepatellar bursa reddened, and ulceration took place. In Case XXI there was pain and tenderness in the bursa, with rise of local temperature.[33] Case IX showed a typical crepitating tenosynovitis, with pain and tenderness. In Case VI the condition was described as an "acute inflammation with effusion," and tenderness was present.

Joint Involvement. The joint neighboring on the involved bursa was involved only once (Case IX); here a condition similar to the bursitis existed in the knee. Three others showed co-existent arthralgia in distant joints; and one suffered from osteocopic pains. In general, joint involvement was conspicuous by its absence. This is quite at variance with the statement of Lang and Ullmann[34] that syphilitic affections of tendon sheaths, tendons, and bursæ are often met with in connection with joint changes; and with that of Voisin,[35] that "in the secondary stage, a subacute arthritis occurs which may involve many joints and *many subcutaneous serous bursæ or tendon sheaths at the same time.*"

The bursitis was *symmetrical* in nine cases, multiple in two.

Duration. Before treatment was begun the duration of the bursitis varied from four days to eight years. Treatment was almost always promptly effectual; in one case the bursitis persisted five months, but the disease disappeared on the average forty-six days after treatment was started.

Bursæ Involved.[36] The site of the bursitis was as follows:

[33] The pain in this case was regarded by Keyes, who reported it, as due in part to an hysterical hyperesthesia.

[34] Ergebnisse der allg. Path. u. Path. Anat., iii, 306.

[35] Loc. cit.

[36] Several other cases of *tendon* involvement are recorded by Chouet, Fournier, and Schuchardt. As these are cases of syphilitic tenosynovitis and not of bursitis, they are not included in the table. The tendons affected were the extensor longus digitorum, peroneus tertius, biceps femoris, biceps brachialis, etc.

Prepatellar bursa:		
Right	4	
Left	3	
Both	5	
Side not stated	1	13
Subpatellar bursa		2
Semimembranosus bursa		1
Between rectus and crureus		1
Anserine bursa		2
Bursa of extens. comm. dig.		4
Bursa of extensors of toes		1
Bursa of flexors of fingers		1
Bursa at radial border first phalanx forefinger		1

Form of Bursitis. Simple hygroma occurred eight times, only once (the author's case) in the tertiary stage. Gummatous bursitis was present ten times; ulcerating or fungous bursitis five times, acute inflammatory bursitis three times.

The picture, then, is one of an indolent affection of the bursæ, involving most often the knees—particularly in women. The disease is quite independent of syphilitic arthritis, the bursæ involved being oftenest those unconnected with the joints, and the neighboring joints themselves being entirely free from involvement.[37] *In the secondary stage simple hygroma is frequent; in the tertiary stage, gummatous, ulcerating, and fungous forms occur. The bursæ involved are those most exposed to trauma; but a study of the cases makes it clear that trauma, as usual in syphilis, only determines the site which the disease will occupy. There is little or no accompanying functional disability; and specific treatment leads to a prompt and permanent cure.*[38] *In view of the marked indolence of the condition and its great similarity to the arthropathies of syphilis, it should be spoken of as luetic bursopathy (of Verneuil)—the possibility of the rare occurrence of more acute symptoms being, however, kept in mind.*

Diagnosis. The diagnosis rests on the following factors: (1) Previous history or co-existent signs of syphilis. (2) Spontaneous development of bursopathy, without particular traumatic history. (3) Slow evolution and chronic course. (4) Marked absence of pain, tenderness, and functional disability. (5) Symmetry of the bursopathy. (6) Site. An indolent bursopathy of the retroölecranal or prepatellar bursa—particularly if symmetricaal—should always arouse suspicion. (7) Absence of other causes of bursal enlargement (tuberculosis, trauma, hysteria, etc.). (8) Absence of joint involvement. (9) Inefficiency of non-specific, and complete efficiency of specific treatment.

Differential Diagnosis. An etiological tabulation of bursitis—constructed from the reported cases—may be of value in this connection:

[37] To this there is one doubtful exception in the table.

[38] In one case reported by Buechler the bursopathy promptly disappeared under treatment, but the other signs of syphilis persisted.

1. Traumatic bursitis:
   (*a*) Acute.
      I. Simple congestive bursitis.
      II. With effusion.
      III. With bloody effusion.
   (*b*) Chronic.
      I. Simple serous.
      II. Proliferating.
      III. Calcareous.
2. Inflammatory bursitis by propagation from neighboring infections.
3. Bursitis due to diatheses (many of these cases were mentioned by the earlier writers; quite possibly a number of them were syphilitic):
   (*a*) Rheumatism.
   (*b*) Gout.
   (*c*) Saturnism.
   (*d*) Hydrargyrism.
   (*e*) Scurvy.
   (*f*) Cholera.
4. Infectious bursitis:
   (*a*) Tuberculosis.
      I. Serous.
      II. Caseous.
      III. Fungous.
      IV. Accompanied by rice bodies.
   (*b*) Typhoid fever.
   (*c*) Pneumonia.
   (*d*) Mumps.
   (*e*) Grip.
   (*f*) Syphilis.
      I. Secondary.
         Congestive.
         Effusive.
      II. Tertiary.
         Gummatous.
         Ulcerating.
         Fungous.
         Effusive (rare).
   (*g*) Gonorrhœa.
   (*h*) Pyohemia.
   (*i*) Erysipelas.
5. Professional bursitis.
6. Positional bursitis (seen in epilepsy, long invalidism, in deformities of the extremities, etc.).

From the acute inflammatory cases syphilitic bursopathy is distinguished by its indolent character, its insidious onset and

chronic course, and its prompt response to specific treatment. From tuberculosis the diagnosis, particularly in the ulcerative cases, may be extremely difficult. I have recently seen a case of right prepateller bursitis in a colored woman, with thickening of the bursa and ulceration. The case was regarded as one of tuberculosis, and the bursa was excised. The ulcerations were certainly very suggestive of syphilis, and at operation the gross appearance was that of gummatous bursitis. Microscopic examination showed a chronic inflammatory bursitis without tubercles. The sections were stained for spirochetæ, and none were found. No tuberculin had been given, and the history was perfectly negative for syphilis; for these reasons the case is not reported as a gummatous bursitis, and is only mentioned as illustrating the difficulty of distinguishing between tuberculosis and syphilis. The diagnosis, however, should be made if the obvious steps are taken (tuberculin reaction, examination of smears for spirochetæ).

Professional bursopathies—particularly housemaid's knee—may simulate syphilitic bursopathy in all respects, and the diagnosis will often rest entirely on the therapeutic test.

Osteitis and periostitis may simulate the rare cases of acute syphilitic bursitis. If careful attention, however, is paid to the exact seat of the condition and the anatomical situation of the bursæ is kept in mind, the diagnosis can usually be made.

When the involved bursa is situated over the tendons of the extremities, the appearance may suggest a perforating ulcer. This occurred in Case XIV, reported by Moreau.

Pathology. The practically complete absence of postmortem and microscopic examinations makes it necessary to study the pathology of luetic bursopathies entirely from the clinical features. The disease occurs in the following forms:

1. Luetic bursopathy accompanying luetic arthopathy. Here the effusion of "syphilitic white swelling" spreads into bursæ which communicate with the joint (*e. g.*, the subquadriceps bursa), and the condition is simply a complication of the joint involvement. As such, it is quite unimportant; the wonder is that it does not occur oftener. But more curious still is the fact that, in connection with luetic arthropathies, the true bursæ mucosæ (unconnected with the joint) are almost never—so far as the evidence in the literature goes—involved in the disease.[39] Luetic bursopathy is an independent affection.

2. Luetic bursopathy, independent of luetic arthropathy. This occurs in three forms.

(*a*) *Congestive Bursitis.* Fournier has insisted that the vague "joint" pains so frequent in syphilis are often due to involvement

[39] There is one autopsy on record, made by Lancereaux, in which, in connection with a syphilitic knee-joint, the "pretibial bursa was occupied by a gummatous deposit." It is possible that such a finding is not rare, though the literature is silent on the point.

of the bursæ of tendons, and for the reason that careful examination of the site of the pain, of the movements which elicit it, and of the direction along which it is transmitted, often establishes its independence of the joint and points to its seat in the tendons. An acute syphilitic tendovaginitis undoubtedly exists. More nearly related, however, to the true luetic bursopathies are the rare cases of acute inflammation of bursæ mucosæ (see Cases IX and XXI), in which the signs are those of congestion with slight effusion.

(*b*) *Hydrops or Hygroma.* This is the most frequent form of luetic bursopathy in secondary syphilis; but the condition offers no special pathology in syphilis. The "effusion" consists of a yellowish, viscid, cloudy, coagulable fluid. No examination of it has ever been made for spirochetæ. Is it inflammatory in origin? Langemak[40] in giving an account of the origin of hygromas, states that the contents of the hydropic bursæ consists of fibrinoid,[41] which results from the solution of the previously undissolved albuminoid collagen, which has been heaped up in the bursa as a result of arterial hyperemia. The origin of hygromas is to be found in the liquefaction of increased connective tissue. This, indeed, is the view held by many investigators. The process begins, according to Schuchardt[42] with a hyperplasia of the fixed connective tissue elements. Numerous mitotic figures appear in the connective tissue cells, the cell bodies swell to an enormous size, and thus large multiform (but usually spindle-shaped) cells are formed. The cellular swelling continues, usually with loss of the nuclei, and peculiar amorphous vitreous structures result. This process is accompanied by a secretion of "Faserstoff," partly within the altered connective tissue cells, partly between them—but always within the tissue itself, and never on the free surface of a space. In the case of the bursæ mucosæ the inflammatory process takes place in the connective tissue surrounding the structure. There is no exudation from the free surface of the bursa. Ricker[43] has reached similar conclusions. The connective tissue elements which immediately precedes the liquefaction he finds to stain yellow, not red, with van Gieson's stain. The fluid comes from a change in the connective tissue, and not from the bloodvessels. All the products of degeneration mix with the liquid and cause further solution. Two types of liquefaction occur—the ganglion or gelatinous type and the hygroma type. These processes are essentially similar, but slightly different in detail. Even in the so-called inflammatory hygromas the process is one of degenerative liquefaction.

[40] Die Entstehung der Hygrome, Archiv für klin. Chir., 1903, lxx, 946. Nachtrag to the above, Ibid., lxxi, 268.

[41] "Fibrinoid" was the term used by Neumann to describe a degeneration product of connective tissue which stains yellow (*not* red) with van Gieson's stain, and is thought to be a transition form between fibrillary connective tissue and fluid.

[42] Die Entstehung der subkutanen Hygrome, Virchow's Archiv, 1890, cxxi, 305.

[43] Die Verflüssigung der Bindegewebsfasern, Virchow's Archiv, 1901, clxiii, 44.

Ritschl,[44] studying the pathogenesis of the ganglion, reached the similar view—previously advanced by Ledderhose—that the condition is one of degeneration and not of inflammation. Contrary views find expression in the literature;[45] but the great majority of the best work is in line with the degenerative theory of the origin of hygromas.

It is obvious that in these descriptions of the formation of hygromas it is not explained just how liquefaction takes place or what stimulates it; and it is not easy to conceive of such a process as entirely spontaneous. The important points seem, however, to be that the process is quite different from that of inflammation as ordinarily understood; that the fluid produced is unlike the usual exudates; that the connective tissue bears an important relation to its production; and that degeneration plays an essential role in the process.

(*c*) *Gumma of the Bursa.* This is the most frequent form of the disease in the tertiary stage. Frequently it goes on to ulceration. In Case XVIII both prepatellar bursæ were involved in a gummatous process; the right bursa ulcerated, but on the left side the affection was kept in check by treatment and no ulceration occurred. The possibility that ulcerative syphilitic processes at the site of a bursa really originated in broken-down subcutaneous gummas, or even in periosteum or bone, must, of course, be kept in mind. This was probably the fact in Case VI of Keyes' table, and I have not included the case in my tabulation.

Luetic Bursophathies in Congenital Syphilis. To this subject, so far as I have been able to find, no attention whatever has been called in the literature; and, although there are intimations that the disease exists, convincing evidence is wanting.[46] This seems all the more strange in view of the fact that congenital syphilis causes in the joints themselves a condition (Clutton's disease) exactly similar, clinically, to the luetic bursopathies of acquired syphilis. Undoubtedly the spread of a joint effusion into the communicating bursa often occurs, is considered incidental, and is not mentioned by

[44] Beitrag zur Pathogenese der Ganglien, Beiträge zur klin. Chir., 1895, xiv, p. 557.

[45] See particularly the article of Riese: Die Reiskörperchen in tuberkulös-erkrankten Synovialsäcke, Deutsche Zeitschrift für Chirurgie, 1896, xlii, 1 This author writes: "All rice bodies in tubercular joints, tendon sheaths, and bursæ are in the last analysis, derived from a fibrin coagulation. Part of this results from coagula which are first formed in the synovial fluid, another part from deposits which are laid down on the wall of the synovial sac and have become partially organized by means of its cells." See also Kreuter: Ein Fall von Bursitis Subdeltoidea als Beitrag zur Hygromfrage, Deutsche Zeitsch. f. Chir., 1904, lxxii, 136. This author found in *his* case *no* fibrinoid in the sense of Neumann, but a lining of granulation tissue, rich in vessels; and the fluid present came, he said, from the blood. The condition was one, *i. e.*, of inflammation and not of degeneration.

[46] Lang and Ullmann (Ergebnisse der allgemeinen Pathologie u. Pathologischen Anatomie, 1896, Jahrg. iii, 307) quote Finger and Lang as authority for the statement that chronic hydrops of the tendon sheaths occurs in hereditary syphilis and may reach a high grade of development. They do not mention the bursæ mucosæ, and the literature is elsewhere similarly silent.

the reporter. In Dureuil's thesis,[47] for instance, a photograph of such a case is reproduced in which a communicating subquadriceps bursa is distended from a joint effusion, but no mention of bursæ is made in the text. It is striking that Clutton, in reporting his eleven cases of symmetrical synovitis of the knee, is equally silent. It seems hardly likely that if the bursæ about the knee, not connecting with the joint, *had* been involved they would have escaped his notice; and he certainly would not have failed to mention their involvement if he had observed it.

Diard,[48] reporting fifty cases of gumma in hereditary syphilis, makes no mention of the bursæ, but two of his cases deserve attention:

Observation XXV. (Case of Fournier at St. Louis Hospital.) A girl, aged twenty-five years, a virgin, with no signs of acquired syphilis. Patient's sister luetic. At sixteen, the patient developed a perforation of the palate; at eighteen, a large gumma appeared on the left shoulder, destroying part of the deltoid, down to the humerus, which could be felt beneath the skin. The lesion partially healed, but later recurred in situ. There were other signs of hereditary syphilis. Diard makes no mention of the bursa in this case; but the condition may well have started in the subdeltoid bursa.

Observation XLII. (Case of Leloir and Perrin, Annales de dermat., 1883.) Patient had a family history of lues. At fourteen, keratitis developed. At sixteen, a swelling appeared in the right popliteal space, as large as a walnut, not painful, sharply demarcated, and not adherent to the skin. The subsequent course is not stated. Here, too, there is nothing said about bursæ; but a bursa may quite well have been the site of the affection.

Three possible cases are also reported in the thesis of Saint Pierre.[49]

Case V (reported by Guterbock). A girl, aged six months, with cachexia and desquamation of the soles of the feet, presented large swellings about the elbows, which ulcerated, forming fistulas which led directly to the denuded humerus. On the anterior surface of the left tibia—about at the site of the pretibial bursa—there was a fluctuating tumor, thought to be a subperiosteal abscess.

Observation XVI. A man, aged twenty-seven years, presented a marked hydrarthrosis of the right knee, and the note is made that the patella was "notablement élargie."

Observation XIX. Double hydrarthrosis of the knees; "les culs-de-sac de la synoviale son largement distendus."

With the exception of these vague references, bursopathies receive no attention in the extensive literature of congenital syphilis. In

[47] Contribution à l'étude des pseudo-tumeurs blanches syphilitiques, Paris Thesis, 1880, No. 148.

[48] Les manifestations gommeuses de la syphilis hérèditaire tardive, Paris Thesis, 1901, No. 548.

[49] Contribution à l'étude des arthropathies de la syphilis hérèditaire tardive, Lyon Thesis, 1900, No. 107.

Fournier's lectures[50] no cases are mentioned, unless the bursæ were involved in some of the patients rather vaguely referred to as cases of "pseudo-tumeurs-blanches syphilitiques." Somma[51] does not include bursopathies in his comprehensive list of the effects of hereditary lues; nor does Ranguedat[52] mention them. Either the condition has been overlooked, or the striking fact is that though essential hydrarthrosis is a distinctive characteristic of congenital syphilis, essential bursal hygroma—which occurs in the acquired disease—is never seen in the hereditary form.

Allied Conditions. Certain effects of syphilis in the joints themselves or in the peri-articular structures present a clinical picture so similar to that of syphilitic bursopathy that some account should be taken of them in this connection.

Syphilitic Arthropathies. Mention has already been made of the great clinical similarity between syphilis of the joints and syphilis of the bursæ; and of the striking fact that when the disease affects one it does not, as a rule, affect the other. Special account should, however, be taken of the symmetrical arthropathies of congenital syphilis. This disease has been particularly described by Clutton[53] who in his first paper reported eleven cases—all effecting the knee. The disease occurred between the eighth and fifteenth years, and was characterized by:

1. Its symmetry.
2. Freedom from pain.
3. Long duration.
4. Free passive mobility of the joints throughout the disease.
5. Co-existent signs of hereditary syphilis.

Attention was usually called to the knees by stiffness in one of them, which on examination was found to contain fluid; fluid was also present in the other knee, which after an interval of some months became similarly affected. The swelling was a flaccid one—due to fluid not under tension. Careful examination showed the joints other than the knees to be normal. Every case showed two or more signs of the Hutchinsonian triad. The swellings remained without alterations for several months; but specific treatment brought about a perfect cure—though not so rapid a one as in acquired syphilis. One case gave an acute history following a fall. Nine of the eleven cases were suffering from an active keratitis, and the following similarities between the two conditions were dwelt upon by Clutton: (1) Both knees and both eyes are affected, with an interval between the onset of the disease in the two sides. (2) The disease is chronic

[50] Leçons sur la syphilis hérèd. tardive, Paris, 1896.

[51] Su di una forma morbosa articolare per sifilide ereditaria, Gior. internaz. de sc. med. Napoli, 1882, n. s., iv, 840.

[52] Des arthropathies dans la syphilis héréditaire, Paris Thesis, 1883, No. 356.

[53] Symmetrical Synovitis of the Knee in Hereditary Syphilis, Lancet, February 27, 1886, p. 391.

in both cases. (3) In neither case does it produce any destructive changes. (4) In both cases a relapse is frequent, and neither condition is strikingly amenable to treatment. (5) The two conditions occur at about the same period of life.

Intermittent Hydrops. The great similarity between the clinical picture of this disease and that of spyhilitic hygroma of the bursæ suggests that certain of the symptomatic cases of joint hydrops may have been luetic in origin. Friedenberg has reported cases definitely due to syphilis; and it would seem wise, in all cases in which the etiology is not clear, to apply the therapeutic test.

Bicipital Serositis. This obscure condition—described by Notta, of Lisieux, under the name of contraction of the biceps—has been observed by several authors and its syphilitic nature asserted. Finger says positively that a tenosynovitis of the tendon of the biceps is particularly characteristic of secondary lues, and that it accounts for many of the vague pains felt at the elbow, the real site of which will be found on careful examination to be the bicipital tendon. And Fournier lays great stress on pain at the bend of the elbow with inability to extend the forearm as characteristic of secondary syphilis. But this syphilitic tenosynovitis, which is quite frequent, is different from the other rarer syphilitic affection at this site, which has its real seat in the bicipitoradial bursa. The condition is characterized by pain and tenderness in the bicipital tendon. The forearm is held slightly flexed and there is difficulty in extension. There is also and most characteristically painful embarrassment of simultaneous flexion of the forearm with supination of the hand. Complete pronation is impossible. The position of the arm and the interference with motion, so far as it is a mechanical affair, have been shown, by injection of the bicipitoradial bursa in the cadaver, to be due to effusion into this bursa. It is interesting that this condition was described by Colles,[54] who did not, however, lay any particular stress on it. "A very common form," he wrote, "of these affections" (joint and bursal effusions) "is observed in the elbow-joint; the patient has not full power of it, he cannot fully flex it, nor extend it much beyond a right angle; very rarely is there any pain or any tenderness from pressure when made upon the prominent parts of the joint. Not infrequently this affection of the joint is caused by effusion into some of the adjacent bursæ; but in all these cases we feel the tendon of the biceps extremely rigid, yet not swollen nor tender to the touch." Distention at this site may effect the radial nerve, as in a case reported by Walther, in which a radial paralysis, thought to be due to a neuroma, was found to be caused by a fibrolipoma of the bursa. It is probable that in some of the cases the cubitoradial bursa of Monro (lying between the tendon of the biceps and tuberosity of radius, and the external border of brachialis anticus

[54] Loc. cit.

tendon and fasciæ of flexor sublimis digitorum) is the one affected. In a case reported by Rieffel[55] there was difficulty of flexion of the forearm, and a walnut-sized tumor appeared at the site of the bicipito-radial bursa. There had been no trauma, and the swelling, which was free from tenderness, appeared suddenly. It was impossible to establish its luetic origin, and it was excised. It contained sero-sanguineous fluid. Weinlechner[56] reported a case of extensive swelling of this bursa—indolent in itself, but causing great interference with motion—which he excised. There is no note as to its luetic origin. Mauriac calls the affection syphilitic, so that some of his cases must have been of this origin; but detailed histories are not given. Indeed, the literature of the subject is so slim and inaccessible that conclusions as to the cause of the disease are impossible. Notta's article I have been entirely unable to find, and have relied on a reference to it in Cadell's review of an article by Mauriac. There seems to be, however, sufficient evidence for mentioning this condition in a consideration of syphilitic bursitis, and for applying the therapeutic test when cases of bicipital serositis occur.

Achillo Bursitis Anterior (*Achillodynie of Albert*). In 1893 Albert,[57] of Vienna, described an affection characterized by pain in the Achilles tendon near its attachment, with the presence of a swelling over the tendon just above the os calcis. Six cases were reported. In all the patients, walking and standing were impossible on account of the pain, which disappeared on lying down. Albert's pupil, Rössler[58] called attention, also, to the efforts made by these patients to keep the musculature of the calf lax by plantar flexion of the foot, and to the accompanying pronation of the foot, which led in time to flat-foot. He also showed, by anatomical studies, that the seat of the disease is the bursa lying between the Achilles tendon and the tuberosity of the os calcis. Heinecke[59] had, years before, mentioned a bursitis is this situation. "The anterior Achilles bursa,"[60] he wrote, is occasionally found to be the site of serous and crepitating inflammations; hygromata of this bursa also occur." Raynal[61] and Kirmisson[62] had also described, under the name "peri-tendinous cellulitis of the Achilles tendon," cases which may well have been due to inflammation of the subtendinous bursa. Pitha[63]

[55] Sur les affections de la bourse séreuse du biceps brachial, Bull. et mém. soc. de chir. de Paris, 1905, n. s., xxxi, 581.

[56] Bericht des k.k., Allgemein-Krankenhaus zu Wien, 1883, p. 262.

[57] Achillodynie, Wiener med. Presse, January 8, 1893, Jahrg. xxxiv, No. 2.

[58] Zur Kenntniss der Achillodynie, Deutsche Zeitschrift für Chirurgie, 1896, xlii, 274.

[59] Anatomie u. Pathologie der Schleimbeutel u. Schnenscheiden, Erlangen, 1868.

[60] The bursa is called "anterior" to distinguish it from an inconstant bursa found about one and one-half inches above the Achilles insertion, lying between it and the fascia. Superficial and deep, or subfascial and subtendinous, would be more accurate adjectives to apply to these bursæ.

[61] Cellulite peri-tendineuse du tendon d'Achille, Archives gén. de méd., 1883.

[62] Ibid., 1884.

[63] Krankheiten der Extremitäten.

regarded "partial evulsion of the insertion of the Achilles tendon" as the cause of an affection about the heel which Albert thought identical with the disease he himself described as Achillodynie.

Gout, tuberculosis, rheumatism, but particularly gonorrhœa, were given by Albert and Rössler as the causes of the condition. No connection with syphilis was mentioned; the possibility of its accounting for certain of the "rheumatic" and "gouty" cases does not seem to have been kept in mind. One patient (that of Weinlechner, reported by Rössler) presented a symmetrical, indolent hygroma of both anterior Achilles bursæ, which had appeared spontaneously—a picture strikingly like that already described for syphilitic bursopathy. The case was regarded by Rössler as one of "bursitis deformans." It is to be remembered that before this condition was described by Albert, pain in the Achilles tendon had been noticed in connection with syphilis (see Keyes).

CHOREA A SYMPTOM—NOT A DISEASE.

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One of the most common conditions with which we have to deal at St. Mary's Free Hospital for Children is what is known as chorea. We admit about 50 cases annually, and always have some cases in the wards under treatment. It follows that the subject of chorea is constantly in mind, and commands the attention of those in charge. During the early years of my service at the hospital, begun twenty-three years ago, the idea was that chorea was a peculiar disease of the nervous system; that it was associated in some unexplained way with rheumatism and endocarditis, and was to be treated and cured by increasingly large doses of arsenic. I venture the opinion that this today represents the general idea of the ordinary medical student or recent graduate, and perhaps of some others.

By following the arsenical plan of treatment, within two years I had caused three cases of hemorrhage from the stomach, several cases of hemorrhage from the kidney, and many cases of severe irritation, if not inflammation, of the conjunctiva. I have seen also arsenical neuritis from this method of treatment. Two things became evident to me: one, that the arsenical plan of treatment, as I used it, certainly was too severe, and accomplished but little; and second, that most of the cases of chorea admitted to the Summer Home, at that time situated on Rockaway Beach, got well quickly

and without medication. Experiences like this made me believe that the then teaching concerning chorea could not be right, that the subject was not even as well understood as the text-books seemed to indicate. Recently American writers seem disposed to take a broader view, which I attribute to emancipation from the English writers, who look upon chorea as a serious disease, and probably rightly, since in England all rheumatic affections seem to be more serious than with us. Still the American writers have not, it seems to me, presented the condition correctly. To offer a satisfactory explanation of what is called chorea, one should be able to suggest a reasonable pathology; but since few cases are fatal, the conclusion must be in large measure theoretical.

The subject has formulated itself gradually in my mind in this way: The great mistake has been in regarding the occurrence of incoördinated muscular movements in the young as a disease, an entity. If we put the idea of a disease out of mind and look upon the incoördinated muscular movements as a symptom, or manifestation of some underlying condition, we immediately solve what has seemed a difficult problem. As a matter of fact, in practice we so regard it, and we treat it as a symptom. The causes of incoördinated muscular movements are many, and such movements we find in the old as well as in the young. In the old we consider these movements when occurring just prior to death, in the course of an arteriosclerosis, as due to faulty nutrition and failure of action of the motor control cells. The same sort of movements appearing in the course of a severe illness, a toxemia, or at a time of great stress, we also explain as due to faulty nutrition or lost action of the motor control cells; as due to poisoning of them, or due to their exhaustion by overaction. We do not consider the incoördinated movements as being a disease in themselves. Why, then, when such movements occur in children under certain conditions, should we regard them as due to something quite different? Especially when we know that the normal condition of childhood is constant muscular activity, and that anything that weakens the child naturally weakens the nervous control, and so may permit unrestrained muscular action. If we regard these incoördinated muscular movements as a symptom, or indication of some underlying cause, we frequently can present a satisfactory explanation of them. Such exciting cause may be anything, a condition, infection, or disease which will bring about a weakness, a disability, a malnutrition, or a degeneration of the motor control cells. Some individuals will succumb to such conditions more readily than others; some are predisposed; all cases will not be due to the same cause, nor will they pursue the same course. If we try to classify the cases in accordance with the more evident exciting causes, we put ourselves in the way of being able to treat them reasonably, and also supply as well a pathology of the condition. We also do away with the diagnosis "chorea," which is

in the same class as the diagnosis "convulsions," "jaundice," "dropsy"—a symptom only.

Gradually, in accordance with their cause, I have learned to separate several classes of cases having incoördinated muscular movements as a predominant symptom. These different classes demand modifications in treatment, and it is because of the need of modifications in treatment that I believe the subject is one of great importance. The largest number of cases of so-called chorea are readily divided into two classes, the main distinction being the presence or absence of an endocarditis, or other infective process.

Class I will comprise the many cases which occur in the spring or autumn months, most commonly in girls, and between the ages of seven to fourteen years. A great proportion occurs among those bright, nervously disposed individuals who strain and push themselves to keep up their standing in school, at the head perhaps of their class. They "run down," as the phrase is, lose sleep, eat badly, become anemic. We find the hemoglobin content of the blood is about 60 or lower. These children get into such a state of nervous fatigue, exhaustion, and irritability, that with an added physical or emotional shock, and many times without any such shock, the control centres lose their activity, their power, their balance. The resultant state is one of uncontrolled muscular movements, and that has been called chorea. In many individuals there exists a badly balanced nervous system and such children succumb more readily than do others. Most of these children have shown nervous irritability even as babies; many have tantrums readily; they easily lose control of themselves, but chiefly when tired physically, as from too long walks, too strenuous play, too hard study, too much worry, or too much irritation from any cause. The same sequence of events in an older child in whom the emotional nature is developing, or is the predominant one, is attended with loss of control over the emotions, and this is what is usually called hysteria. We think of such a state as due to nervous exhaustion, shock, the result of an illness, or some toxemia, and we try to establish nervous control by rest, by forced feeding, by diversion, or whatever will aid in restoring the exhausted nerve cells. In the younger child the muscular system is the developing one, and here nervous exhaustion is shown by loss of muscular control. I believe the two states, so-called hysteria in the young adult, and incoördinated muscular movements in the young child, are entirely analogous. The course of the two states is apt to be similar, but it is not regular. Progress and duration depend upon the individual nervous system, on the care received, and on the general recuperative capacity of the individual. In many children the two conditions occur together, loss of emotional and will control as well as loss of muscular control. In this way the changed mentality so frequently seen is accounted for. In some cases the loss of will control is greater than the loss of motor control; the varia-

tions are many; in general terms the conditions are those of nervous exhaustion, and should be treated as such.

Treatment. Such children should be put to bed in a quiet room, relieved of their anxieties, aided to sleep, and be stuffed with plain nutritious food. They also should be given tonics, as iron, arsenic (in small tonic doses), cod-liver oil, marrow, or mixed fats. During the acuteness of the illness the violent movements and restlessness frequently require the administration of quieting drugs, such as the bromides, codeine, trional, veronal, asafœtida, etc., but they should be for temporary amelioration of symptoms only. One should do everything that will aid in restoring the nerve cells to their normal state and function; in fact, give a sort of rest cure. In some cases the result of such treatment is a cure in a short time, particularly if the child can be sent to the country and have the benefit which that means to a city child in the way of air and quiet and freedom from fret. Other cases are very slow in their recuperation, as is the case with individuals after any illness. At St. Mary's the effort is always to keep a cheerful, happy atmosphere in the wards, and to practise, particularly with such cases as have an hysterical element present, a seeming neglect. The purpose of this is to try to keep the child's mind off itself. It is surprising how hysterical some young children can be. Our results with such principles of treatment are most excellent. I have carried out the same idea in the few cases met with in private practice, but the fact that I have seen but few cases of so-called chorea in twenty-five years of private practice, yet have seen many cases of nervousness, makes me more confident that my point of view is correct, for among intelligent parents the first indications of nervous fatigue and breakdown in their children are recognized, and the patients are removed from the sources of strain and placed in the best possible environment. This is something that is not done by the less intelligent parent, and cannot be done by the tenement house parent.

Class II will comprise the large number of cases due to infections; some cases have been caused by the malarial plasmodia; some cases have attended or followed pneumococcic infections, some have followed typhoid infections; by far the largest number are due to the rheumatic infections; and, since there are probably several different varieties of germs causing rheumatic infections, it is easy to understand why so many different germs have been separated and presented by observers as the cause of chorea. In many cases there is present a pericarditis as well as the commoner endocarditis, and I believe there also may occur an endarteritis of some of the cerebral vessels (this of course is theory). I consider that the incoördinated muscular movements are brought about in most of these cases, much as they are in the first variety, by the infecting process causing a profound anemia and malnutrition, or innutrition of the control centres, both motor and sensory

An endarteritis, as suggested above, may be the cause of this in some cases. Whatever the process may be, the important point to be recognized is, that the patient is under the influence of an infection, and should be treated accordingly. The development of an endocarditis is apt to be slow, and the development of the condition of lost motor and emotional control is also apt to be slow. If one is alert to notice such symptoms, he may be able to avert an attack by early treatment. Many cases first show irritability of temper and perhaps weakness of certain muscles, notably of the arm, and are apt to drop articles. In some cases in which an endocarditis is present there is pretty surely some myocarditis and a certain amount of cardiac dilatation, which in itself becomes a source of great nervous strain, unless the patient is properly cared for. The cardiac dilatation may readily follow an attack of so-called tantrums. This is not an uncommon occurrence in these children.

Treatment. Rest in bed is imperative and for an indefinite time. We should administer such drugs as overcome, or combat at least, the infecting germs; quinine, if indicated, sometimes arsenic, the different salicylates, and, probably, in large doses; and remove the ordinary channels of infection, as diseased tonsils, adenoids, decayed or decaying teeth, which are I am sure a common nidus of such infections. Such treatment should be kept up indefinitely, until normal temperature, blood tests, and general nutrition, physically, composure and happiness, mentally, indicate that the child is no longer under the influence of an active infection. No one can possibly tell the duration of such a condition. Meantime, one should not ignore the indications of the endocardium or the other membranes of the heart, or of the vessels. Of course, there are cases of the first class, that is, without infection, which are difficult to separate from the second class, because there will be in some cases cardiac murmurs due to anemia, and cases of dilatation due to the same cause. Fortunately the treatment of rest in bed applies to both classes, and if carried out can only do good. The harm arises from letting such patients run about and be subjected to the strain of ordinary life. I have little doubt that an immense amount of suffering has been entailed by allowing these little people to keep on their feet, because "they only have chorea." Any one who has witnessed the excessive irritability, both mental and physical, of nervous children when fatigued will understand what I mean. Under such conditions it is no unusual thing for the entire nature of the individual to be completely altered for days at a time. Not infrequently we have cases of acute endocarditis brought into the hospital with active choreic movements, in which, after a day or two of quiet rest in bed and sufficient sleep, the movements subside. There are all varieties of cases and all degrees of severity presented. The important thing is to recognize a condition of infection and not to be led to treat the symptom, due only to the processes caused by the infections.

Class III comprises the grave cases, so called chorea major, chorea insaniens, cases which are usually fatal, and which in my opinion are really cases of septic or malignant endocarditis. There have been two such cases with autopsies in my service at St. Mary's. In them the movements were of such violent character that the children were with great difficulty kept in bed, and it was impossible to take temperatures, or to make satisfactory examinations of the heart, but the diagnosis was made before death, and autopsy showed the lesions of septic endocarditis. Dr. B. Sachs[1] reports two such cases under the title "Grave Chorea and its Relation to Septicemia," and makes remarks which agree with much that I have said in this present paper; he too is evidently doubtful as to the entity of chorea, but still he does not get away from thinking of chorea as a disease in itself, and the main disease in the cases he reports; streptococci were demonstrated to be present in the blood of one of Dr. Sachs' patients during life; in both cases the lesions of septic endocarditis were found postmortem. I am sure we shall do better by recognizing the state as infectious or septic endocarditis with choreic movements. If such infectious diseases present in their course convulsions, or delirium, or jaundice, we do not think of such symptoms as the disease. I fail to understand why we should still continue to regard the class of cases to which I refer as chorea major or chorea insaniens or grave chorea. We simply blind ourselves to the real condition, and by teaching that there is such a disease as fatal chorea, we lead our fellow practitioner far astray and allow him to run the risk of failing to understand the true state and of giving his patient no intelligent treatment. I believe the whole idea of the illness has been wrong; the chorea is a subordinate element. To be sure, the outcome is likely to be the same whatever we call it, but one likes to treat even fatal diseases in an intelligent manner.

Class IV.—We have all been taught to regard what has been called Huntington's chorea as the one variety of chorea with a lesion. If one looks at it as due to arteriosclerosis affecting the vessels of the motor cells, one removes everything unusual about it. For there is nothing especially strange about the fact that some families have a tendency to degeneration of a certain set of cerebral bloodvessels. We are misled by the term chorea applied as describing a distinct disease, or a distinct variety of disease, but we are not surprised at the occurrence of incoördinated movements in the development of arteriosclerosis. Observers seem to have been impressed by the violence and largeness of the muscular movements, and have considered such movements the disease.

Class V.—No one probably questions the idea that so-called chorea gravidarum is due either to a toxic state or to nervous depression in a pregnant woman. We all certainly regard these movements as symptomatic only, and treat them so.

[1] New York Medical Record, March 28, 1908.

Other conditions, showing incoördinated movements in their development, doubtless occur. I trust that some observers will agree with me in the conclusion that these movements when occurring in the young also are symptomatic only; that our teaching concerning so-called chorea should be broadened; that it should not be that chorea is a disease per se and to be treated as such, but rather that the occurrence of the symptoms described under this old term should be studied with the view of finding the causative element, and that the treatment should be directed toward it.

THE PRACTICAL VALUE OF THE ASSOCIATION TEST.[1]

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The association test, or experiment, was converted into a practical aid in diagnosis when Jung discovered that a lengthening of the association time gave a clue to the subject's past emotional experiences. Prior to Jung, this variety of psychological analysis concerned chiefly the character of the correlated ideas which were aroused. Wundt blazed the trail with his external and internal association: Internal, in which the stimulus word and the reaction word are connected by the same idea—for example, desk-furniture; and, external, in which the two words are connected by time or place or sound—for example, stimulus, *hand*, reaction, *ring;* stimulus, *snow*, reaction, *country;* stimulus, *bell*, reaction, *tell.* Kraepelin, and especially Aschaffenburg, went farther with this line of inquiry in normal persons and in such pathological conditions as toxic states, maniac states, and exhaustion. In normal subjects, they showed that external associations exceed in ratio the internal. In the pathological states they demonstrated particularly that sound associations indicate a lowering of intellectual function. Ziehen also has shown that reaction words in children relate to what has been actually experienced by the child; for example, *water, swimming,* the child being fond of swimming; *insolent, Elizabeth,* Elizabeth not being a favorite of the subject.

The time element in associations had also been studied before Jung. Galton, in 1878, had measured association time, and this feature was also not the least of the results of the experiments of Kraepelin and Aschaffenburg. These latter showed that external associations are quicker than internal; that manic states have a lengthened association time, in spite of the flight of ideas, and that in alcoholism the associations are first quickened and later slowed.

[1] Read at a meeting of the Section on Nervous and Mental Diseases of the American Medical Association, Atlantic City, N. J., June 10, 1909.

But it remained for Jung to point out a hitherto unknown significance of lengthened association time. He found that a lengthened interval in one or more reactions in the series indicates that the stimulus word has touched upon an emotional complex, or a constellation of ideas which exists independently of volition and which is dominated by an emotion of some kind. This discovery immediately converted the association test into a practical and reasonably speedy method of psychic analysis. Previous methods were time consuming. Their results were general, revealing the characteristics of the disease or state, rather than of the individual.

The Jung test is primarily individualistic. The stimulus word, like the hunting dog, shows where an emotional complex is lurking. It shows what the subject likes and what he dislikes; where he has suffered and what has pleased him; what he regards as friendly and what as hostile; what he fears and to what he is indifferent. These biographical data come to light without the subject's knowledge and sometimes against his will, and furnish a rational basis for psychotherapy. Psychotherapy is a treatment by suggestion, by explanation, by persuasion, by a regulation of environmental influences. The physician's own experience and judgment furnish motives and points of view, delivered with the purpose of changing the patient's attitude toward himself and his surroundings. The association test furnishes the information indispensable for such psychic guidance so easily and quickly, and the test itself is so much less cumbersome than other methods of psycho-analysis, which require the use of instruments of precision, that it seems remarkable that it has not received more prompt adoption.

Scripture, Brill, and others have called it to the attention of the medical profession in this country. It is part of the routine examinations in several psychiatric clinics and laboratories, and is becoming a favorite topic in the magazines. But the physician generally seems to have passed it over. It has appeared to me worth while, once again, to call it to public notice, and to mention some instances in which it has proved useful.

The method is as follows: A list of one hundred current words (see Chart) is printed on a pad and so distributed that there is an alternation of nouns and adjectives, and every fourth word a verb. These words are printed in a vertical column, and between every three or four of them a space is left, so that if there seems to be a need of a word not on the printed list, it can be written in. Each word is numbered, so that it can be referred to by number without pronouncing the word. On the same horizontal line as the number and the word three spaces are left—one for the time record, one for the reaction word, and one for the reproduction, or the response which is given on the second trial. If the reproduced reaction is identical with the original reaction, it is indicated by +. The method is explained to the patient, and the words are then read to him.

Name.....

	Time.	Reaction.	Reproduction.
1 Head:			
2 Green:			
3 Water:			
4 To prick:			
5 Angel:			

Chart showing a convenient printed form for the association experiments. Note the blank space left for additional words.

This may be done by the physician, but is better done by some third person. The association time is taken by a $\frac{1}{5}$ second stop watch, and it and the reaction word, or the first word spoken by the subject in response to the stimulus, is entered in the space reserved for it. The second trial, or reproduction, generally necessary for suspicious words only, should be delayed for at least twenty minutes. Failure to reproduce has been shown to be evidence that the stimulus word has touched upon a complex. The sheet containing all the facts becomes a permanent record. The test gives its most brilliant results when it brings to light an emotional experience which is no longer a factor in the conscious life of the individual, but which, nevertheless, has started a chain of persisting symptoms. Ordinary history taking can hardly hope to reveal such buried psychic causes. An example is the following:

A man had suffered for seven years from tic convulsif. The closest questioning failed to show anything in his past career that might be regarded as of etiological significance. The association test showed, in general, prompt reactions. But the word *lung* took thirteen seconds for the reaction *man*. Questioning brought out that seven and one-half years previously the patient had been examined for life insurance, and declined by two companies in succession. The reason for rejection was pulmonary tuberculosis, of which the candidate had been ignorant. Shortly after this shock the convulsive movements began. The patient had no conscious association between the two occurrences.

Few of us are prepared to accept the theory of Freud that sexual traumas in childhood are the mainsprings of hysteria. But Freud's teachings have been of great value in pointing out that painful emotional experiences in children are responsible for many neurotic symptoms, which can only be overcome when the cause of them, long since perhaps passed from conscious memory, has been fully brought to consciousness. A lady had suffered practically all her

life from sudden feelings of weakness, from feelings of inadequacy and depression, and from occasional losses of consciousness. She was sent to me with the diagnosis of petit mal. Her associations were mostly prompt, that is, between 1.5 and 2.5 seconds. The following were exceptions:

To threaten	to anger	3.5 seconds	R., to paralyze
Water	bread	4.5 "	R., milk
Trapeze	rope	7.0 "	R., fall
Woods	fear	3.5 "	R., +
Journey	trip	4.0 "	R., faint
Sympathy	lack of sympathy	4.0 "	R., +
Procession	night	4.0 "	R., plight
Suicide	hell or fire	7.0 "	R., hell

The first four of these reactions had reference to events in childhood, the others to later experience. The reaction *to threaten, to anger, to paralyze*, brought her back to when she was a child of eight. A coachman, having enticed her to his room, took her on his lap, pressed her to him, kissed her, and then threatened to injure her if she ever told. *Water* recalled an experience, also in early childhood, when she saw her brother drowned; *trapeze*, when, also as a child, the rope of a trapeze she was on broke, giving her a bad fall; and *woods*, when she, with other little girls, was badly frightened by being told that Indians were behind the trees.

All of these were painful experiences, about which she rarely spoke. Her husband was the only person to whom she had mentioned the affair of the coachman. On the theory that they were concealed complexes which "mental catharsis" might mitigate, a companion was provided, and to the companion the patient talked very day on these painful subjects. The other complexes aroused, being of recent date, were not regarded as of causative significance. The effect of the free externalization was prompt and pronounced. The patient emerged from a life of invalidism to one of activity; she had no more petit mal attacks; and while she is still annoyed by some of the old nightmares, she is better than she ever has been since childhood.

The association experiment has proved of great use in the history taking of young adults beneath the surface of whose variegated nervous phenomena lurked a paranoid condition. The experiment itself did not finally fix the character of the paranoid state. Whether it was psychasthenic, or a beginning paranoia, or dementia præcox, had to be decided from outside considerations. But the complexes aroused pointed the way for inquiry, and revealed frames of mind which would have been difficult to reach in any other way or which might have escaped identification altogether.

A boy of twenty-one had always been eccentric, solitary, and at times depressed, but never had given expression to insane ideas. He was an only son, and did about as he pleased. He would change

his school frequently, and his education suffered. He refused to eat in his own house, and took his meals at a restaurant. He had various notions as to what was best for him, most of which were entirely erroneous. He was a problem for his family, who were utterly at a loss to explain his peculiar behavior. Confronted with the association test, he was prompt in his reaction. The following words were noted as suspicious:

Friendly	unfriendly	2.8 seconds	R., +
Insolent	ugly	2.5 "	R., unfriendly
Angry	foolish	3.2 "	R., ugly
Goat	billygoat	3.0 "	R., butt
Luck	chance	4.2 "	R., +
Estranged	to separate	3.4 "	R., fear.
Scorn	depart	4.6 "	R., despise.
Affection	hate	4.0 "	R., despise.
Chance	trial	5.2 "	R., luck.

In conversation about these reactions, he admitted that people were not friendly, that they were insolent, that their treatment made him angry, that he was estranged from his family, that he had no luck. The word *goat* brought out this embryonic delusion of persecution in a remarkable way. He said in regard to his reaction to that word that he could not help thinking of how often he had been made a butt at school and how unfriendly the boys were to him. The stimulus word *scorn* recalled a definite occurrence. The complex concerned dated from a period of great excitement, when he thought his family scorned and despised him, so that he decided to leave the house, *to depart*. He was prevented from doing so by his father.

In this case the test made clear that there was a slowly forming paranoid state, with convictions of injustice and feelings of resentment. To break them up, the patient was sent abroad in the companionship of a University man with psychological training. The instructions were that full and free conversation should be held on all unpleasant topics. These instructions were carried out. But it is interesting to record that after three months' intimate conversations, nothing had been added to the results obtained in one interview by the association test. The patient improved in deportment and spirits and lost most of his eccentricity. And the following year he did well at school, though far behind in his studies. But the fancies in regard to the esteem in which he was held by others never faded completely, and the chances seem to be that he must continue to suffer from a non-dementing delusional form of mental disorder.

In his monograph on dementia præcox, Jung has drawn a parallel between the emotional complexes of hysteria and those of dementia præcox. Both may be aroused by a stimulus word. But experience teaches that the complexes in dementia præcox are less accessible, more clouded; and in it a larger proportion of the

lengthened reaction times will remain unexplained than in a pure psychogenic disorder like hysteria.

I have in mind the case of a boy who wrote me: "I have not succeeded in what I most desired to attain, that of living the most normal life like other people of my own age. As long as I can remember, I have felt this deficiency, and wished to overcome it. This is especially in regard to other people. I have a sense of inferiority when with people of my own age, and do not seem to get the respect they give each other. From this I get an idea that I am alienated from them. I have never made a warm friend of my own age, and I got an idea that people were unfriendly and I could not get on with them."

This from a lad, aged seventeen years; one also who lacks initiative except when he flies into violent rages and who has never profited from any discipline; one who for two years past has constantly complained that something was wrong with him, that people were talking about him, that no one liked him. The clinical diagnosis was dementia præcox, and the association test confirmed the general trend of the outlined mental attitude. For example:

Friendly	man	12.0 seconds	R., unkind.
Insolent	manner	14.0 "	R., failed.
Sick	feeble.	11.0 "	R., will.
To threaten	angry.	6.0 "	R., anger.

But in addition to these emotional reactions, which agreed with the general mental tone of the patient and which were decipherable, there were many other lengthened reactions, the causes of which all inquiry failed to disclose. And this fact, that together with some complexes of paranoid type there were others, many of them, which eluded analysis, strengthened the diagnosis of dementia præcox.

There is a class of patients in whom the association test seems to give little useful information. These suffer from a form of nervousness which is the product of the times and environment. Domestic unhappiness, disproportionate wealth, idleness, all acting on limited brain equipment, fail to produce that beauty in the inward soul which Socrates prayed for. The symptoms, too familiar to need comment, come and go under the stress of provoking circumstance and shifting varieties of therapeutic procedure. In these individuals the association experiment may start a clue for judicious inquiry. It may bring to light that a nervous woman has an extramural attachment, or that a trustee has turned speculator. But a sagacious physician soon finds out such things, and the association experiment will not help him much.

Jung believes so fully in the association test that he thinks it will give information against the will or desire of the subject. This is true to a certain extent. But I have always doubted the value of the

results when the subject was plainly unfriendly to the inquiry. Certain cases of hysteria illustrate this.

One lady intelligent in many ways, who had for years been treated by various forms of faith cures, became afflicted with peculiar paroxysmal spasms of the neck, abdomen, and legs. These proved to be phantoms of the sexual act, and dated from a time when a male hypnotist treated her by "laying on of hands" on the abdomen. This patient rebelled against all direct questioning; and in the experiment, to which she was opposed, she gave long association times to nearly all stimulus words; and often said she could "think of nothing." Her chief complex was erotic, but she cunningly defeated all experimental efforts to disclose it.

The same difficulty has arisen in cases of hysteria which were litigated. One plaintiff, suing for $50,000 damages, and perfectly able to converse intelligently, examined by the physician for the railway company, gave an average association time of 9.7 seconds, which, if not an effort to thwart the purpose of the investigation, was at least negativeness inspired by suspicion. But even in such cases the underlying principle exerts some influence. This plaintiff gave his longest association time—20 seconds—for the word *damages;* and in another somewhat similar case, in which the plaintiff had been the victim of a railway wreck, with an average time of 6 seconds, the longest reaction was for the word *collision.*

Similar uncertainties exist in criminal inquiries. Jung's original case was medicolegal, and the association experiment aided in the proof of stealing. Professor Münsterberg, in a popular article so ingeniously worded that the reader might infer that the discovery of this method of inquiry emanated from the psychological laboratory at Harvard, proclaimed the test as a final one for crime. It has won no such title. It may point to lines of inquiry which, if followed up, would furnish proof of guilt. But the possibility of the voluntary cloaking of complexes is too little understood as yet to make the experimental investigation of associations reliable in cases controlled by the strongest motives for deception. It seems further that reliance on this test might result in gross injustice. Immediately an individual is accused of crime the accusation itself creates a complex. And in the case of an innocent man this artificially created complex might lead to changes in association time as significant as though he were guilty.

I am unable to say how large a proportion of innocent persons accused of crime would react promptly to a list of 100 to 200 words. If an accused person could go through such a test without the arousal of complexes directly connected with the charge, it would be a strong presumption of innocence. This negative result, which seems to be the chief use of the test for the criminal investigator, is even more valuable in purely clinical cases. A person clinically suspected of insanity, is rarely swayed by the motives for deception

which actuate a prisoner. And an investigation with negative results in such a case would be strong proof that the suspected mental state was within normal limits. To such uses the test may be constantly put.

A young girl was the object of much solicitude on the part of her parents, her physician, and her teacher. She was impulsive; did not care to study; defied her parents; on two occasions she marched out of school, vowing she would never come back. And one day, without a word to any one, she cut off all her hair. They all thought that some hidden delusion or some insane impulse alone could explain such behavior. The reaction words in the association test showed plainly the "schon erlebtes" of Ziehen—for example, the childish type of mind in which the associations aroused have to do with personal experiences, not abstract ideas. There was no lengthening of association time, except for a few recent and unimportant emotional complexes. There were no traces of sexual traumas, paranoid associations, or psychic points of origin of morbid impulses. The result of the test was, therefore, negative. And therein lay its value. For the negative findings excluded the probability of the existence of motives of insane character. And these negative findings were further entirely in accord with outside information in the case. The child was headstrong and self-willed, and had missed judicious parental supervision. But these facts by themselves were not sufficiently evident to have made it possible to regard the patient's peculiarities as within the limit of normal. With the association test such a diagnosis became almost certain.

THE IMPORTANCE OF BLOOD CULTURES IN THE STUDY OF INFECTIONS OF OTITIC ORIGIN.[1]

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Although we made some studies of the bacteriology of the blood in cases of otitic infection some years ago, our interest in the subject was particularly aroused by a case in which the clinical diagnosis could not be definitely established, and in which the finding of streptococci in the blood led us to believe that it would be wise to explore the mastoid process, because the patient had a moderately

[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 11 and 12, 1909.

marked otitis media, and no other focus could be found from which the streptococcus might have entered the blood current. Not only was mastoid disease found, but there was also a sinus thrombosis present. A short time later a second case of the same type came under observation. We then decided to follow up the bacteriology of the blood more systematically in cases of acute and chronic ear disease, and encountered some other cases in which the bacteriological examinations were very valuable. In the last two years we have paid particular attention to this subject and will report in this paper a study of 163 blood cultures in 100 cases.[2] We have considered it advisable to present our results up to the present time for several reasons. In the first place, we believe that not sufficient attention has been paid by the internist to the border line work between otology and internal medicine. And it is the internist upon whom the responsibility often devolves of deciding whether an otological case should be operated upon or not. In the second place, the work illustrates very well how valuable a comparatively simple laboratory method may be in leading to important diagnostic and therapeutic conclusions. And thirdly, the studies, we believe, will be of interest because of the light they throw upon the question of the significance of certain forms of bacteriemia.

We shall not discuss the literature to any great extent, partly because we wish to include it in a later larger publication, and partly because much of it is based on bacteriological methods, the results of which cannot be accurately compared to the results obtained by our present methods. One of us (Libman) has given a brief summary of our earlier results on some former occasions.[3]

As part of our study we investigated the bacteriology of otitis media (particularly the acute forms) to ascertain the relative frequency of the organisms most commonly involved in the causation of the disease. We studied 277 cases with particular reference to the occurrence of streptococci, Streptococcus mucosus, and the pneumococcus. The organisms that are next most commonly found are the staphylococci, Bacillus pyocyaneus, and Bacillus proteus. Mixed infections were not uncommonly found.[4] We classified the streptococcus, Streptococcus mucosus, and the pneumococcus according to the classification that we have adopted as the result of studies made in our laboratory, particularly by Dr. Buerger[5] and later by Dr. Rosenthal together with one of us (Libman[6]).

[2] The actual number of cases is somewhat smaller than this, as some of the cases are listed twice, because they were first admitted to the hospital for otitis media and mastoid disease and then discharged and later returned with a suspected sinus thrombosis.

[3] Gruening, Libman, and Epstein, Transactions of American Otological Society, 1906; Libman, Archives of Otology, 1908, No. 1, p. 22.

[4] For fuller details see Libman, Archives of Otology, 1908, viii, No. 1.

[5] Jour. Exper. Med., August 25, 1905, vol. xiii.

[6] Trans. New York Path. Soc., February, 1908. A full report of studies will be published in the near future.

In the 277 cases which we studied we found streptococci present alone or with other organisms in 189 cases (81.46 per cent.), Streptococcus mucosus twenty times (10.34 per cent.), the pneumococcus nineteen times (8.2 per cent.). The earlier authors found the pneumococcus present more frequently than the streptococcus, but the more recent writers (Leutert, Funke, Siepfle, Wittmanck, Mark) found the streptococcus present more frequently than the pneumococcus. It is to be noted that while Streptococcus mucosus is morphologically a streptococcus, it is more closely allied from a biological standpoint to the pneumococcus.

Notwithstanding the fact that the pneumococcus has been found frequently by many authorities in the middle ear, and notwithstanding the fact that we have found the organism in a certain number of cases, there are hardly any records of sinus thrombosis being due to the pneumococcus, and we have not met with any such cases. The few cases that are to be found in the literature are not definitely proved, because the methods used for identifying the organisms as pneumococci are not stated. It has been shown in our laboratory by Drs. Buerger and Ryttenberg[7] that the pneumococcus can be so changed by its sojourn in the blood that it may be very difficult to differentiate it from the streptococcus. This fact must be taken into consideration, for it might occur that we might find an organism that looked like a streptoccocus in the sinus and it might prove to be a changed pneumococcus. We have investigated this point, and have not had any such experience in our sinus thrombosis studies. It is necessary to study a large number of cases so as to determine definitely how frequently the pneumococcus causes a sinus thrombosis and whether the pneumococcus really causes a less serious local form of otitic disease than the streptococcus.

The literature concerning blood cultures in cases of otitic infection is very meagre. Besides the work of Kobrak, which we shall discuss directly, there are practically no systematic studies of the subject. A review of what has been done along this line up to 1907 is given by Hasslauer.[8] The work of various investigators which is cited by Hasslauer cannot be used for comparison with our own work for the reason that so few casess were studied by each author, that the bacteriological methods varied very strikingly, and that hardly any of the authors made a comprehensive study of the primary focus, the blood in the general circulation, and the metastases. Some examined only the blood of the sinus. In general, one can

[7] Jour. Infect. Dis., 1907, vol. iv.

[8] Internationales Centralblatt f. Ohrenheilkunde, 1907, v, 1. See also Leutert, Archiv f. Ohrenheilkunde, lxxiv, 23. He gives references to a few further cases in which bacteriological examinations were made either of the sinus blood or of the blood of a peripheral vein. A case of general infection is mentioned briefly by Lenhartz (Septische Erkrankungen, 1903, p. 335), in which there was a remarkable decrease in the number of colonies of streptococci in the blood in a case of otitic infection with recovery. No data are given as to whether the patient had a sinus thrombosis or not, or whether any operations were performed.

draw the conclusion that the streptococcus was found in most of the cases.

Kobrak[9] found streptococci predominating in the primary focus He then studied 20 cases of acute otitis media, which were accompanied by fever in the first few days, believing that he would find that a bacteriemia was responsible for the fever. In all these cases the blood proved to be free from bacteria. He later took blood cultures only in cases of marked severity with fever of long duration and he states that he found positive results only if the fever was pyemic, that is in so-called "septic cases." In 11 cases in which a positive blood culture was obtained, he found streptococci seven times, Streptococcus mucosus twice, the pneumococcus once, and the staphylococcus once. Of the 7 streptococcic cases, 4 died, all, as he states, "markedly septic." He states that in one case there was only a peribulbar abscess, and in another there was a direct infection from the ear. In the 2 mucosus cases the organism was also found in the cerebrospinal fluid.[10] His total studies of streptococcic cases comprise 28, with general infection in 7. In 17 pneumococcic cases he found the pneumococcus in the blood once only.[11] He remarks that in cases of otogenic "sepsis" the decrease in the number of colonies in the blood and in their hemolytic power is of no significance, but rapid increase in the hemolytic power or in the number of colonies renders the prognosis very bad. The author does not give details as to the clinical course of these cases, nor the details of the post-mortem reports in the fatal cases. It can not be determined in how many cases it was possible that the patient had a sinus thrombosis from which he recovered without operation, nor can it be determined whether autopsies were performed in the fatal cases, and if so, how carefully the veins were examined.

Since we have paid closer attention to this subject we have made blood cultures in 50 cases of mastoid disease with recent or old otitis media, and in 10 cases of uncomplicated otitis media. Some of the cases of mastoid disease were accompanied by extradural abscesses. About one-half of the cases had at some time or other a temperature of 102.5° F. or over, some had a lower temperature, and some were practically afebrile. In a few of the cases the otitis media was bilateral. In our older series of cases we had positive results in two fatal cases of otitis media, but in both instances the blood cultures were taken but a short time before the fatal termination. In neither case is there a record of an accurate examination of the sinuses and the jugular bulbs.

[9] Allg. med. Centralzeitung, 1907, lxxvi, 299. See also Verh. der Deutsch. Otolog. Gesellsch., 1907, xv, 93.

[10] As will be seen later, we have also had positive results in otitic meningitis due to Streptococcus mucosus and to the pneumococcus.

[11] He does not state how the pneumococcus was identified.

Our method, in brief, was to withdraw from 10 to 25 c.c. of blood from one of the arm veins (if necessary, from some other vein), and to incubate the material in agar, glucose agar, serum agar, glucose serum agar, and in bouillon and glucose bouillon with and without serum. If a large amount of blood was obtained, nearly all of these media were used. When a small amount was used, preference was given to the media containing glucose and serum. We often tested the value of using the blood in smaller and larger amounts in the various media. No differences were found. The plates and flasks were incubated for five days, subinoculations being made from the flasks (after shaking) in glucose bouillon and on serum glucose agar. In children we have often been compelled to use a smaller amount of blood, particularly in infants. We might state here that our studies in infants and young children are not yet extensive enough to warrant deductions.

Kobrak is not the only author who claims to have found a general infection in cases of otitis media without involvement of the sinuses or meninges. There are other scattered reports of such cases. It is essential to study a very large series of cases of uncomplicated otitis media and mastoiditis to determine the real frequency of such an occurrence.

Our positive results in otitis cases have been found only in those complicated by meningitis or sinus thrombosis (of course we include here thrombosis of the jugular bulb). In the meningitis cases we have found the pneumococcus and Streptococcus mucosus in the blood. In some of these cases a complete postmortem examination was not permitted, and we cannot state whether an infection of the veins was also present or not.

Before the cultures were made exclusively by one of us (Celler), we had a series of 14 cases in which the blood cultures were taken in different cases by different men, and in which repeated cultures were not made. Of these 14 cases, bacteria were found in 9, giving a precentage of 64. The later series comprises 16 cases, of which 14 gave positive results, giving a percentage of 87.5. In one negative case the clot obtained at the time of operation was found to be sterile; it was inoculated on media after being thoroughly ground up. In the other case we did not have the opportunity of seeing the clot, but the surgeon informs us that it was parietal and that it looked like an organizing non-purulent thrombus. It is possible that the high percentage of positive results is due to the fact that repeated cultures were made and that in a number of cases the cultures were made earlier than we were accustomed to make them a few years ago. If we reckon together both sets of cases, we have a total of 30 cases with 23 positive blood cultures (77 per cent.).

In the old series of cases streptococci were found in all of the 9 positive cases (during part of this time an accurate identifica-

tion of Streptococcus mucosus was not yet being made in the laboratory). In our second series streptococci were found eleven times, Streptococcus mucosus twice, and Bacillus proteus once. It will be noted, as stated before, that we have not found the pneumococcus in any of the cases. In our subsequent discussion we shall base our remarks only upon the cases in which the streptococcus and Streptococcus mucosus were found, classifying them for clinical purposes together.

In one positive case Streptococcus mucosus had been found in the primary otitis media and in the pus from the mastoid, but an ordinary streptococcus was isolated from the blood. The patient had been discharged from the hospital with an open wound, in good health, and returned later with a sinus thrombosis. The wound could have easily been secondarily infected by a streptococcus during the weeks that elapsed between the time of the mastoid operation and the time of development of the sinus affection. In cases in which there is a mixed infection of the middle ear, only one of the organisms may be found in the pus in the mastoid bone. In the case of general infection by Bacillus proteus, which we have cited above, the cultures from the middle ear also showed a large number of streptococci.

According to our recent experience, it would appear that nearly all cases of sinus thrombosis are accompanied by a bacteriemia at some time or other. We are inclined to believe, from a general survey of all our records,[12] that there may be early no bacteriemia, that the bacteria may then be found perhaps in increasing number, and that it is possible for the bacteria to decrease in number and even to disappear before there has been any operative interference.

It is of interest to attempt to explain the occurrence of the negative results in some of the cases. Different explanations probably hold for different cases. In some cases (probably rarely) the clot is sterile and it is easy to explain the absence of bacteria in the blood. In some cases the blood culture may have been taken before the bacteriemia had been established or after the bacteria had disappeared from the blood. Bacteria may be present in the blood in too small number to be found in the amount of blood used in making the cultures. This would not necessarily mean that there had not been many bacteria discharged into the jugular vein, because there is a possibility that large numbers of the bacteria may be rapidly killed off, in which event few or none might be found in the blood obtained from one of the peripheral veins.[13]

It is also possible that in certain cases there is below the infected clot an obturating, non-infected clot which prevents the bacteria

[12] It is difficult to draw conclusions from any one case, because cultures cannot be taken frequently enough to make thorough observations.

[13] See Leutert, infra.

from entering the circulation. One can also imag ne the possibility of there being an infected clot which completely closes the vessel. Another possible explanation is that, in the course of the infection the blood may acquire a higher bactericidal power.

In some instances a bacteriemia is cut short by tying the jugular vein. In such cases metastases may develop even though the blood is free from bacteria. They may develop from bacteria or bits of clot deposited in various parts of the body before the vein has been ligated. All such foci need not appear at once. We must also remember that at the time of operation, particularly if the sinus itself is exposed before the jugular vein is tied, and there is a thrombosis present, bits of infected clot may be scattered. In this way also metastatic foci could develop without a persisting bacteriemia.

In some cases in which the jugular vein has not been tied, metastatic abscesses may be found in the lungs and there may be no bacteria in the circulation. In these cases, which are generally older ones, we are often dealing with a softened clot from which pieces are broken off which contain bacteria which set up secondary foci. The probable explanation is that free bacteria are promptly killed off, but that those carried in pieces of thrombi are protected during their passage through the blood from the bactericidal power of the latter. In our experience, the metastatic foci in cases of so-called "pyemia" do not cause a bacteriemia.

Our studies show that cases in which streptococci are found in the blood after the disease of the mastoid is properly operated upon, and in which clinical symptoms persist and no other cause can be found for a streptococcemia, are cases of sinus thrombosis. It is very important to consider carefully the possibility of other causes of streptococcemia (or other bacteriemia) in such cases. In our investigations the cases of otitic disease which even before operation showed a streptococcemia, and in which other causes for a streptococcemia could be excluded, showed sinus thrombosis at operation or at autopsy. We would not be willing to state absolutely, however, that in all the cases in the latter group a sinus thrombosis would be found, because our studies in cases of acute otitis media and mastoiditis uncomplicated by sinus thrombosis are not yet extensive enough. But if the middle ear has been properly drained and the mastoid has been operated upon and there is no meningitis present and no other cause found to explain the persisting bacteriemia and persistent clinical symptoms, we come, practically by exclusion, to the assumption that a lesion is present in the veins which causes the bacteriemia.

It is necessary, in studying such cases, to take a number of points into consideration. There is always a possibility that a patient is suffering from a general infection in the course of which he develops otitis media or mastoid disease. This is particularly liable to occur in cases of general pneumococcic or streptococcic infection, cases of

pneumonia,[14] and cases of typhoid fever. In cases of typhoid fever it is essential to know whether or not the otitis media developed after the patient had already had fever. The presence of typhoid bacilli in the blood, if one were in doubt as to whether marked symptoms were due to an otitis media by itself or to a complicating typhoid, would make us feel that we could ascribe the fever to the typhoid infection alone. The rest of the diagnosis would have to be based on clinical signs and on the development, possibly, of a streptococcemia, although it is to be remembered that rarely a patient with typhoid fever suffers from mixed infection with streptococci. In some of these cases it might be very difficult to come to a conclusion as to the exact condition in the mastoid and surrounding parts.

It is also important to note the possibility of a bacteriemia following operative interference upon the middle ear or the mastoid process. In all of our cases in which we found streptococci in the blood after the mastoid had been operated upon, a sinus thrombosis was found. But from our experience with postoperative bacteriemia after operations in other parts of the body, we believe that one should be very careful in judging of positive blood cultures if the blood cultures are taken directly after there has been operative interference.

It has often been pointed out that difficulties might arise because of the possibility of a developing erysipelas or the presence of an angina. It has been feared that one would have difficulty in interpreting a streptococcemia in a case of otitic disease if either of these conditions were present. As a matter of fact, in our experience we have very rarely found streptococci in the blood in erysipelas, and then it has only been in fatal cases. This experience seems to be shared by other investigators of the blood in this disease.

The question of a possible confusion because of the presence of an acute angina is a very important one. Streptococcemia in such cases is very infrequent. In our experience we have fortunately had no trouble from this source.

Leutert[15] and, later, his assistant, Nuerenberg,[16] have suggested a method for attempting to overcome the difficulty involved under the conditions mentioned above. Leutert has suggested taking blood cultures from the sinus and from the jugular vein and an arm vein. He believed that if more streptococci were found in the blood of the sinus than in the blood of the jugular vein or an arm vein, or if organisms were found in the blood of the sinus and none in the blood of the peripheral vein, one could conclude that the bacteria were gaining entrance to the circulation from the sinus, and that the bacteria were not entering the blood from the coincidental angina

[14] If in a case of otitis media due to streptococci, pneumococci should be found in the blood, one would have to think of a developing lobar pneumonia.

[15] Archiv f. Ohren., lxxiv, 18.

[16] Münch. med. Woch., No. 51, p. 5227.

or erysipelas. They describe several cases in which they found this method valuable, but they admit what at once must strike any one as a serious objection. The results cannot always be depended upon, because the sinus is aspirated through an infected area. In one case we aspirated the sinus because we could not obtain blood from one of the peripheral veins. The blood from the sinus was sterile; a clot found at operation was also sterile.

In our cases the number of bacteria found in the blood in cases of sinus thrombosis accompanied by bacteriemia has varied from less than one to the cubic centimeter up to 245 to the cubic centimeter. In all of the cases, except those that died within a few days after admission, we attempted to ascertain how rapidly the bacteria disappeared from the blood after operative interference. In all except three cases, which we will describe later, the bacteria disappeared rather promptly. As a rule, the bacteria did not disappear until the jugular vein was tied. There were two cases, however, in which the bacteriemia disappeared after a clot was removed from the lateral sinus and the jugular vein was not tied.

In our early studies we took the secondary cultures forty-eight to seventy-two hours after operation, and we found that the bacteria had disappeared in all the cases except in the three soon to be described. In some of the later cases we took the blood cultures twenty-four hours after ligation of the jugular vein, and these cultures also gave a negative result. It is very remarkable to see as many as 245 streptococci to the cubic centimeter of blood disappear within twenty-four to forty-eight hours after ligation of the jugular vein. In one case there were seven colonies of streptococci to the cubic centimeter of blood; within eight hours after ligation of the jugular vein the blood culture was negative and remianed so.

The three cases in which bacteria did not disappear promptly after ligation of the jugular vein were of particular interest. In one the streptococcus persisted in the blood after the clot had been removed from the lateral sinus and the jugular vein had been tied.[17] The bulb of the jugular vein was then exposed, a clot removed, and then the bulb packed; the bacteriemia (there were two colonies to the cubic centimeter of blood) then promptly disappeared in less than four hours. In this case we believe the continuance of the bacteriemia was probably due to infection of the general system by way of the inferior petrosal sinus.[18]

In another case the bacteriemia disappeared after ligation of the jugular vein, but a couple of metastatic deposits developed.

[17] In all these cases in which the jugular vein was tied a section of the vein was resected. The operation on the sinus usually consisted of removing as much clot as possible, and packing.

[18] We shall attempt to continue the study of the rapidity of the disappearance of organisms from the blood of these cases by making cultures at still shorter intervals after operation than we have heretofore done. We shall report any results that we may obtain at a later time.

The patient also had a right-sided mastoiditis, which was operated upon. Later, streptococci were again found in the blood, the patient developed more metastases, and finally recovered. Although at the time we did not think of the possibility, we now believe that it is probable that in this case there was also a thrombosis on the right side, from which the patient recovered without operation. We know that some cases of sinus thrombosis with or without metastases have recovered without operation. The same holds true of infected veins in other parts of the body.

In a third case in which the bacteria persisted even after the local focus had been thoroughly dealt with (and this was confirmed at autopsy), the persistence of the bacteriemia was due to the development of an acute (ulcerative) endocarditis.

The studies on the disapperance of the bacteria in these cases have almost the value of experiments. They show that the bacteriemia is due to discharge from the local focus, and that the bacteria remain in the blood after the local focus has been thoroughly dealt with (when that is possible) only if multiplication of bacteria occurs in the blood or if an acute endocarditis is established. Both these conditions have been very unusual in our experience.[19]

What then, according to our experience, is the value of blood culture studies in cases of otitic infection? We will discuss first the significance of positive results and then briefly the value of negative results:

1. Significance of Positive Results. (*A*) A positive blood culture is valuable when there is doubt as to the existence of a sinus thrombosis after a patient has been operated upon for mastoid disease. In many cases the diagnosis is clear enough clinically, but in a certain number of cases in which the mastoid disease had been thoroughly dealt with, and in which there were clinical symptoms still present, and in which the otologist was not sure that there was still further local disease, the finding of streptococci in the blood has given the indication to explore the sinus, and in all the cases of this type a sinus thrombosis was found. As we have stated before, it is essential to exclude all other causes of streptococcemia.

A case worthy of citation here is that of a man, aged thirty-three years, who developed pain in the left ear, following an attack of grippe, four weeks before admission to the hospital. There was increasing tenderness over the mastoid process and slight fever, but at no time any ear discharge. The drum membrane was slightly reddened. It was also noted that there were very tender and large lymph nodes behind the angle of the jaw. During the first three days of the

[19] In the case of general infection by Bacillus proteus, which is mentioned earlier in the paper, the bacilli also persisted until death, but in this case the local focus could not be thoroughly dealt with, because the thrombosis had extended into both petrosal sinuses on the same side, both cavernous sinuses, and into the lateral sinus of the opposite side.

patient's stay in the hospital his temperature ranged between 100° and 102° F., the pulse rate varying from 70 to 84. On April 20 the temperature rose to 103° and he was operated upon. A chronic mastoiditis was found. After the operation the temperature dropped, so that on April 24 and 25 it was practically normal. On April 26 there was a slight chill and rise of temperature to 102° F. During the following four days the temperature was about 101° F., once reaching 103°. On April 29 the patient was allowed to be out of bed. The pulse rate was 72 to 80. The leukocyte count was 21,200, the differential count showing 80 per cent. of polynuclears. In the early afternoon of May 2 a blood culture was made, the patient being up out of bed in a chair. On the evening of the same day the patient had a chill; his temperature rose to 104.6°. The following day, when the temperature was down to 100°, we reported that there were 210 colonies of Streptococcus mucosus to the cubic centimeter of blood. As there was no other cause for such an infection, it was decided to operate again, in the expectation of finding a sinus thrombosis. The operation was performed the same afternoon. A thrombus was found in the lateral sinus. The jugular vein was ligated in two places, the intervening part being removed. On the following day, May 4, the blood culture was found to be negative. The temperature was normal by May 6. In this case the clinical symptoms which suggested making the blood culture were the moderate irregular fever, the increase in the leukocyte count, postmastoid tenderness, and a rather sickish look of the patient.

Another very striking case was that of a girl (M. L.[20]), aged twenty-two years, who was admitted to the hospital on January 28, 1908. The patient at first suffered from la grippe, followed by spontaneous perforation of the left drum membrane with purulent discharge. Four days before admission the right drum membrane was incised; this was followed by discharge. The temperature at the time ranged between 99° and 100°. On the day before admission the temperature rose to 103°, the patient complaining of marked deafness. There was pronounced nystagmus, some nausea and vomiting, and considerable frontal headache. On January 28 and 29 the temperature was between 101° and 102°. On January 30 the temperature was 103°; pulse, 90 to 104. On the same day operation was performed on the left mastoid. Pus was found in the cells of the tip of the mastoid, in the antrum, and over the sinus; the sinus itself appeared normal. On January 31 the temperature dropped to 101°. On Februray 1 it rose to 104°.

On the same day operation was performed for acute mastoiditis on the right side. There was found moderate necrosis of the cortex and of the tip. Some pus was found in the antrum. On February 2 the temperature was 103°. On February 3 the temperature rose

[20] This and the previous case occurred in the private practice of Dr. Seymour Oppenheimer.

to 104.4°. A blood culture was made, and three and one-half colonies of streptococci to the cubic centimeter of blood were found. On the following day, when the report was handed in, the temperature was 100.8°. On February 5 another blood culture was taken at a time when the temperature was 103°, and we found fifteen colonies of streptococci to the cubic centimeter of blood. We advised exploratory operation for sinus thrombosis.

But during the next week the patient appeared to be doing so well, the temperature not rising above 103°, as a rule, that the otologist did not consider that he was warranted in operating any farther on the patient for the time being. On February 13, at a time when the patient had a temperature of 103.4°, another blood culture was taken, and there were found to be present sixty colonies of streptococci to the cubic centimeter of blood. On the following day, when the report was handed in, the temperature was only 101°, and operative interference was again deferred. On February 15 the temperature rose to 103°, and on the 16th it dropped almost to 99°. A blood culture was taken on that day at 4 P.M., the temperature being 99.6°, and again there were found to be sixty colonies of streptococci to the cubic centimeter of blood. For the next two days the temperature did not rise above 100.4°, and the patient seemed to be doing remarkably well. On the morning of February 19 the temperature was 99°. At noon the temperature was 100°; in the afternoon the temperature began to rise.

On examination of the patient there was found to be tenderness in the region of the jugular bulb, and there were some enlarged tender lymph nodes along the course of the jugular vein. A blood culture taken at this time, 3.30 P.M., showed two colonies of streptococci to the cubic centimeter of blood. At 4 P.M., when the temperature was about 102.5°, the patient was operated upon. The jugular vein was exposed in the neck and part of it resected. About one-half inch above the entrance of the facial vein the wall of the jugular vein was white in appearance, very firm, and the vein appeared to be flattened. There was marked inflammation of the lymph nodes about the vein. A non-suppurative clot was found in the vein which closed the vessel as far down as the point of ligation and extended slightly on the wall of the vein for a short distance below the upper ligature. The temperature the same night rose to 103°, but dropped the next day to 99°, and never rose above 99.8° after that, the patient making a complete recovery. A blood culture made on February 22, three days after ligation of the jugular vein, proved to be negative.

This case is one of the most remarkable in the entire series which we have studied. The suspicion of a sinus thrombosis was aroused by the blood culture results over two weeks before it was clinically considered proper to operate for supposed sinus thrombosis. It is interesting to note that the streptococci gradually increased in

number, and then decreased even before the jugular vein was tied. If the jugular vein had not been tied at the time it was, we believe it is possible that the blood cultures might have become negative before the ligation was performed. There are a number of other points of interest about this case, especially in regard to the leukocyte counts, a discussion of which we must postpone for another time.

(*B*) In certain cases, after the sinus has been explored and a clot has been removed and the jugular vein has not been ligated, the persistence of streptococci in the blood has given the indication to tie the jugular vein, and, as we have previously stated, in almost all of the cases the bacteria then promptly disappeared from the blood. A case showing in a striking manner the value of such a finding will be given in the case (Levin) to be described under the next heading.

It should be noted here that while the bacteria usually promptly disappeared after ligation of the jugular vein, the clinical symptoms (particularly the fever) do not always as promptly abate. In some cases the patient will still have fever, due possibly to local conditions in the wounds, or due to metastases previously developed or being developed, or due to some other intracerebral complication or to some intercurrent disease.

We have already pointed out that in one case the bacteria did not disappear even after the jugular vein was ligated, but did disappear after the jugular vein was exposed, a clot removed, and the vessel packed. This case (Klein) will be described briefly under the next heading.

(*C*) The most valuable field for the blood culture studies has been in the border line cases, in which it would have been very difficult to come to a definite conclusion without the use of the cultures. The first case of this type, a male, aged seventeen years, was admitted to the hospital in the service of Dr. Rudisch on January 3, 1904. For the first three days after admission he had an almost constant temperature of 103° to 105°. For the following three days the temperature ranged between 100.5° and 103°, the pulse rate varying from 92 to 100. The history given was that the boy had suffered from headache and malaise for eight days. He had then become feverish and chilly and remained in bed. He had then developed abdominal pain for one day. Three or four days before admission he had severe shaking chills followed by sweating. The boy was sent into the hospital for supposed typhoid fever. On examination by one of us (Libman) on the day of admission the boy did not give the impression of suffering from typhoid fever. There were no rose spots present. There was no enlargement of the spleen. On thoroughly examining him it was found that he had a moderately marked otitis media on the left side.

The otologist was then called in to see the patient; he punctured the left drum, and a little seropus was evacuated. The leukocytes

numbered 17,600. There was some tenderness over the tip of the mastoid. The otologist did not believe that the otitis media by itself could explain the clinical picture. The Widal examination proved to be negative. In was then suggested that a blood culture be made so as to determine whether any organisms were present in the blood. It was thought that if typhoid bacilli were present in the blood the case would be regarded rather as one of typhoid fever with a complicating otitis media, but if streptococci were found in the blood it would be proper to advise operation upon the mastoid, because there was no other demonstrable focus found from which the organisms might be entering the blood current. At this time we believed that more extensive trouble might be found than there appeared to be according to the local findings. On January 4 the blood culture was taken, and on January 5 it was reported that streptococci were in the blood. The mastoid region was explored on the same day by Dr. Gruening. Not only was mastoiditis found, but there was a clot in the lateral sinus which extended down as far as the exposure would allow. A perisinuous abscess was also found. The patient's condition became so poor that the jugular vein could not be tied off. The temperature dropped next day to 100°, but during the next two days rose again to 102°. A blood culture was again taken on January 6, and streptococci were again found. At that time we did not understand the significance of the persisting bacteriemia, and did not urge further operation, but on January 8 Dr. Gruening, because of the persistence of the clinical symptoms, considered it advisable to tie the jugular vein. A blood culture taken on January 12 proved to be negative, as was also one taken on January 19. The patient developed a few metastatic abscesses which we believed were probably to be explained by the dissemination of bits of clot before the jugular vein was tied.

After the diagnosis in this case had been cleared up a history was obtained by careful cross-questioning of the patient and his relatives, which differed from that obtained on the admission of the patient to the hospital. Eight days before admission the patient had developed pain in the left ear. This pain lasted for three days. It was slight in the morning and moderately severe in the evening. The pain then disappeared, and the patient suffered from headache, which was first located in the left side of the head and then was frontal in character. He then developed abdominal pain.

We have previously in this paper drawn attention to the difficulties that might arise at times in the differential diagnosis between sinus thrombosis and typhoid fever. The complete question of differentiation, including the question of the clinical phonemena, we hope to take up on another occasion.

Shortly after seeing the case which we have just described, on January 21, 1904, a female patient, aged twenty-seven years, was admitted. The history given was that she was sick for fifteen days

suffering from severe occipital headache with stiffness and tenderness of the neck. She had had chills and fever coming on every second day and followed by profuse sweats. On examination of the patient perforations were found in both drum membranes. On the left side there was fairly marked tenderness behind the mastoid process and along the occipital ridge. There was no tenderness on the right side. There was no rigidity of the neck. On January 21 the patient had a chill and temperature of 105°. On January 22 the temperature was down to 102.4°; the leukocyte count was 13,000. On January 23 there was a chill in the early morning and a rise of temperature to 105°; the leukocyte count was 10,000. The spleen was palpable one finger below the free border; it was very firm. The otologist did not believe that in this case there was sufficient evidence of local trouble to warrant operation. The clinician in charge of the case was inclined to believe that the patient was suffering from malaria, but plasmodia could not be found.

We suggested that, before waiting for the result of treatment by quinine, it would be advisable to take a culture, which was accordingly done on January 23. On January 24 it was reported that there were streptococci in the blood. It was considered warrantable to explore the mastoid process, as there was a certain amount of suspicion from the local examination that there was more trouble there than the old otitis media, and because there was no other focus found from which the streptococci were gaining access to the blood current. The cortex of the mastoid was found thick and hard. In the antrum there was granulation tissue. The lateral sinus was found to contain a clot, part of which was decolorized. The clot extended around to the torcular. On January 25 the temperature was 98.8.° On January 26 the temperature was 102.8°. The margins of the wound were found to be necrotic. On the same day a blood culture was taken, which was found to be negative. The patient's temperature was normal by January 31, and she was discharged practically well on February 26. No metastases developed in this case.

A third illustrative case we will cite briefly: This patient was admitted to the hospital on February 28, 1909. He was a male, aged nineteen years, who had had pain in the left ear ten days before admission to the hospital; there was also profuse discharge. The following day the patient developed the same symptoms on the right side. At the time of admission to the hospital there was no discharge from the left ear, but the right ear continued to discharge pus and there was tenderness at the tip of the mastoid process. The patient stated that he had had several chills. On examination it was found that the drum membrane was reddened and there was a medium-sized perforation. There was tenderness over the tip of the mastoid process and slight tenderness in the region of

the jugular bulb.[21] The temperature was irregular from February 28 to March 1, reaching 103° and 104°, and on March 2 only reaching 101.4°. It was not believed by the otologist that there was sufficient evidence of local trouble to warrant operation. The examination of the patient otherwise was negative, and a blood culture taken on March 2 showed streptococci, there being seven colonies of streptococci to each cubic centimeter of blood. The patient was operated upon on March 3. Just before the operation streptococci were again found in the blood, there being nine colonies to the cubic centimeter. At the operation the mastoid cells were found only slightly diseased. The wall of the sinus was thickened. A large amount of white thrombus was scraped out of the sinus. Free bleeding was established from the cerebral end of the sinus, but only a moderate amount of bleeding occurred from below. The patient was in too poor condition to tie the jugular vein. A blood culture taken a few hours after operation showed 165 colonies of streptococci to the cubic centimeter of blood (in this case the postoperative increase can be fairly ascribed to the operative interference). The following morning the blood culture showed less than one colony of the streptococcus to the cubic centimeter of blood. On March 5 it was decided, because of the persistence of streptococci in the blood and the continuance of clinical symptoms, to tie the jugular vein, which was accordingly done. A drain was passed down into the bulb of the jugular vein from above. The following day there were still streptococci in the blood, 200 colonies to the cubic centimeter, and streptococci persisted in the blood until the patient died. At the autopsy no further trouble was found in the sinus or in the jugular vein, but an ulcerative endocarditis was present.

In some cases it is very difficult to come to a conclusion unless one judges carefully all the facts. We have had two cases in which the conditions were particularly complicated. Unfortunately, both patients arrived at the hospital in very bad condition, and died within a few days.

The first case was a female, aged seventeen years. She gave a history of fever and headache lasting for two weeks, being accompanied by severe dry cough. On the afternoon of admission the right ear began to discharge. On examination there was found profuse purulent discharge from the right ear. There was tenderness over the tip of the mastoid and some tenderness along the jugular vein. The examination of the fundi showed some dilatation of the veins on both sides, more marked on the right side. The leukocyte count was 14,200; polynuclears, 76 per cent. The temperature on admission was 105°. On examining the lungs, crepitant

[21] In a subsequent communication by one of us (Libman), dealing with a method for testing patients for their sensitiveness to pain, it will be shown that much stress cannot be laid on tenderness in the region of the jugular bulb unless the patient is normally hyposensitive.

rales were found at both bases. At the left base there was dulness, and on auscultation numerous rales and a rather large area of bronchial breathing. In this case the ear surgeon was not ready to operate because of the presence of the pulmonary phenomena, particularly on the left side. It was decided to wait before exploring the mastoid process until the blood culture was made. If pneumococci were found, it was decided that they might be considered an accompaniment of the lung condition. But if streptococci were found, it was considered that it would be justifiable to explore the mastoid region, as it was possible that they were gaining their entrance from that point, and the lung lesion might be secondary. The blood culture was taken on the day of admission at 7.30 P.M., and the following morning streptococci were found (one colony to the cubic centimeter). The mastoid process on the right side was explored. Granulations and pus were found. Pus was found in the lateral sinus. The jugular vain was then ligated. The patient died the following morning. A postmortem examination was not permitted.

The other case was that of a man,[22] who was admitted to the hospital January 30, 1907. The patient had suffered for a number of days from a severe inflammation of the alveolar border of the right lower jaw, with an enlargement of the submaxillary lymph nodes. A few days later he developed pain in the right ear. A puncture was made, but no pus was found. A few days later there was a discharge of pus. The patient had high temperatures for several days before admission. There was tenderness and swelling of the muscles of the right calf, and the skin over them was reddened. There was but little mastoid tenderness. The patient was evidently severely sick and it appeared that the trouble in the right calf was a developing metastatic abscess. There was also some pain and redness of the first joint of the left thumb.

The question arose as to whether the metastatic abscess was due to infection of the sinus or to the infection of the lower jaw. We argued that in this case the presence of streptococci in the blood would suggest an exploration of the sinus, because in our experience the infections of the lower jaw, particularly those at the alveolar border, do not give a bacteriemia, and because one does not generally find secondary foci in such cases. Further, we knew that infections of the sinus were apt to be accompanied by a bacteriemia and that in some cases of otitic infection there was a tendency for metastatic processes to be established in the muscles.

The otologist decided in this case not to take any chances by waiting because the patient was so severely sick, and the patient was operated upon on the day of admission, a blood culture being taken just before the operation. At the operation a purulent clot was found in the lateral sinus and a clot in the upper part of the

[22] This case was studied through the courtesy of Dr. Carl Kaller.

jugular vein. The jugular vein was resected. Streptococci were found in the blood (two and one-half colonies to the cubic centimeter). Streptococci were again found in the blood a few days later (three colonies to the cubic centimeter). The patient died a few days later; postmortem examination was not permitted.

These cases show clearly in what manner we have attempted to utilize the positive results in helping to diagnosticate doubtful cases. As we have already pointed out, it would seem from our studies that the streptococci remain in the blood in the cases of sinus thrombosis either because there is still trouble present in the veins or because endocarditis has been established. The establishment of endocarditis in these cases occurs, according to our experience, very infrequently. Multiplication of streptococci as a possible cause of persistence of streptococci in the blood, after proper attention has been paid to the local focus, must also be reckoned with as a cause of persistent bacteriemia. We have not found any instance of this occurrence in the series of cases upon which our report is based.

2. Significance of Negative Blood Cultures. (*A*) We have shown that negative results can be obtained, even though not frequently, in cases in which a sinus thrombosis is present. The absence of bacteria in a doubtful case would make one hesitate in exploring for a thrombus until the possibility of all intercurrent diseases had been excluded, or until the symptoms had become so severe as to justify an exploratory procedure.

(*B*) If the blood culture should be negative and the symptoms should persist in a given case, whether there is a sinus thrombosis or not, acute endocarditis can be excluded. In such cases it is advisable to take two blood cultures. In our experience in cases of acute endocarditis due to ordinary streptococci (and staphylococci) bacteria are always found in the blood if two cultures are made.

(*C*) As we have already stated several times, if there has been a sinus thrombosis and bacteria have been present in the blood, and the jugular vain has been tied, a negative culture is of value in showing that the general invasion has been stopped.

(*D*) Occasionally a negative blood culture has been of value in cases with a clinical picture of rheumatism. It is very valuable in such cases to know that we are not dealing with an arthritis due to a general invasion by the ordinary bacteria.

The data which we have given are sufficient to show the great value of the investigation by means of blood cultures in cases of otitic infection. It is important to know that in no line of work are routine rules more disadvantageous in attempting to draw conclusions. Each case must be made the subject of a careful clinical and bacteriological study. We hope that our work will stimulate others, who are in possession of larger material, to investigate their cases according to the methods which we have suggested.

Most of the cases which form the basis of this paper occurred in the service of Dr. E. Gruening, to whose kind coöperation we owe the possibility of having made our studies. The recent cases were investigated through the kindness of Dr. F. Whiting. We are also much indebted to Dr. S. Oppenheimer for the privilege of making studies in a number of his cases and for the use of the clinical histories.

Addendum.—Since reading this paper we have made cultures in 17 additional cases of otitis media and mastoid disease. In only 1 case were streptococci found in the blood, and in this case at operation a purulent thrombus was found in the lateral sinus. The streptococci disappeared from the blood within seven hours after operation. We have therefore now had 75 cases of otitis media or mastoid disease without sinus thrombosis or meningitis, and in all these cases the blood has been sterile.

Drs. A. Duel and J. Wright,[23] of New York, reported a series of blood cultures made in cases of otitic disease, in which they obtained positive results in cases of uncomplicated otitis media and mastoid disease. The method used in the study of these cases was somewhat different from our own, inasmuch as the authors in most of their cases made a practice of incubating 10 c.c. of blood in flasks containing 150 c.c. of bouillon or glucose bouillon; after two to three days 15 to 30 c.c. of the contents of the flask was centrifuged and cultures were made on solid media from the sediment. Before reading the paper, Drs. Duel and Wright were kind enough to inform us of the method which they were using in the study of their cases, and since that time we have employed it in 11 cases of otitis media and mastoid disease, comparing it with our own method. The results were identical, all the cultures being negative. Besides this we made a study of 65 cases of local infections in different parts of the body of a type which, according to our experience, is unaccompanied by a bacteriemia, using both the strong concentration of the blood, as suggested by Dr. Wright, and also employing our own method. In all these cases the blood cultures were negative.

[23] At a meeting of the American Otological Society held in Boston, June 1, 1909.

TUBERCULIN-TREATED GUINEA-PIGS IN THE RECOGNITION OF TUBERCULOSIS.

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The experiments upon which this paper is based were undertaken in order to determine to what extent the tuberculin reaction in guinea-pigs might be of value in the diagnosis of human tuberculosis. The problem which presented itself was to reduce the length of time required for the determination of tuberculosis in guinea-pigs that had been inoculated with material containing tubercle bacilli.

The method now most commonly used is to introduce the suspected material either into the peritoneal cavity or the subcutaneous tissue of the abdomen of a guinea-pig, and after four to eight weeks examine the animal for tuberculosis. Block[1] modified this procedure by introducing the material in the region of an inguinal gland and then, by squeezing the gland, so lower its resistance that he could recover bacilli in nine to eleven days. F. Klemperer,[2] by a similar method, except that he did not squeeze the lymph gland, found tubercle bacilli in stained sections of it in ten to fourteen days. He found, in using Block's method that the pigs often died of mixed infection. Nattan-Larrier found that if virulent tubercle bacilli were injected into the base of the mammary gland of a lactating guinea-pig the bacilli could be found in the milk in from five to ten days. An indirect test has been described by Mérieux,[3] who obtained the tuberculin reaction in tuberculous guinea-pigs after the injection of serum from a tuberculous patient.

Anderson,[4] while determining the presence of tubercle bacilli in milk, found if he injected 2 c.c. of crude tuberculin into guinea-pigs two months after they had been inoculated with tubercle bacilli, that death almost invariably followed in six to eighteen hours. Guinea-pigs with slight lesions in two or three instances did not die, but became sick. All of the animals died when the lesions had caseated. As much as 7 c.c. given to a healthy guinea-pig caused only temporary discomfort. Anderson believes this test of distinct service in differentiating tubercle bacilli from other acid-fast bacilli. The statement has been made by Wolff[5] that while 0.5 to 2 c.c. of tuberculin will be tolerated without discomfort by a healthy guinea-pig, an injection of 0.1 to 0.15 c.c. will cause the death of a tuberculous pig within twenty-four to forty-eight hours. Attempts to utilize

[1] Berl. klin. Woch., 1907, No. 17, 511.
[2] Ibid., 1908, 171.
[3] Revue de méd. 1906, xxvi, 2.
[4] Hygienic Laboratory, Bulletin, No. 41, United States Public Health and Marine Hospital Service, January 1, 1908.
[5] Berl. klin. Woch., 1908, 297.

the von Pirquet reaction in guinea-pigs have been nearly uniformly failures.

I endeavored first to determine how soon after being inoculated a pig would die from the tuberculin reaction. Injections of tuberculin in doses of 0.25 c.c. and 0.5 c.c. given to healthy guinea-pigs resulted in no appreciable effects. Three guinea-pigs were then inoculated under the skin of the abdomen with an emulsion of very small numbers of tubercle bacilli. At the end of eight, twelve, and fifteen days the animals were given in turn 0.25 c.c. of tuberculin. The one given tuberculin on the eighth day showed no effects at the end of twenty-four hours, and even gained slightly in weight. The one given tuberculin on the twelfth day was sick the next day, but recovered. The nodule at the site of inoculation was larger than it had been the day before, and the pig lost slightly in weight. The one given tuberculin on the fifteenth day was sick the two following days and in other respects behaved like the one preceding, losing slightly in weight and showing a larger harder nodule on the abdomen. All three pigs were autopsied on the seventeenth day. In each there was slight caseation at the site of inoculation and much serous exudate in the adjacent cellular tissue. In this exudate, as well in the caseous material, very large numbers of tubercle bacilli were found. In the pig that had received tuberculin on the eighth day there were miliary tubercles in the liver and spleen. In all the pigs the inguinal lymphatic glands were caseous and contained many tubercle bacilli.

Having failed to get satisfactory results by awaiting the death of the guinea-pigs, but judging that the very large numbers of bacilli found at autopsy were in some way due to the tuberculin, a second series of pigs was inoculated with 0.5 c.c. of an emulsion of tubercle bacilli made so dilute that only two or three bacilli could be found microscopically in an hour's search. These pigs were given 0.25 c.c. of tuberculin at various intervals and chloroformed and autopsied at the end of twenty-four hours. In this manner tubercle bacilli were recovered from the point of inoculation in the subcutaneous tissue as early as the sixth, seventh, eighth, and ninth days. Pigs autopsied on the fourth and fifth days were negative. In one killed on the third day three bacilli were found in a search of an hour and a half. A dose of 0.25 c.c. of tuberculin is such an overwhelmingly large one for a guinea-pig that it was found that size or age of the animal made no difference in the results.

Attention was then given to the possible clinical value of the foregoing suggestions. Pigs were inoculated with suspected material by injecting an emulsion of it under the skin of the left side of the abdomen, the other side being left for the subsequent subcutaneous injection of tuberculin. Each pig was chloroformed and autopsied twenty-four hours after the latter injection. The results were as follows:

1. Pus from empyema. Microscopically no tubercle bacilli; 2.5 c.c. of emulsion of this injected into pig. Tuberculin sixth day. Autopsy, seventh day. A few tubercle bacilli found at site of inoculation.

2. Fluid from tenosynovitis of knee of long standing. No tubercle bacilli microscopically. Tuberculin sixth day. A few tubercle bacilli found at site of inoculation on the seventh day.

3. Suspected tuberculosis of the knee of two years' duration. Not possible to demonstrate tubercle bacilli in fluid from the joint. First pig given tuberculin on sixth day. Autopsy on seventh day. Negative result. Second pig given tuberculin on fourteenth day. Death of animal in twenty hours from reaction. Very large numbers of tubercle bacilli at point of inoculation, which showed caseation and much serous exudate. There were also miliary tubercles in the liver and spleen.

4. Suspected tuberculosis of the ankle-joint. Scrapings emulsified and injected. No tubercle bacilli microscopically. Tuberculin on seventh day. A few tubercle bacilli found at autopsy on the eighth day.

5. Suspected tuberculosis of the kidney. Patient a woman, aged thirty-four years, considerably emaciated, slight irregular fever and a large tender displaced right kidney. The urine contained pus and blood and very large numbers of acid-fast bacilli which were decolorized by alcohol. Two pigs inoculated. Both given tuberculin on the fourteenth day. An abscess had formed under the skin. Pus from this showed no tubercle bacilli after the tuberculin. Pigs killed six weeks after inoculation showed no tuberculosis. Patient has recovered.

A second group showed negative results:

1. Scrapings from fistula into suspected tuberculous hip-joint. Pigs examined on seventh and sixteenth days.

2. Scrapings from fistula into suspected tuberculous hip-joint. Pig examined on seventeenth day.

3. Fluid from symptomatic hydrocele in case of suspected tuberculous testicle. Sections of the testis did not reveal tuberculosis. Pigs examined on the seventh, fifteenth, and thirty-first days.

4. Emulsion from caseous peribronchial gland, which contained tubercle bacilli microscopically, from a child dead of tuberculous meningitis. One pig examined on the eleventh day. The failure in this case suggested the possibility that the child had died of bovine tuberculosis and that tuberculin from human bacilli had no effect.

Of the 62 guinea-pigs used, none died of mixed infectious. One died from tuberculin reaction on the fourteenth day. One with advanced pulmonary tuberculosis of eight weeks' duration, in which there was caseation, survived 0.25 c.c. of tuberculin four days. Pigs No. 51 and 52 were inoculated with the same quantity of the same material at the same time. Both were autopsied on the seventeenth

day, one having been given tuberculin the day before. In both tubercle bacilli were found at the point of inoculation, but by far the larger number were present in that pig to which tuberculin had been administered. It appears that overwhelming doses of tuberculin given to a guinea-pig the subject of tuberculosis breaks down its resistance to the tubercle bacilli and results in their much more rapid multiplication.

The method employed in carrying out this test has simplicity to commend it and the fact that positive results are conclusive. The technique consists of making an emulsion of the suspected material in normal salt solution, if it is not already fluid enough to be injected through a course syringe or sharp-pointed improvised pipette. Three guinea-pigs are inoculated by injecting a considerable quantity under the skin of the abdomen. At the end of six days one pig is given 0.25 c.c. of tuberculin hypodermically on the other side of the abdomen, and is chloroformed at the end of twenty-four hours, and autopsied. The fluids at the site of the original inoculation are smeared upon a slide and examined in the ordinary way for tubercle bacilli. If the first pig results negatively, another is examined in the same way by giving tuberculin at the end of two weeks, and making the autopsy at the end of twenty-four hours. If this is also negative, the third pig is given tuberculin at the end of four to six weeks, and the result of its examination is a check upon the first two.

The test offers a means, in a considerable proportion of cases, of making a diagnosis at the end of a week instead of awaiting until the lapse of four to eight weeks. It also presents a method of distinguishing tubercle bacilli from other acid-fast bacilli, especially when, as often happens, they are present in large numbers.

REVIEWS.

THE FAITH AND WORKS OF CHRISTIAN SCIENCE. By the author of *Confessio Medici*. New York: MacMillan, 1909.

THE author of that delightful book *Confessio Medici*, a little volume which received less notice than its charm of style and contents deserved, has made an examination of the pretensions of Christian Science, that is amusing, instructive, and enlightening. It is probable that no book, however well done, will convert any believer from the queer faith of Eddyism, but a work like this may serve at least as a prophylactic measure. The large number of the half-informed will find here cool consideration of the foggy inconsequences, the feminine illogicalities, the crude half truths of the New Thought, with an amazing list of the kind of cures on which it founds its claims of miraculous healing. The most miraculous thing about the whole matter is that in a nation with so highly developed and so widely diffused a sense of humor as the American, many thousands of people can be found who can read, nay, have some sort of feeling which they call belief in, such preposterous juggling with words as this, for example:

"Erudite systems of philosophy and religion melted for Love unveiled the healing promise and potency of a present spiritual afflatus." "Unveiling" an "afflatus," and a spiritual one at that, must be a really remarkable feat. With such scraps from a feast of languages the author plays pleasantly now and then, but there is enough of serious criticism, too—criticism of the indecent parody by Christian Science of the Christian Faith; criticism of the intellectual cruelty, of the contempt for the weak, which is one of the results of the crooked upside down nonsense about the absence of pain, of death, of sickness; criticism of a most caustic kind of the contradictions of which it is so full; and finally, a list of 200 cases of "cures," given with little comment. Most of these for the doctor carry their own criticism, and although many quotations might be made from the book, we will select only a few of these amazing case histories as samples:

Case 2: "Headaches" and "bowel trouble" healed after two years of Christian Science treatment." Every practitioner knows as a matter of daily experience that patients mean constipation when they speak of "bowel trouble"—so this seems a longish spell of treatment. *Case* 18 was healed of "stomach trouble" and of headaches.

"Stomach trouble" is another dispensary synonym for constipation. *Case* 34 broke one of the bones of her leg. It was properly set and bandaged by a medical man. In *three weeks* she began to walk round the room. *Case* 37 for ten years had a "complication of diseases." Also had weak eyesight and "stomach trouble." *Case* 38 for ten years studied "mysticism, occultism, and Vedastic philosophy." At the end of this time she felt "confused, restless, impatient, irritable, and nervous!" Healed.

The book will furnish admirable ammunition for anyone who wishes to argue the Christian Science question, and is excellent reading besides. J. K. M.

THE EMMANUEL MOVEMENT IN A NEW ENGLAND TOWN. By Rev. L. P. POWELL. New York: Putnam, 1909.

THE Rev. Lyman P. Powell, who has written books on Christian Science and on the art of sleep, has cultivated successfully the Emmanuel movement and keeps it going in Northampton. He starts off with gathered quotations from doctors who have deprecated the excessive use of drugs as formerly practised, in a page which sums them up, as if Sir Frederick Treves, Hayem, Metchnikoff, Behring, Roux, Erlich, Barker, and others had condemned the use of drugs altogether. It would be curious to examine the statements in the originals from which he takes these views. It is certainly interesting that of the seven continental names which he quotes, at least five are associated in the minds of the profession with treatment by physical agencies differing widely from the purely mental means of the Emmanuel movement. We do not count Dubois among the authorities, although, with that lack of perspective and real scientific knowledge which characterizes the use of authorities in Dr. Worcester's *Religion and Medicine*, his pupil has spoken of Dubois as if Dubois and Hayem, Behring and Erlich, were all in one class.

It is difficult to criticise in any reasonable fashion the kaleidoscopic changes of the Emmanuel Movement and the rapid changes of its promoters, at first independent of the doctors, although with a kind of dummy board of directors, then with a staff of attendants and a combination medicospiritual clinic, then receiving patients with the approval of the doctors, and finally giving up the church feature altogether and abrogating clinics, and now only receiving patients when recommended directly by physicians.

Mr. Powell, however, has at least a better atmosphere in his book than prevails in Dr. Worcester's. It gives an impression of honest effort, without an eye on the gallery, and none of that general effect of promoting a clearing house for nervous diseases, which some

neurologists thought was likely to be the result of the Boston movement. It is perhaps some such fear that has caused the changes in Emmanuel Church itself. Like the rest of the followers of the Emmanuel movement, he calls a good deal on great names and conjures with Professor William James and Dr. Weir Mitchell. Perhaps, like Dr. Worcester, he has forgotten that while Professor James has taken to spiritualism and the worship of all sorts of strange gods, Dr. Mitchell has busily and publicly denied approving of the Emmanuel movement or having anything to do with its promotion beyond those suggestions which Dr. Worcester drew from the neurologist's published works.

Mr. Powell is not so inclined to sneer at the doctors as Dr. Worcester and his assistants, but he remains equally astonished with his teacher at having secured results which every doctor who knew his business and who had especially studied this kind of thing had been securing for some thousands of years. One observes with pleasure that he mentions that he has had failures. From most of the Emmanuel literature one would suppose that the treatment had this divine quality, if no other, that it could never be wrong.

J. K. M.

The Matter with Nervousness. By H. C. Sawyer, M.D., San Francisco: Cunningham, Curtiss & Welch, 1909.

This is an awkwardly extravagant little book, fantastic in its language, yet full of common sense, and driving home, even with its extravagance, excellent lessons in the hygiene of nervousness. In the introduction the author puts the case thus; "Priests, psychologists, laymen, are all taking a hand at it (nerve cure); only the doctors are a little abashed. . . . Why should our lay brothers feel impelled to discuss nerve cure? It must be because we want something in this field; we are not always wise, earnest, helpful here. . . . There is one lesson we all need to learn and to heed: every movement away from the authority and the care of scientific medicine marks some real limitation of medical science or else some limitation of the individual men and women who represent it. . . . There are three medicines, the medicine of the spirit, the medicine of the mind, and the medicine of the body. . . . Our gospel today . . . says and insists and repeats even to weariness two pregnant truths. Nervousness is no merely functional trouble; there are no merely functional troubles; . . . whether it be slight or severe, transient or permanent, [it] is always a surface sign of deeplying bodily deteriorations that are as real as the fracture of a bone. And nerve cure is no marvel of miracle-making for modern men, no mere parlor magic, [but] a struggle just as real, just as earnest, just as desperate sometimes as bone cure is."

To the neurologist this is almost a truism, but certainly it is a lesson that needs enforcing on the general practitioner. Dr. Sawyer takes up the various items of original faulty material, defective construction, wear and tear of the nervous apparatus, and goes into all the details of nose, throat, tooth, womb, eye, and ear disturbances that make for nervousness, the external poisons, the chronic fatigue. They are all listed, but without much perspective and with scarcely sufficient stress on the central fact which, nevertheless, the author evidently wishes to insist upon, namely, that any of these causes are active for ill only when the resistance is lowered in some way. In places this is said in the book, but the whole tone and style is so forced and one might say turgidly colloquial that the point is sometimes missed. The best thing in it is clearly and well put thus: "The largest nerve cure is that which tries to comprehend all the forces that make and mar the nerves; the wisest is that which discovers the individual need that exists in every case—and ministers to it."

J. K. M.

A Text-book of Physiological Chemistry for Students of Medicine and Physicians. By Charles E. Simon, B.A., M.D. Third edition. Philadelphia and New York: Lea Bros. & Co., 1908.

The Elements of the Science of Nutrition. By Graham Lusk, Ph.D., M.A., F.R.S. (Edin). Philadelphia and London: W. B. Saunders Co., 1908.

Chemical Pathology, being a Discussion of General Pathology from the Standpoint of the Chemical Processes Involved. By H. Gideon Wells, Ph.D., M.D. Philadelphia and London: W. B. Saunders Co., 1908.

The three books mentioned in the heading constitute together a very useful mass of information for either advanced student or practitioner. We are now in a period when it is generally recognized that studies of the body chemistry constitute one of the most important branches of investigation in medicine, and while the importance of other lines of work is sometimes slighted in the enthusiasm over work of this class, it cannot be denied that more advances are probable along this line than along most others in the course of the next few years, and certainly more have already occurred along this line recently. The work by Simon is more particularly valuable in the class-room in giving students a clear comprehension of the laboratory side of physiological chemistry. It discusses general questions in relation to physiological chemistry and subsequently gives a chemical discussion of the albumins, carbohydrates, and fats, of the cleavage products of the albumins, of ferments, of the diges-

tive fluids, and the processes of digestion and absorption, of the feces, urine, blood, muscles, nervous tissue, and so forth. It is clearly written and constitutes a very useful text-book on this subject.

As Hofmeister remarked when first putting forth his Beiträge, we may speak of physiological chemistry on the one hand and of chemical physiology on the other hand, and he intended his Beiträge to deal more particularly with the latter subject; the distinction being, of course, that on the one hand the matter was considered chiefly from the chemical standpoint, while from the other point of view chemistry was to be used as the handmaid of physiology. The second and third books under discussion belong to the latter group, in which the attitude is that of dealing with physiology or pathology, using knowledge obtained almost entirely through chemistry in furthering these subjects, but presenting it entirely from the standpoint of the medical sciences rather than from the standpoint of the chemist. Professor Lusk's book is an extremely valuable one and one that cannot fail to be of great use to many men in medical science and to practically every man in the practice of medicine, if he wishes to have a comprehension of that which is in many ways the most important and valuable of all things for him to comprehend, namely, the manner in which the nutrition of the body is accomplished and the methods of furthering or limiting this, together with a discussion of some of the most important disturbances of nutrition. So far as the practitioner is concerned, it is presented with perhaps a little too much of the attitude of the physiologist, but aside from this criticism the book is one that will be of very great service to anyone who desires to gain greater familiarity with the subject; and the fact that it is presented from the standpoint of the careful and precise scientific worker will make it in many ways all the more valuable to the clinician, because there has been altogether too much vague and indefinite impression as against precise investigation prevalent in the clinical study of matters of this kind, and it is only by exact investigations of metabolism that reliable working conclusions can be reached, whether it be for clinical purposes or for more purely scientific purposes. As an example of the truth of the latter statement one may refer to the common indefiniteness in dietetic regulations and to all the vague and erroneous speculation that has gone on in regard to the purin metabolism in its relation to gout, which investigation has so largely checked progress because so many inaccurate conceptions have been exploited and acted upon as a consequence of most unsatisfactory and inexact studies. Dr. Lusk's book takes up seriatum, after an introductory chapter, the study of the feces, starvation, the regulation of temperature, the influence of proteid food, the specific dynamic action of the foodstuffs, the influence of the ingestion of fat and carbohydrate, the influence of mechanical work on metabolism, and then discusses—points that should be most carefully studied by every clinician—the

normal diet and the food requirement during the period of growth, and then metabolism in anemias, in diabetes, in fever, in gout, and other conditions. One point about the book that will be of much value to many persons, and that is only too often eliminated in American books, is a reasonably full and very good working bibliography.

The latter point is shared by Dr. Wells' book on chemical pathology. This is on a subject that is so constantly shifting and changing its position because of the extremely active investigation that is going on in it that any book written on the subject must necessarily be likely to have a limited life, since a very short time serves to alter the point of view in regard to many particulars. Nevertheless, there is much in the book that remains permanent knowledge, since it is based upon well-established facts. It is chiefly in the details that the point of view will change frequently from time to time. Nevertheless, the fact that change must occur rapidly and that therefore books in regard to such subjects are of transitory life is true of any questions that are going through especially active periods of investigation and that are therefore questions of great importance at the time; hence books on such subjects become the more essential in order to have a comprehension of the movements that are occurring. This book in many ways meets the demand that physicians so often make, the demand, that is, for some general discussion that will make comprehensible to the practitioner who has no practical knowledge of chemistry or chemical physiology or pathology the great mass of clinical or semiclinical literature that he sees, which relates to this subject; and a discussion that will at the same time give him a philosophical comprehension of the chemical side of physiology and pathology. The book takes up the chemistry and physics of the cell and of the cell constituents, then considers enzymes and the manner of their action and their general importance in relation to the body processes; bacteria are then discussed from a chemical standpoint, including a consideration of questions of immunity, the chemical means of defence, inflammation and other elementary pathological processes, the disorders of circulation and diseases of the blood, œdema, and the chemistry of tumors; and then a discussion of pathological states, such as uremia, acid intoxication and other intoxications, and the auto-intoxications of gastro-intestinal origin are set forth; finally, the actual diseases of the thyroid, hypophysis, and adrenals, gout and diabetes are discussed at some length.

As has been indicated, these books together present a view of an extremely active field of investigation in a very satisfactory and comprehensive way. D. L. E.

Progressive Medicine, A Quarterly Digest of Advances, Discoveries, and Improvements in the Medical and Surgical Sciences. Edited by Hobart Emory Hare, M.D., Professor of Therapeutics and Materia Medica in the Jefferson Medical College. Assisted by H. R. M. Landis, M.D., Demonstrator of Clinical Medicine in the Jefferson Medical College, Philadelphia. Vol. II, 1909. Pp. 317. Philadelphia and New York: Lea & Febiger, 1909.

Vol. II of *Progressive Medicine* for 1909 opens with an excellent discussion of hernia (42 pages) by William B. Coley. Special attention is directed to hernia associated with undescended or maldescended testicle, the radical cure of hernia, accidents occurring in hernia operations, pseudo-incarceration of hernia, and diaphragmatic hernia. Edward M. Foote devotes 78 pages to the surgery of the abdomen exclusive of hernia, especially shock, early rising and thrombosis and embolism after abdominal operations, postoperative ileus and peritonitis, enteroptosis, dilatation of the stomach, gastric and duodenal ulcers, intestinal anastomosis, appendicitis, diverticulitis, megacolon (Hirschsprung's disease), angiocholitis, abscess and cirrhosis of the liver, cholelithiasis, pancreatitis, etc. John G. Clark discusses recent advances in gynecology (74 pages), especially cancer, fibroid tumors, endometritis, metritis, uterine hemorrhage, dysmenorrhœa, sterility and amenorrhœa of obesity, tubal pregnancy, gonorrhœa, genital tuberculosis, etc. Alfred Stengel devotes 70 pages to diseases of the blood and hemopoietic system and disorders of metabolism, especially pernicious anemia, chlorosis, splenic anemia and Banti's disease, polycythemia, Hodgkin's disease, exopthalmic goitre, gout, diabetes, etc. Edward Jackson devotes 26 pages to ophthalmology, especially the ophthalmic reaction to tuberculin and other bacterial extractives, keratitis, inflammations and other diseases of the uveal tract, glaucoma, diabetic cataract, night blindness and other disorders of the retina, etc. The volume is well up to the standard of its predecessors, and the series as a whole is a virtual necessity to him who will keep abreast of progressive medicine.

A. K.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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The Pathogenesis of Pernicious Anemia.—Berger and Tsuchiga (*Deut. Archiv f. klin. Med.*, 1909, xcvi, 252) have investigated cases of "cryptogenic" pernicious anemia—cases in which autopsy revealed no adequate cause of the anemia. Tallqvist has shown that Dibothriocephalus latus, which may cause an anemia similar to pernicious anemia, contains a powerfully hemolytic lipoid which can be extracted from the body of the worm, and has been able to demonstrate quite similar substances in the mucosa of the human digestive tract. Berger and Tsuchiga have pursued investigations of like nature, the results of which follow: (1) In pernicious anemia there is present in the gastric and intestinal mucosæ a lipoid substance soluble in ether, which is about ten times as powerful a hemolysin as the lipoid obtained from the normal mucosa. (2) Tested experimentally on animals, this lipoid shows weak but definite hemolytic properties, when administered either subcutaneously or by the stomach. That obtained from the normal mucosa exerts much less action or none at all. (3) The resulting anemia has the characteristics of pernicious anemia. (4) In dogs it is possible to obtain a lipoid similar in action to that in pernicious anemia, after first producing a gastro-intestinal catarrh. (5) The conclusion seems justified that the origin of the so-called cryptogenic form of pernicious anemia is to be found in the hemolytic action of this lipoid material, with secondary insufficiency of the bone marrow. (6) The place of origin of this powerfully hemolytic lipoid is in all probability the gastro-intestinal mucosa; the cause of its production seems to be a chronic inflammation of the mucosa.

Paroxysmal Hemoglobinuria.—Meyer and Emmerich (*Deut. Archiv f. klin. Med.*, 1909, xcvi, 287) have made a careful and comprehensive study of four cases of paroxysmal hemoglobinuria, confirming the work of Donath and Landsteiner, Hoover and Stone, and others, adding some that is new. They demonstrated the autohemolysin described by Donath and Landsteiner in all four of their cases. In some instances this has been missed by other observers, but the authors find that these negative results are probably due to the fact that often, and especially after a paroxysm, the complement is largely, if not quite, exhausted in the patient's blood. Hemolysis may, however, always be obtained by adding fresh complement. Local lowering of temperature leads to the formation of new complement in the body, probably locally, so that one attack does not prevent an early recurrence of the attack. A few days after a paroxysm the blood contains the normal amount of complement. During the paroxysm both systolic and diastolic blood pressures rise, even before the chill, to decline with the height of the fever. Between attacks there is a lymphocytosis of 30 per cent. to 35 per cent. During an attack the lymphocytes are diminished to 9 to 10 per cent., the decrease being parallel to the severity of the attack; at the same time the eosinophiles diminish or disappear from the blood. The dissolved hemoglobin is excreted partly as urobilinogen. The hemolytic amboceptor is bound to the red blood cells during cooling; it can be separated from them by repeated washing with warm normal saline solution. The red blood cells of patients with this disease are less resistant to changes in temperature and to dilute acid and dilute saponin solutions than those of a normal individual. Toward cold alone they are not more sensitive. Against the patient's hemolysin, however, his own red blood cells are more resistant than normal red corpuscles, which are usually agglutinated by the patient's serum. The serum of the hemoglobinuric possesses hemopsonins, since it causes normal macrophages to phagocytic red blood cells.

The Value of Schurmann's Color Reaction in Lues.—Galambos (*Deut. med. Woch.*, 1909, xxxv, 976) has investigated the color reaction for the diagnosis of syphilis, recently proposed by Schürmann. He finds that the reaction has nothing to do with the luetic or non-luetic nature of the patient's blood serum, and that it is, therefore, valueless as a diagnostic test.

Disseminated Miliary Tuberculosis of the Skin.—Wilder Tileston (*Archiv. Int. Med.*, 1909, iv, 21) calls attention to the eruption of disseminated miliary tuberculosis of the skin in infants and its importance. Of 32 cases of tuberculosis, osseous, glandular, pulmonary, intestinal, peritoneal, and meningeal, 7 (21 per cent.) showed the eruption. All the children with spots which came to autopsy were found to have miliary tuberculosis. The eruption consists of scattered discrete papules, which at the beginning are the size of pin-heads, and are soon capped by tiny vesicles with purulent contents. Later, the vesicle ruptures or dries up, and its place is taken by a crust. When healed, a tiny white scar surrounded by a halo of brownish pigment remains. They appear in crops so that all stages may be present at

the same time. The distribution is fairly constant, the majority being situated on the buttocks, genitalia, and adjacent portions of the thighs. This is probably due to irritation by urine and feces, with consequent lowered resistance of the skin. Histologically, they vary from necrotic areas to tubercles and caseation. The cause is the tubercle bacillus. It was demonstrated in 70 per cent. of the cases. It probably reaches the skin by the blood stream. This specific eruption Tileston claims is of great diagnostic importance, and of even greater prognostic value than the positive tuberculin reaction. It indicates almost certainly a fatal outcome.

The Condition of the Pancreas in Achylia and Anacidity of the Stomach.—Ehrmann and Lederer (*Deut. med. Woch.*, 1909, xxxv, 879) point out the widespread belief, based on Pawlow's experiments, that hydrochloric acid is the specific stimulant of pancreatic secretion. In a large number of cases of achylia and anacidity they have tested the gastric contents and feces by Fuld's method and have found an actual increase of trypsin the rule. Their observations were made on 98 patients. The following results were obtained: (1) In achylia and anacidity the pancreatic function, contrary to the general supposition, is not diminished and is often increased. (2) It is, therefore, unnecessary to give hydrochloric acid to stimulate the pancreas in these conditions. (3) The author's experiments indicate that HCl is not the specific stimulant for pancreatic secretion. (4) With Volhard's oil breakfast and Ewald's breakfast, trypsin is usually demonstrable in achylia and anacidity. (5) In hyperacidity it is usually impossible to demonstrate trypsin in the gastric juice. (6) The more bile the gastric contents hold, the richer the trypsin. (7) Exceptions to (6) are not infrequent. (8) The absence or diminution of trypsin in hyperacidity is probably not due to acid alone, but possibly to an absolute decrease in ferment secretion and to the pyloric action. (9) Whether fats cause a physiological passage of pancreatic juice into the stomach of man, as is the case in the dog, remains undetermined.

The Influence of Radium on Uric Acid Salts.—Gudzent (*Deut. med. Woch.*, 1909, xxxv, 921) has shown in a previous communication that (1) uric acid forms two groups of primary salts, which can be separated solely by their different solubility. The first group (*a*-salt) changes at the time of its production into the second group (*b*-salt), which is more stable. The most probable cause of this change is an intramolecular rearrangement. (2) The *a*-salt, the first to be formed, has a solubility, which at 18° C. is 33.4 per cent. greater than the *b*-salt, while at 37° C. it is 33.9 per cent. more soluble. In the blood, owing to physical chemical laws, it so happens that the solubility of each of these salts is less than in water, the uric acid being present in the blood as the mononatrium urate. In gout the *b*-salt plays a prominent role. The author has sought a method to prevent the formation of the less soluble *b*-salt from the *a*-salt and also to change the *b*-salt back to the *a*-salt. He finds that radium is capable of inhibiting the formation of the *b*-salt. He also believes that he has been able, by this means, to convert the *b*-salt into the *a*-salt, which is twice as soluble as the former in the blood.

The Bacteriology of the Blood in Convalescence from Typhoid Fever.—Coleman and Buxton (*Jour. Med. Research,* 1909, xxi, 83) examined 46 cases of typhoid fever in regard to the bacillemia. They found in the majority of instances that the bacilli disappear from the blood as the temperature falls; that the defervescence and bacillemia are conterminous. They believe that the atrium of infection is the lymphatic structure of the intestinal wall. Thence the bacilli invade the general lymphatic system and spleen, where they grow chiefly and where relatively few are destroyed. A limited number may filter through into the blood in the incubation period of the disease. When the development of the bacilli has reached a certain grade, they overflow in quantity into the blood, where they are destroyed and their endotoxins set free. The symptoms represent the reaction to this. As the disease advances, the growth of the bacilli in the organs may be gradually controlled by the immunity processes, fewer bacilli are discharged into the blood, and the symptoms lessen in severity. At or about the time of defervescence the bacilli practically disappear from the blood. A few may filter through but the symptoms become less and convalescence proceeds. The post-typhoid elevations of temperature may be due to discharges of limited numbers into the blood, the growth in organs not being fully checked. The enlargement of the spleen and subsequent associated relapse suggest that the growth of bacilli there has been brought under only temporary control, and later, rapid multiplication occurs bacilli are discharged into the blood and destroyed, and the symptoms of a relapse become manifest.

A Comparison of the Guaiac and Benzidene Tests for Invisible Hemorrhage in Diseases of the Digestive Organs.—White (*Boston Med. and Surg. Jour.,* 1909, clx, 733) states that the clinical value of the guaiac test for occult blood is established beyond question. The method is simple, has given uniformly negative results with healthy persons on a meat-free diet, and has proved capable of detecting minute invisible hemorrhages in the gastro-intestinal tract. It is everywhere in use as an aid to the diagnosis of cancer and ulcer from the many diseases which may closely resemble them. The clinical value of the benzidene test is less well known. White thinks it important to recognize and use both methods. A preliminary step is necessary in both tests to exclude sources of error from food ferments. Acetic acid-ether extraction is best for the guaiac test, and boiling in the benzidene test. Metallic salts, potassium iodide, and charcoal must not be given when the stomach contents are to be submitted to the latter method. Meat, fish, and their juices must be excluded from the diet and no hemoglobin derivatives administered for two to four days previously. The stomach tube often introduces error by injuring the mucous membrane. Therefore, feces are more reliable. The Weber method is the best guaiac test for routine clinical work, namely, the addition of 10 drops of fresh tincture of guaiac and 30 drops of well-ozonized turpentine to an acetic acid-ether extract of 10 c.c. of feces. Schroeder's statement that each strength of blood solution has an optimum strength of guaiac tincture to obtain the deepest and clearest blue color is correct. It is well, therefore, to use 1, 2, and 10 drops of the usual guaiac tincture. Schlesinger and Holst's modification of the benzidin test is from two to five

times as delicate as the guaiac test. It consists in adding 2 to 3 drops of the boiled feces mixture (a pea-sized piece in 4 c.c. of water) to a mixture of 10 drops of fresh benzidin solution (a knife tip of benzidin in 2 c.c. of glacial acetic acid) and 3 c.c. of commercial hydrogen peroxide. A clear green or blue appears when positive. A negative benzidin test has greater value than a negative guaiac test, and if both tests are used as a control much time will be saved by using the benzidin method first. It takes only two minutes, and if negative, renders further testing for blood unnecessary.

A Serum Reaction in Mental Diseases.—Much and Holzmann (*Münch. med. Woch.*, 1909, lvi, 1001), in a preliminary report, describe a characteristic serum reaction discovered in the examination of psychiatric cases, which promises much as an aid to diagnosis and prognosis. The principle of the reaction is simple: If human serum is added to cobra venom, hemolysis of washed human corpuscles is not prevented; but if serum be obtained from a patient suffering with dementia præcox or maniac depressive insanity, it is capable of inhibiting hemolysis. The technique used in performing the test is the following: 0.35 c.c. of serum is mixed with 0.25 c.c. of a 1 to 5000 solution of cobra venom (dilution made with physiological salt solution). To the mixture one adds 0.5 c.c. of a 10 per cent. suspension of human corpuscles and then places the tubes in the incubator for two hours and afterward in the ice chest for twenty-two hours. In examining the tubes, they are thoroughly shaken, and if the contents are turbid (lack of complete hemolysis), the result is positive. Controls must, of course, be made with normal serum. In a number of cases examined in this way, the reaction, which Much and Holzmann call the "psychoreaction," has been positive only in dementia præcox or maniac depressive insanity or in a member of a family in which either of these diseases or circular insanity (at times with epilepsy) has existed. Much and Holzmann promise a more detailed report later. They have been unable to obtain the reaction in the cerebrospinal fluid.

Sodium Chloride in Nephritic Œdema.—Blooker (*Deutsch. Arch. f. klin. Med.*, 1909, xcvi, 80) has made a careful study of six cases of nephritis with œdema with reference to sodium chloride intake and output. The value of restricted sodium chloride in the treatment of nephritic œdema is still, he believes, an open question. His observations lead him to the following conclusions: (1) Œdema in nephritis is caused by primary sodium chloride retention only in exceptional cases. (2) A salt-free diet is rarely of value in the treatment of nephritic œdema.

The Influence of Various Carbohydrates on Glycosuria.—Werbitzki (*Ztschr. f. exp. Path. u. Ther.*, 1909, vi, 235) has made a very careful study of the sugar output in diabetes following the eating of various carbohydrate foods. He believes as a result of his observations and a study of the literature that various carbohydrate foods, even though they contain one and the same carbohydrate, may nevertheless lead to greatly varying sugar output. Of the foods rich in starch, oats occupies a conspicuous position, since in many cases of diabetes it leads to no increase in the sugar excreted in the urine. This leads to the supposition that a chemical difference exists between the carbohydrate of oats

and that of most other foods. An oatmeal diet exercises a favorable influence in the majority of cases, not only on the glycosuria, but also on the general condition of the patient, and for this reason it should be more commonly tried in the treatment of diabetes. It should be used to the entire exclusion of other carbohydrates, at any rate until a fair trial has been given it.

The Cultivation of Spirocheta Pallida.—Schereschewsky (*Deut. Med. Woch.*, 1909, xxxv, 835), in a preliminary note, gives the result of his attempts to cultivate Spirocheta pallida. Preivously it has been possible to grow the organisms in a collodion sac placed in the peritoneal cavity of an ape. As a culture medium Schereschewsky has used horse serum which he heated to 60° C. until a jelly-like consistency was attained. The culture was made by placing a fresh fragment of a luetic papilloma or condyloma in a centrifuge glass two-thirds full of the medium and corking it after the material had sunk to the bottom. In three to five days the organisms are found in large numbers in the medium. Schereschewsky believes he has made a successful transfer of his culture. A pure culture has not been obtained.

Alimentary Lipemia.—Leva (*Berl. klin Woch.*, 1909, xlvi, 961) has proposed a simplified technique for the study of alimentary lipemia. In the method of Neisser and Braeuning the patient is given a definite quantity of cream; 15 c.c. of blood are then taken, after a given internal of time, from an arm vein, the serum is allowed to separate, and then the layer of "cream" on the serum is measured after standing four days in the ice chest. Leva examines the serum directly with dark field illumination, and estimates the degree of lipemia by means of the apparent number of fat droplets in the serum. As a test meal he proposes 45 grams of white bread, 20 grams of butter, and a cup of tea without milk. As yet, the number of patients examined is too small to permit one to draw any definite conclusions. He has tested olive oil and cream nutrient enemas in that way and finds that very little of the fat is absorbed. Leva confirms the work of other observers to the effect that hemoconia are in reality fatty particles.

Meat as a Source of Infection in Tuberculosis.—Littlejohn (*Practitioner*, 1909, lxxxii, 843) states that the risk of contracting tuberculosis by eating the meat of tuberculous animals is not so great as generally believed. Imperfect inspection makes the risk greater than it should be, a risk which concerns especially those who buy cheap meat and eat such commonly infected organs as the lungs, udder, and mesenteries. The relative infrequency of tuberculosis in all but cattle and pigs makes them the only food animals at all dangerous to man. The flesh of tuberculous animals is not necessarily infectious, and when so, in many instances, it is due to secondary contamination by means of the knife, butcher's cloth, and dressing. Cooking is sufficient ordinarily to destroy tubercle bacilli on the outside of a roll or joint, but not in the centre. Digestion, salting, or smoking have little or no disinfecting effect. However, the rarity of tuberculous foci in the flesh, even in advanced cases, makes ordinary cooking efficient if the thick rolls and joints are avoided and the meat is well done.

SURGERY.

UNDER THE CHARGE OF

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The Operative Treatment of Ascites Due to Cirrhosis of the Liver.—Soyesima (*Deut. Ztschr. f. Chir.*, 1909, xcviii, 390) reports three cases in which Routte's operation for draining the peritoneal fluid into the long saphenous vein, was performed. In the first case this operation had been preceded by several others: August 31, 1906, the omentum was sutured to the subcutaneous tissue of the abdominal wall; October 9, 1906, the left kidney was decapsulated, and six weeks later the right kidney; December 17, a silver cannula was introduced into the peritoneal cavity in the ileocecal region for permanent drainage. These operations were without important results. December 10, 1907, an incision was made in the upper inner surface of the left thigh over the course of the long saphenous vein, from a point 2 cm. above Poupart's ligament, obliquely downward and inward, about 17 cm. long. From its entrance into the femoral vein, the long saphenous was freed about 8 cm. downward, and divided somewhat obliquely. The diameter of its lumen was about 3 mm. The peripheral stump was ligated and the central stump turned upward and outward over Poupart's ligament. Just above the ligament an incision was made about 1 cm. long in the peritoneum, and the open end of the central stump of the saphenous vein was anastomosed into this opening and fixed by five catgut sutures, No. 0, so that not a drop of the ascitic fluid escaped. Gauze drainage was used and a collodion dressing applied. The drainage was removed on the second day. Healing by first intention followed. The quantity of urine increased somewhat after the operation, but the abdomen again gradually distended. On December 19, 1907, a similar operation was done on the right side. On July 7, 1908, it was noted that the daily quantity of urine was, on an average, 1460 c.c. No more punctures were necessary since the last operation, and the general condition was much improved. The abdomen was still moderately distended, but was not tense. The pulse was strong and regular, 72 per minute. On December 11, 1908, the patient felt well and the abdomen was becoming smaller. In the second case the operation was not successful, and the patient died. Autopsy showed both saphenous veins completely obliterated. In the third case at autopsy the communications between the saphenous vein and peritoneal cavity were about large enough to admit a pin. On the right side it could not be probed, but on the left side it could be probed about 12 cm. The Talma-Drummond operation, the production of adhesions between the

omentum and anterior abdominal wall, with its modifications, is indicated only in the early stages of the disease. The Routte operation, the anastomosis between the peritoneal cavity and saphenous vein, is worthy of trial in very bad cases.

The Surgical Treatment of Gastric Ulcer.—Clairmont (*Mitt. a. d. Grenzgeb. d. Med. u. Chir.* 1909, xx, 330) says that of 259 cases of gastric ulcer operated on in one of von Eiselsberg's clinics during the past ten years, the mortality has been diminished from 24.5 per cent. to 6.6 per cent. By the employment of posterior, retrocolic gastro-enterostomy it was diminished from 16.6 per cent. to 3.5 per cent. The late results after gastro-enterostomy are not as favorable as in Krönlein's statistics, Krönlein reported a complete cure in 61 per cent., and a considerable improvement in 24 per cent. In Clairmont's cases complete healing occurred in 52 per cent. and improvement in 15 per cent. The influence of gastro-enterostomy on gastric ulcers will depend upon the position of the ulcer in the stomach. The nearer the ulcer to the small intestine the more favorable will be the prognosis. Ulcers at the pylorus or in its neighborhood were influenced favorably by gastro-enterostomy in 62 per cent. of the cases, those removed from the pylorus in 47 per cent., and those in the duodenum in 73 per cent. The failures occurred particularly in those cases in which the ulcers were distant from the pylorus, those in which there developed peptic ulcers at the anastomosis, and in those in which the anastomotic opening became secondarily contracted or closed. Since in only 3 per cent. of his cases there were lacking such changes in the stomach wall that the site of the ulcer could not be recognized from the outside, the situation of the ulcer in the stomach can be determined by the surgeon in almost all cases, which fact is of value in determining the choice of operation. Often the location and time of the occurrence of the pain will permit a proper topical diagnosis of the ulcer. Those ulcers having associated with them an inflammatory tumor formation, as well as the penetrating ulcers, give an especially favorable prognosis from the standpoint of operation, provided they are not too far removed from the pylorus.

The Results of Spinal Anesthesia, Especially in Laparotomies.—Zahradnicky (*Archiv f. klin. Chir.*, 1909, lxxxix, 371) reports 1650 cases in which spinal anesthesia was employed, since 1900. He followed closely the indications given by Bier and Dönitz and strongly approves of them. In four cases he failed to enter the spinal canal. This happened particularly in old people in whom the cartilage is usually ossified, in those in whom the lumbar spine is flat, or when there was an abnormal curve of the spine. In two cases the injection needle was broken off in the removal of the needle, and an incision was necessary to find and remove the broken off part. In the beginning α-eucaine was employed (81 cases). β-eucaine was used in only 7 cases. Better results were obtained from the use of tropacocaine, which was employed in 367 cases. Suprarenal extract combined with each of these substances improved and deepened the anesthesia. Still better results were obtained from the use of stovain and novocain. The latter was finally selected as the best anesthetic and was employed in 859 cases.

Suprarenal extract was combined in 667 of these, but in the last 192 cases the novocain was used alone. Gradually the suprarenal extract was found to be undesirable. The novocain was given in doses of 0.17 gram, which gave complete anesthesia in 91.1 per cent. of the cases, and this lasted one and one-half, two, or three hours, and was deep. Incomplete anesthesia was observed in 2.3 per cent. of the cases, and general narcosis was added in 3.9 per cent. Collapse occurred in 0.03 per cent., vomiting during the operation in 10 per cent. (very frequently in laparotomies and herniæ). The pulse was slowed or increased in only 1.7 per cent. of the cases, while in 52.5 per cent. paresis of the sphincter ani and lower extremities occurred, but these were only transitory and without consequence. The temperature only rarely (2.2 per cent.) rose to 38°. Headache also was not frequent and was of slight intensity (13 per cent.) Vomiting after operation occurred in 9.8 per cent., mostly in laparotomies and herniæ. Sleeplessness was observed in 33.5 per cent. of the cases, while retention of urine and hyperalgesia of the wound was observed in only a few cases. The Trendelenburg position aided much in obtaining anesthesia in all affections of the abdominal organs. In 226 laparotomies complete anesthesia was obtained in 212, incomplete in 14, while in 8 cases general anesthesia was necessary. The anesthesia generally reached up to the costal arch and mammary glands, many times to the clavicle. Only in a few cases did it fail to reach the costal arch, and the lowest level to which it extended was the umbilicus. Spinal anesthesia has the advantage over general anesthesia that it produces no bad effects on the heart and lungs, and, according to Zahradnicky's experience, no bad effects on the kidneys, which occurred with the use of stovain.

Late Development of Deformity in Consequence of Fracture.—Zesas (*Archiv gen. d. chir.*, 1909, iii, 471) reports the case of a man, aged thirty-two years, who in November, 1906, fractured the upper fourth of the right leg. The limb was in very good position. Three days after the accident a plaster cast was applied and was left on fifty-two days. On its removal the fracture had consolidated, and the leg was straight, without the least deformity. Crutches were then used and there was no pain. At the end of the fourth month the patient began to work. About twelve months after the accident the patient began to have acute pain at the seat of fracture, principally at night. A little later he observed a slight deformity of the leg, but he continued to work. Zesas saw him for the first time in November, 1908, two years after the accident. Four months before this time the pain had disappeared, but the patient continued to limp, although the deformity had made no progress. At the juncture of the upper and middle thirds of the leg was a well marked curve, concave internally, giving the limb the aspect of a genu varum. There was a voluminous solid callus at the seat of fracture. In walking the foot rested chiefly on its external surface. In connection with the reports of similar cases, two pathogenic theories are offered—deficient ossification of the callus and repeated fractures of the callus. The callus remains cartilaginous in some cases longer than usual, and it is possible that in the cases which develop these deformities, it had not ossified when walking was begun. Such callus may harden later,

making the deformity permanent. Sometimes the callus is strong enough to permit walking, and then yields to violence not sufficient to break a normal bone. In Zesas' case, when the patient began to walk the callus was solid, and there was no movement or pain at the seat of fracture. The patient said that he had sustained no violence or traumatism since the accident. The pain which preceded and accompanied the development of the deformity can only be explained by an inflammatory process in the callus. Probably in certain cases an inflammation of the callus can produce a rarifaction and softening of the bony tissue. While it would be difficult to determine the exact cause of the osteitis, it is not impossible that it was due to slight breaches in the newformed osseous tissue.

Concerning Uranoplasty.—Ranzi (*Archiv f. klin. Chir.*, 1909, lxxxix, 609) reports the results of a statistical study of 61 operations for cleft palate, performed in v. Eiselsberg's clinic, between 1904 and 1908. Of 22 cases operated on by the Lane method, 5 resulted in a complete closure of the cleft, in 7 a small defect was left, in 4 a partial closure resulted, and in 6 the operation was a failure. Two patients died as the result of the operation and three died later. In spite of the good results reported by Lane in 200 cases, this operation has not many supporters among German surgeons. Of 32 cases operated on by the Langenback (Billroth) method, with its modifications, in 13 there was a complete closure of the cleft, in 7 a small defect was left, in 6 a partial closure was obtained, and in 6 the operation was a failure. There was no death from the operation in this series, but one patient died later. A comparison of the results shows that both operations were followed by about the same proportion of failures and partial closures. From the Lngaenbeck operation, however, there were 27.7 per cent. of complete closures, while from the Lane operation there were only 22 per cent. In 31 per cent, there were left small defects. The dangers of operation soon after birth, when Lane advises operation, are very serious. Of operations done in the first days after birth, the mortality was 66 per cent., in the first year 37 per cent., and in the second year 12.5 per cent. When the cases of complete healing of the cleft and those with small defects are collected together, it is found that there were none from the operations done in the first days of life, 37.5 per cent. in the first year of life, 37.5 per cent. in the second year, 68 per cent. from the second to the sixth year, 77 per cent. from the sixth to the fourteenth year, and 75 per cent. above the eleventh year. These figures speak strongly against the early operation. On account of the lessened tension on the flaps in the Lane operation, it is especially adapted for cases with wide clefts. On the other hand in the Langenbeck-Billroth operation, the nourishment of the flaps is better, so that it should be preferred for operations on narrow clefts. The Bunge method of introducing the sutures is to be preferred, because it gives the best anatomical results. Uranoplasty is best done soon after the second year.

A New Method of Operating for Habitual Dislocation of the Shoulder by Muscle Transplantation.—Clairmont and Ehrlicher (*Archiv f. klin. Chir.*, 1909, lxxxix, 798) accept the causes and the groups of

cases given by Perthes, which the latter based upon the previously published autopsy and operative findings in habitual dislocation of the shoulder. Perthes divided the cases into three groups: (1) Consisting of the cases in which there is a tearing away of the muscles from the greater tuberosity; (2) cases in which there is a tearing or breaking away of the capsule from the margin of the glenoid cavity; and (3) cases in which the capsule is relaxed. After critically examining the various methods of operation, he can see that the only cause of success of the operations is in the contraction of the capsule, which was done in all of them. Still we should seek simpler methods. The incision for exposing the capsule, especially when the deltoid is temporarily detached, makes relatively too severe an operation. The writers have obtained good results in two cases by transplanting a muscle flap taken from the posterior portion of the deltoid and attaching it to the head or surgical neck of the humerus, in order that it may prevent the head from passing forward into the dislocated position, from the contraction of the muscles on the inner side. In view of the numerous pathological causes, based upon anatomical grounds, for the occurrence of these dislocations, there is need of a uniform method of operation, applicable to the greatest number of cases, and less severe than those now in use. Since the habitual dislocation is actually due to a lack of coördination of the normal muscular contractions, one is justified in ignoring the changes in the articulating surfaces and the capsule, and in preventing the tendency to luxation by the effect of an antagonistic muscle. This can be accomplished by the formation of a flap from the posterior portion of the deltoid muscle, with its base at the spine of the scapula. The free end is passed through an opening made for it around the posterior part of the neck of the humerus to the anterior medial part of the deltoid, where it becomes united, with preservation of its contractility. This method can be employed in the greatest number of cases. It avoids opening the joint. The free return of movements is rapid, without the necessity of after treatment.

An Anatomical and Mechanical Study of the Shoulder-joint.—Goldthwait (*Amer. Jour. Orthop. Surg.*, 1909, vi, 579) made an anatomical and mechanical study of the shoulder upon the cadaver, and combined this with a study of clinical cases. Most of the cases could be arranged in three groups. The first and largest group was characterized by pain and limitation of movement in the shoulder, chiefly of rotation. These cases have been most often designated as rheumatism, periarthritis, or neuritis. The second group consisted of those cases with instability of the joint and recurring dislocations. In the third group are those cases with pain in the arm, frequently referred to definite points, sometimes to the distribution of a single nerve, most often the ulnar. The anatomical features seem to be the same in all, as well as many of the principles of treatment. Goldthwait calls attention to the variations in form, length, and position of the coracoid and acromion processes, and says that normally the tip of the coracoid rests against or is opposite the lesser tuberosity. The amount of contact between these bones depends upon the shape of the bones and the position of the shoulder with reference to the rest of the body. In the position of drooped shoulder

the point of contact of the coracoid with the humerus is made necessarily lower and must be more constant. There is from 50 to 60 degrees more rotation of the humerus in this than in the erect position of the shoulder. In a similar manner, an acromion process, longer than normal, is brought in contact with the greater tuberosity, an abduction of the arm, earlier and more forcibly than usual occurs, and then the head of the humerus tends to be forced out of the glenoid cavity. To prevent undue irritation from the impingement of the bones against each other, bursæ exist, the subdeltoid or subacromial, on the one hand, and the subcoracoid or coracobrachialis on the other. The painful shoulders result usually from inflammation of one or other of these bursæ, which is aggravated or may be caused by the position of round or drooped shoulder, since in this position the bony surfaces are brought more forcibly together. After the acute symptoms have subsided the development of adhesions gives rise to limitation of motion in the shoulder. Goldthwait accounts for the third group of cases, the brachial neuralgias, by the position of the head of the humerus in drooped shoulders. In this position the ulnar nerve lies directly under the head of the bone, this being the most posterior of the branches of the plexus, and it is pressed upon as it crosses the second rib. If the subcutaneous fat tissue is present in considerable amount, the pressure exerted is more apt to be upon all the axillary structures, the pressure upon the individual nerve being less easily possible, and the more general pain is the result. The treatment of all the different conditions, although widely different clinically, is practically the same except for minor details. The first requisite consists in changing the forward position of round or drooping shoulder, so that it is held erect. There is then no undue pressure on any of the structures; the stability of the shoulder-joint is increased, so that displacements are difficult of occurrence; the bursæ are no longer unduly irritated; and the axillary structures cannot be compressed. If adhesions exist between the bursal surfaces, they can be overcome by proper manipulations under an anesthetic.

THERAPEUTICS.

UNDER THE CHARGE OF

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The Treatment of Gastric Ulcer by Immediate Feeding.—Spriggs (*Brit. Med. Jour.*, 1909, i, 825) summarizes the principles of the Lenhartz treatment of gastric ulcer as follows: (1) Complete rest in bed for four weeks; (2) feeding the patient from the beginning of the attack with small quantities of beaten-up eggs and milk, the quantities being increased daily; (3) the application of an ice bag to the epigastrium;

(4) adding to the dietary boiled rice, minced meat, and other semisolid and solid foods after the first week; (5) the administration of bismuth and iron in suitable form. Of these, the complete rest in bed, the bismuth and iron, and the ice bag are commonly advised by physicians; but the plan of allowing food to be put into the ulcerated stomach at the beginning of the attack is contrary to the usual practice. The detailed routine of the Lenhartz treatment was given in an article in this journal (1908, cxxxvi, 18). Spriggs discusses the theory of the action of the Lenhartz treatment and calls attention to some of the disadvantages of the old method of rectal feeding. He mentions, as chief among these disadvantages, the considerable period of starvation or semistarvation and the less successful the treatment is, the longer is the period of starvation. Rectal feeding is tedious, unpleasant, and difficult of application outside of hospitals. Spriggs gives a brief review of the published cases treated by the Lenhartz method. Wagner, in 1904, reported 60 cases, 35 of which had no pain from the beginning of the treatment. In only six of this series did the pain persist beyond the first week of treatment. Haberman, in 1906, reported 135 cases treated by Lenhartz, with only three deaths. Hemorrhage occurred in only 8 per cent., as compared with 20 per cent. in 100 cases treated by the older method. Wirsung reported 42 cases with very favorable results. He found that 27 of these cases showed a less amount of hydrochloric acid after test meals as a result of the treatment. Lenhartz, in 1906, reported 140 cases, all with recent hematemesis or melena, with a mortality of 2.14 per cent. Lambert, in 1908, reported five severe cases, all of which did well on the treatment. Berger thinks that the treatment may be of diagnostic value, for, if patients do not do well, the disease is probably not ulcer. He cites 6 cases in which the failure of the diet led to the diagnosis of a gastric cancer, confirmed in four of them by operation and in two of them by autopsy. In another case, hemorrhage was due to cirrhosis of the liver. He says, further, that in cases of epigastric pain without bleeding, which do not yield to the Lenhartz treatment, nervous disease may be suspected. Schnütgen, in 1907, reported that Lenhartz had then treated 201 cases. with a mortality of 3 per cent., compared with 195 cases treated by Leube with a mortality of 4 per cent. Leube has collected a total of 556 cases treated by nutrient enemas, with a mortailty of 2.2 per cent. Many of these cases, however, were not severe enough to be hospital cases. Ewald uses nutrient enemas for three days after hemorrhage, then substantially follows the Lenhartz method. In 1906 he reported 34 cases, with 14 deaths, occurring either during the treatment or after operation. He condemns the Lenhartz treatment as unsatisfactory, but, as Spriggs remarks, the Lenhartz results are better than Ewald's. Spriggs' paper is based upon 33 cases treated by the Lenhartz method and 34 cases treated by the ordinary methods. He arrives at the following conclusions: (1) The Lenhartz treatment is not more dangerous than treatment by nutrient and saline enemas followed by a graduated milk diet. In these particular cases the recurrence of hemorrhage was less frequent, and there were no deaths. (2) The pain suffered by the patient in the course of the treatment is less on the Lenhartz diet. (3) The diet gives far more nourishment than can be introduced into the body by nutrient enemas, and is, therefore, more desirable in patients, who have frequently been for a long time in a state of semistarvation, or have suffered a loss of blood, or both. In

addition, the Lenhartz treatment enables the patient to take ordinary diet without pain in a much shorter time than the treatment by nutrient enemas. (4) In patients treated by this method rectal injections may be entirely avoided. This is an advantage in a hospital and a still greater advantage in treating patients at their homes, where rectal injections are not only regarded as extremely unpleasant, but are seldom efficiently administered.

Bismuth Poisoning and a Non-toxic Substitute for Bismuth in Roentgen-ray Work.—Lewin (*Münch med. Woch.*, 1909, xiii, 643) maintains that the symptoms of bismuth intoxication are due to the bismuth itself, and not to the production of nitrites. Consequently, he believes that it is useless to substitute other forms of bismuth for the subnitrate in Röntgen-ray work. In this opinion he disagrees entirely with the majority of other observers upon the same subject. Lewin has experimented in order to discover a substitute for bismuth and recommends for this purpose the magnetic oxide of iron. This is insoluble in the stomach and intestines, and admirably arrests the rays. He states that Röntgen pictures taken with the magnetic oxide of iron are far superior to those he has ever obtained with bismuth.

Vaccines in Typhoid.—Walters and Eaton (*Boston Med. and Surg. Jour.*, 1909, xvi, 509) report a further series of cases treated by the hypodermic administrations of vaccines made from dead bacilli. They give in detail the histories of a family consisting of a mother and three children, all attacked by the disease at the same time. Two of the children, who received vaccines improved rapidly. A rapid fall in temperature was noted soon after the treatment was begun. The youngest child had been the least sick, and was not inoculated. The child became steadily worse, and on the fourteenth day of the disease the temperature was 104.8°. The vaccine was then administered and the temperature fell immediately with a marked improvement in the other clinical symptoms. They note that of 77 cases of typhoid fever treated during 1908, 31 received the vaccine treatment. The remaining cases received the ordinary routine treatment. The mortality of the cases treated with vaccines was 3.2 per cent. as compared with a mortality of 11.1 per cent. in the cases not thus treated. In all, Walters and Eaton have now observed about forty cases treated by this method, and they believe that the early administrations of vaccines shortens the duration of the disease and also lessens the severity of the symptoms.

Physiological and Therapeutic Properties of Serum of Milk (Lactoserum).—Blondel (*Lancet*, 1909, 1,1038) has performed some very interesting experiments with the serum of cow's milk. He obtains this serum in the following manner: Fresh milk is warmed to 30° C. and rapidly coagulated by a few drops of hydrochloric acid. The curd is removed by filtration through fine muslin. After exact neutralization with sodium carbonate, the mixture is filtered through papier chardin. Finally, the filtrate is again filtered through a d'Arsonval bougie under pressure of carbonic acid gas. Chemically, lactoserum is a solution in which sodium chloride, milk sugar, and phosphates of the alkalies and

alkaline earths form the principal constituents. Lactoserum, in addition, contains certain ferments, some of which are found in many extracts of animal organs and animal sera. Blondel injected lactoserum subcutaneously and found that the specific action of these ferments was pronounced. His results seemed to show that lactoserum had a marked effect in lowering the arterial blood pressure, or what might be more correctly termed a regularization of blood pressure. He gives an injection of 10 c.c. and repeats it every twenty-four hours until the blood pressure falls to normal. After this the injections are continued for four or five days longer, and then a weekly injection for a month. If the pressure rises the injections may be more frequent. When the pressure remains normal, a monthly injection is given for three months. The injection is made into the muscles of the thigh or the gluteal region. Sometimes a mild erythema is seen. Usually the injection is not painful. No abscess has occurred in more than 1000 injections. Blondel made use of these injections for the treatment of arterial hypertension in arteriosclerosis, with striking results. A fall in the blood pressure was regularly noted with an alleviation of the general symptoms. Several of these patients had albuminuria, and apparently the lactoserum increased the albuminuria during the first days of the treatment. Blondel believes that if the albuminuria depends upon the hypertension alone, it will disappear quickly and permanently when the treatment is maintained. If, on the other hand, the albuminuria is the result of a true nephritis, it will return to its original degree after the initial increase. He believes that this fact may serve to determine whether an albuminuria is due to an organic kidney lesion or simply to the hypertension. Blondel also used lactoserum in the treatment of certain infections, with variable results. The serum had no apparent effect upon cases of puerperal infection, postoperative peritonitis, and typhoid fever. In pneumonia he had rather remarkable and constant results, though the number of cases (twelve) so treated does not allow any definite conclusions. His cases of pneumonia seemed to defervesce usually after the second injection, though at times defervescence occurred after the first injection.

The Therapy of Gout.—Umber (*Therap. d. Gegenwart.*, 1909, ii, 73) arrives at his conclusions regarding the treatment of gout after considerable investigation of the metabolism of gouty and of normal individuals. His researches have been carefully conducted from a chemical point of view and include studies on the metabolism of 137 cases of gout. The elimination of uric acid, according to Umber, follows typical curves. The amount of endogenous uric acid grows less and less until the onset of an acute attack of gout, when there is a rise in the curve. This rise attains its maximum height two or three days after the onset of the acute attack. Then there is a gradual falling off in the uric acid elimination to amounts below the normal during the intervals between the attacks. These curves of uric acid excretion are typical of gout, as Umber shows by comparison with other diseases in which there is a urie-acidemia. In demonstrating the failure of the tissues to rid themselves of the small amount of endogenous urie acid, even after prolonged use of purin free foods, Umber's results confirm the researches of Brugsch and Schittenhelm. In view of this fact, it is to be expected that the excretion of the

exogenous uric acid following the ingestion of purin rich foods would be much less prompt than in normal subjects, and this is found to be the case. Umber concludes from these studies that there is no medicinal treatment of gout other than measures directed to relieve the symptoms at the time of the acute attacks. In the intervals therapeutic effort must be directed entirely to a consideration of the diet. The gouty person should never take more than 200 grams of meat a day, and when an attack is impending no meat should be allowed. It is still further advisable to arrange several purin fast days in each week, and thus give the body the opportunity to eliminate its excess of uric acid. Umber is emphatic that this regimen must be kept up for months and years in order to avoid the later severe stages of this disorder. Drinking large amounts of water aids in the excretion of uric acid, but, contrary to the long accepted opinions. Umber declares that alkaline waters have the opposite effect. None of the alkaline or mineral waters has any specific effect upon gout, and this is especially true of the lithia waters. The good results obtained from spa treatments are due to factors other than the waters. Umber is of the opinion that none of the various remedies directed against the uric-acidemia has any effect, since it is not the amount of uric acid in the blood that causes the trouble, but the deposition of sodium urate in the cartilages. The blood is always able to dissolve more uric acid than it contains. No remedy has been found to effect the sodium urate deposit. It has been noted that the excretion of uric acid diminishes after exercise, even when the diet is purin free. Umber thinks that this signifies that an increased oxidation brings about a lysis of the purins in the body. He believes, therefore, that carefully regulated exercise is of value in the treatment of gouty patients.

Falkenstein (*Berl. klin. Woch.*, 1908, xxxvi, 1649) advises the use of hydrochloric acid in the treatment of gout on the following principles: He says that by the use of hydrochloric acid the proteins are more readily broken down, and consequently less nitrogen remains in the body, and that with an increased output of nitrogen, there is a diminished formation of uric acid. Furthermore, the body alkalies are diminished by the hydrochloric acid treatment, and therefore there is a lack of the necessary alkalies to combine with the uric acid and form the insoluble sodium urate. For similar reasons he prefers the use of sodoglodine (a combination of iodine with vegetable proteid) in the treatment of gout. Sodoglodine takes up sodium and potassium from their combinations and is excreted in the form of sodium or potassium iodide. By so doing the sodoglodine withdraws alkalies from the body. He also believes that sodoglodine increases the excretion of nitrogen and diminishes the formation of uric acid. He gives it in tablet form after meals, over long periods of time, gradually lessening the dose.

The Influence of Hydrochloric Acid upon Deposits of Urates.—Staal (*Zeitschrift f. Phys. Chem.*, 1908, ii, 97) speaks of the experiments of von Loghen, and adds observations of his own of contradictory nature. Von Loghen found that when uric acid is injected underneath the skin it becomes soluble in the serum and is excreted. However, it is possible to cause a precipitation of the insoluble urates, though this does not occur in normal tissues. Von Loghen produced this deposition

of urates by feeding his animals alkalies. If the urates are precipitated, von Loghen believes that they are absorbed by the action of phagocytes. Furthermore, he noticed that the urates in the protoplasm of frog leukocytes are made soluble by the action of acids. Von Loghen then fed hydrochloric acid to rabbits, and found that he could control the precipitation of the insoluble sodium urate. Staal says that the solubility of the urates depends upon the sodium ion concentration of the dissolving fluid, and if we accept the solvent action of hydrochloric acid we must assume that the sodium ion content of the body fluids is reduced by its use. The theory that hydrochloric acid withdraws alkalies from the body is a very plausible one, but lacks proof. Staal gave hydrochloric acid to rabbits, and by very exact methods determined the sodium and chlorine content, the specific conductivity, and the freezing point of the blood serum and of the serum expressed from the subcutaneous connective tissue. He made controls on rabbits not fed with hydrochloric acid, and found that the osmotic concentration remained the same. The sodium content of the hydrochloric-acid-fed animals was indeed not lessened, but increased above that of the controls. Therefore, Staal says, a different explanation must be advanced for the possible therapeutic benefits derived from the use of hydrochloric acid. However, the conclusion that hydrochloric acid is of no therapeutic use in gout cannot be drawn from these experiments, because of the probable differences between human gout and artificial deposits of urates.

Iron as a Substitute for Bismuth in Röntgen-ray Work.—Talge (*Münch Med. Woch.*, 1909, xv, 758) speaks of Lewin's suggestion to use the magnetic oxide of iron as a substitute for bismuth in Röntgen-ray photography. He calls attention to some practical disadvantages of the magnetic oxide of iron. It is usually not carried in stock by druggists, and when obtained it must be pulverized, which takes considerable time and is expensive. Therefore, Talge advises, for practical reasons, the use of the red oxide of iron, which may be obtained in the finely pulverized form commonly known as polishing rouge. This is much less expensive than bismuth and answers the same purpose as the magnetic oxide of iron. Talge, himself, has taken 50 grams of the red oxide of iron with no untoward effects.

A Report of Eleven Cases of Staphylococcic Infection, Treated with Leukocyte Extract.—Hiss and Zinsser (*Jour. of Medical Research*, 1909, iii, 245) have treated eleven cases of staphylococcic infection by the injection of leukocyte extracts. In all but three of these the processes consisted of furunculosis of chronic nature, and had lasted, in spite of surgical and dietetic treatment and in some cases in spite of the most desirable hygenic conditions, for periods ranging from several months to four or five years. In all but one of the more chronic cases there was apparently complete cure, and that one case was much improved. The local and general improvement following the injections were even more striking in the acute cases. Hiss and Zinsser say that they have no hesitation in concluding that marked improvement in, and often cure of, localized staphylococcic infection may be obtained by careful and systematic treatment with leukocyte extract.

PEDIATRICS.

UNDER THE CHARGE OF
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The Pathology of Noma.—CRANDON, PLACE, AND BROWN (*Boston Med. and Surg. Jour.*, 1909, clx, 473) report 47 cases of stomatitis in the course of measles, 7 of which went to the stage of definite gangrene of the lips and cheeks; 2 of the latter recovered. Their investigation concerning the pathology of the condition leads them to believe noma to be due to a fusiform bacillus, which grows best along the edge of the necrotic tissue adjoining living tissue. In some cases it excites almost no inflammatory reaction; in other cases the reaction is considerable. In the necrotic tissue the fusiform bacilli rapidly die out and the tissue becomes invaded by various other bacteria. Noma is not a contagious disease and patients suffering with it need not be isolated. Any uncared for mouth, particularly in a sick child, and especially after measles, may contain Bacillus fusiformis and Spirocheta gracilis. In such a mouth these organisms may be found without ulceration, or in the lesions which have been described as stomatitis gangrenosa, Vincent's angina, and noma. The lesions, in other words, may be only round the roots of teeth, on the tonsils and pillars, on the inner side of the cheeks, in the nasal fossæ, on the external ear, and about the genitals. Any of these conditions, including the extensive gangrene and sloughing of so-called noma, may be different stages of the same disease, which may be, therefore, considered as not necessarily a specific disease, but as the successful ingress of mouth bacteria into tissue rendered non-resistant by uncleanliness and preceding disease.

Appendical Abscess in a Twenty-seven-months-old Child; with an Analysis of Infantile Appendicitis in the Johns Hopkins Hospital.—CHURCHMAN (*Johns Hopkins Hosp. Bull.*, 1909, xx, 31) reports the case of a boy, aged two and one-quarter years, who was admitted because of painful urination, which had existed, however, on and off for more than a year. A vesical calculus was supposed to exist, partly because of the history and symptoms, and partly because of a shadow seen on *x*-ray examination, but a stone could not be found by the examination under ether. The pain on urination disappeared, however, after the examination. Several weeks later there were abdominal cramps and fever, and abdominal distention; pain and tenderness, especially in the right side, developed, and a diagnosis of appendicitis was made on the fifth day; there were no urinary symptoms or vomiting. The operation revealed an abscess, which was drained and counterdrained through the flank; the temperature did not return to normal until the ninth day. This as well as the high pulse existing (180) were thought to be due to iodoform poisoning, but the removal of the gauze did not improve conditions. Plain gauze he considers better for drainage in young children. Of 1223 cases of

appendicitis noted at the Johns Hopkins Hospital, 9 were in children under 5; 6 of these were boys. In one of the instances 2 paternal uncles had been operated on for the disease. Five of the patients gave no history of previous attacks; in all 5 the appendix was found perforated; one of them had had general peritonitis, 1 spreading peritonitis, 2 had abscesses, and 1 a gangrenous appendix with beginning abscess. The remaining 4 patients had had previous attacks: 1 six months before admission; 1 had had two attacks, another had had an attack two years before the operation, and the fourth was attacked shortly before the operation. In 4 the diagnosis presented difficulties, tuberculous arthritis, general peritonitis, cerebrospinal meningitis, and vesical calculus being the diagnoses made respectively. All urinary symptoms should suggest the possibility of appendicitis, and in infants with apparent hip-joint disease, particularly if the thigh be flexed, appendicitis should be kept in mind. Palpable resistance on the right side by rectal examination is one of the most frequent findings in appendicitis. The tendency to early perforation in children (7 cases) must be remembered; also the frequency of spreading peritonitis. Intestinal obstruction occurred in 4 cases. The outlook, if early operation is performed, is good, unless peritonitis has set in, when the case is almost hopeless. Besides intestinal obstruction, the only complication which arose after the operation was pneumonia in one case.

OBSTETRICS.

UNDER THE CHARGE OF

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Uterine Contractions.—Bell and Hick (*Brit. Med. Jour.*, March 27, 1909) report the following results in their studies upon the physiology of uterine contraction. They found that extracts prepared from the ductless glands increase uterine contraction, and in varying degrees raise blood pressure. The pituitary and adrenal extracts act upon the involuntary muscle in general. They also increase blood pressure. Thyroid extract lowers blood pressure, but increases uterine contractions. If convulsions occur, the blood pressure rises. Ovarian extract from pregnant animals seems to control uterine contractions, especially in their expulsive efforts. The secretion obtained from the uterus acts upon the inactive uterus, as do the pituitary, adrenal, and placental extracts. It does not, however, raise blood pressure. Uterine secretion and placental extract are somewhat similar in their effect. Uterine secretion seems to act as a specific in producing uterine contractions. Calcium salts show a definite influence upon uterine contractions, with a sight but definite rise in blood pressure. A slow, forcible, and rhythmic contraction is obtained—the type seen in labor. Uterine secretion and calcium salts possess the most specific action in causing uterine

contractions of all the substances studied. The normal non-menstruating, non-pregnant uterus does not contract at all. During menstruation the uterus is in a condition of constantly recurring expulsive contractions. During normal pregnancy the uterus contracts and relaxes without expulsive force. This develops actively during labor. At present no satisfactory uterine extract or specific calcium salt is available for therapeutic purposes. The pituitary extract will soon be available in hypodermic form. A dose of 2 c.c. of the infundibular extract should be given hypodermically, and repeated if necessary. It is especially valuable in cases of shock, in which it is desirable not only that the uterus should contract, but that the blood pressure should be raised. This may also be used in place of physostigmine in paralytic distention of the bowel. Calcium salts have a valuable effect in slowing the heart and strengthening the heart beat.

Adrenalin in Cesarean Section.—Bogdanovics (*Zentralbl. f. Gyn.*, No. 12, 1909) reports the case of a primipara, aged thirty-one years, with flat rachitic pelvis. As spontaneous labor failed, the patient was delivered by Cesarean section, and a hypodermic injection of ergotin was administered to secure uterine contractions. A packing of uterine gauze was carried through the uterus into the vagina. The uterine incision was closed with catgut. Although the uterus at first contracted well, it dilated later, with considerable hemorrhage which was not promptly controlled by massage and hot salt solution. Accordingly 1 c.c. of 1 to 10,000 tonogen solution was given in the uterine wall. The uterus promptly contracted, the patient making an uninterrupted recovery.

Extensive Stenosis of the Vagina following Spontaneous Labor.—Samson (*Zentralbl. f. Gyn.*, No. 12, 1909) reports the case of a patient who gave birth to a dead child after a labor lasting twelve hours. The head of the child was visible for eight hours before its expulsion. The labor pains ceased for four hours and then returned. The laceration of the perineum was not closed. A vesicovaginal fistula developed. The patient had for some time a brownish discharge, and made a very tedious recovery. On examination the vagina was found full of scar tissue, with a vesicovaginal fistula beneath the urethral orifice. The scar tissue extended directly to the cervix, and it was difficult to recognize the cervix in the scar tissue. An operation was performed to close the fistula by loosening the tissue first, and using tampons of gauze. Six months after this operation it was possible to close the fistula. The final result of operation was satisfactory.

Internal Hemorrhage in a Pregnant Woman.—Brindau and Jeannin (*l'Obstétrique*, February, 1909) report the case of a multipara brought by ambulance to a hospital in the eighth month of pregnancy. During the night preceding admission she had been taken with severe pain more pronounced on both sides of the abdomen. This pain had increased in severity. The patient gave a history of two abortions, in one of which interference with the uterus had been practised. On admission she was suffering from shock, and had vomited a greenish

material. The abdomen was not distended and showed a uterus pregnant eight months. The uterus was firmer than normal, and it was difficult to palpate the fœtus. There was no vaginal discharge. The cervix was slightly dilated, the fœtal head was in the pelvic brim, and the membranes unruptured. The urine contained no blood. An exact diagnosis could not be made, but it was determined to empty the uterus as soon as possible. The cervix was dilated to 9 cm. by Bossi's dilator, and a dead child, very pale, was easily delivered by Tarnier's forceps. A hand introduced within the womb found upon the right and upper portions of the uterus the placenta completely separated and a clot of blood within the membranes. The hand could not detect perforation of the uterus. An intra-uterine injection was given and lacerations in the vagina were closed. The uterus and vagina were tamponed with sterile gauze. . On the following morning the patient vomited freely, the pulse rose to a high degree of rapidity, abdominal pain developed, but the uterus remained contracted and there was no uterine hemorrhage. Although free stimulation was given, the patient rapidly failed and died. At autopsy the peritoneal sac contained a large quantity of blood, especially abundant in the region beneath the liver. The uterus showed a solution of continuity upon its posterior aspect, circular in shape, and about the size of a silver quarter. The edges of this opening were composed of cicatricial tissue, and the perforation had occurred over the centre of the placental attachment. The perforation had evidently been an old one; the placenta had been inserted over its site and had separated, followed by hemorrhage, the blood escaping through the perforation into the peritoneal cavity, and giving rise to peritonitis. The perforation had possibly occurred during some former intra-uterine manipulation, probably during the treatment or production of abortion.

The Treatment of Placenta Prævia.—Pfannenstiel (*Monats. f. Geb. u. Gyn.*, 1909, xxix) draws attention to the fact that while it is desirable to treat placenta prævia surgically, it may be impossible to transport the patients to a hospital. Students and practitioners must be taught those methods which they can carry out under ordinary circumstances. The use of the elastic bag and artificial rupture of the membranes are especially valuable. The bag must be introduced with the aid of a speculum, the cervix being dilated sufficiently. A forceps with a suitable pelvic curve is necessary. The fœtal membranes must be ruptured through the introduction of the bag. If the placenta covers the os completely the thinnest portion of the placenta must be selected for puncture. The bag should be distended with 500 c.c. of sterile salt solution, and the weight of one kilo should be attached to the bag. This method gives admirable results.

Thies (*ibid.*) draws attention to the value of premature rupture of the membranes in placenta prævia, vaginal Cesarean section, and the use of the elastic bag within the fœtal sac. The cases must be selected in accordance with the conditions present in the individual patient.

Vaginal Ovariotomy during Labor.—Röhl (*Monats. f. Geb. u. Gyn.*, 1909, xxix) reports the case of a primiparous patient who had noticed marked increase in the abdomen during the latter months of gestation.

She had indefinite abdominal pain for eight days, and the amniotic liquid had escaped twenty hours before admission. On examination Douglas's cul-de-sac was filled with a slightly elastic tumor. The cervix was very difficult to find, and just above it the fœtal head could be felt through the lower uterine segment. A vaginal operation was performed and a cyst removed containing four separate compartments. It was necessary to empty this to deliver the tumor. After removal of the tumor the child was delivered by the high application of forceps without especial difficulty. The placenta was manually delivered. The vagina was then opened in Douglas's cul-de-sac and the tumor found limited by adhesions, which were separated with considerable difficulty. The tumor was finally delivered through the vagina and its pedicle ligated. The patient made a good recovery.

Delivery with the Formation of a Uterine and Abdominal Fistula.—Dobbert (*Zentralbl. f. Gynak.*, No. 11, 1909) reports the case of a primipara, aged nineteen years, who was taken, after examination by a midwife, with a chill. The pelvis was contracted so that spontaneous birth was impossible. As the chill seemed to indicate infection, the patient was delivered by extraperitoneal section, followed by the formation of a uterine and abdominal fistula. The patient made an excellent recovery, and involution proceeded in a satisfactory manner.

The Repair of Recent Lacerations of the Perineum.—Sigwart (*Zentralbl. f. Gynäk.*, No. 10, 1909) draws attention to the method of closing recent lacerations of the perineum by the use of clamps. In 125 cases so treated all but 2 closed by primary intention. The clamps take the place only of those external stitches which were formerly inserted with silk or silkworm gut. In extensive lacerations the mucous membrane of the vagina must be closed with catgut.

GYNECOLOGY.

UNDER THE CHARGE OF

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The Use of Bacterial Vaccines in the Treatment of Septic Infections.—Oastler (*Amer. Jour. Obst.*, 1909, lix, 594) reports a case of chronic sepsis following a miscarriage at three months, steadily getting worse with little or no hope of recovery, improving steadily after commencement of the vaccine treatment. An abdominal section had been made and the wound persistently refused to heal, discharging very freely, but responding after vaccine infections. Several cases are reported, and Oastler believes that: (1) Apparently favorable clinical results have been obtained with the use of vaccines of streptococcus, staphylococcus, Bacillus coli, Bacillus mucosus, and gonococcus. (2) All but gonococcic vaccine should be autogenous. (3) Vaccines are not "cure-alls"

but seem to aid materially in combating the septic process, the effect being gradual and progressive. (4) Vaccines are required especially when the blood shows poor resistance, that is, low leukocyte count and high polymorphonuclear. (5) In violent cases of acute sepsis, no resistance can be created and no effect obtained. (6) The negative phase is rarely obtained. (7) The positive phase is more often obtained. Blood cultures are generally negative. (9) Wound discharge increases soon after injections. (10) Good results have been obtained with all the organisms tried, but the least satisfactory with Streptococcus longus. (11) The pulse may remain rapid sometimes after the temperature falls. (12) As yet there have appeared no ill effects from injections. (13) The dose is problematical and purely experimental. The site of the injection is the same, as is also the frequency. Too large a dose does harm, and small doses give better results than large doses. (14) If the negative phase appears, one should delay the injection and reduce the dosage. (15) The opsonic index is uncertain. The leukocyte count with polymorphonuclear count gives the best indication of the resistance of the patient.

Endometritis.—Mundell (*Amer. Jour. Obst.*, 1909, lix, 608) believes: (1) The classification of endometritis should be acute and chronic. To further subdivide these leads to much confusion and does not materially aid the clinician. (2) Uncomplicated cases are not very common and an uncomplicated chronic case is a very rare condition. (3) Normally, the endometrium is free from bacteria. (4) Treatment of chronic endometritis, to be successful, should always to be carried on in conjunction with the treatment of the associated conditions.

A Case of Ovarian Transplantation.—Casalis (*Jour. Obst. and Gyn., Brit. Emp.*, 1909, xv, 325) removed the appendages of a woman, aged twenty-six years. Immediately thereafter he sutured a part of one of the ovaries, of the size of a small walnut, to the denuded surface of the uterus, using a number one, circular catgut suture. Two and a half months later menstruation occurred. It was painless, lasted two days, was of a fair quantity, and continued regularly every twenty-four to twenty-six days. At the end of three years the patient began to have premenstrual attacks, violent headache followed by intense abdominal pain, three or four severe rigors, accompanied by blueness of the face and extremities, became almost pulseless, vomited incessantly, and had one or two very malodorous stools. Four to five hours later a reaction sets in, noted by heavy perspiration, flushed face, bounding pulse, and the appearance of menses. The flow at the end of four years is very scant and irregular. Casalis believes an early surgical menopause is occurring.

Intestinal Injury in Gynecolcgical Operations.—A. Barth (*Monatssch. f. Geburtsh. u. Gynäk.*, 1909, xxix, 153) states that of fourteen cases of intestinal injury during gynecological operations reported from Chrobak's clinic, in 1903, eleven died, 78.5 per cent. Barth, on the other hand, observed nine such cases with no deaths, although some of the cases were as difficult and as complicated as any reported. He arrives at the conclusion that with expert treatment the patient may not

only be safely tided over the primary dangers of abdominal infection, but intestinal function may also be restored. Shock, which was formerly so greatly feared as it was usually the direct cause of death, at the present time is an unimportant factor in the light of the modern methods of anesthesia and intravenous infusions of saline solutions. The prognosis in accidental injury of the intestines during gynecological operations may therefore be considered very favorable.

Ovarian Fibroma with a Twisted Pedicle.—Meriel (*Ann. de gyn. et d'obst.*, 1909, xxxvi, 117) had a patient, aged fifty-six years, who consulted him for indigestion. She had no pelvic symptoms. An examination of the abdomen revealed the presence of the tumor and considerable ascitic fluid. The abdomen was opened and the ovarian fibroid, weighing two kilograms, and considerable ascitic fluid were removed. The pedicle was twisted one complete revolution and the circulation was not materially modified. The uterus and opposite ovary seemed to be normal. The absence of symptoms other than indigestion was considered unique

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Submucous Perineorrhaphy.—W. Wayne Babcock (*Jour. Amer. Med. Assoc.*, 1909, lii, 1568) describes a method of perineorrhaphy for secondary operation devised by him in 1902 and named submucous perineorrhaphy. The term infers a procedure different from that illustrated and described in the text of the paper. Babcock refers to the illogical features of various procedures for restoration of the lacerated perineum, and mentions the special advantages of his operation as follows: (1) No tissue is removed or extensive denudation made. (2) Buried, absorbable, layer sutures are used exclusively, none of which penetrates the skin or mucous membrane. (3) The operation is done from the outside of the vagina, rendering the introduction of sutures easier and the exposure of tissues better than with those operations done from within the vagina. (4) Each structure is sutured with precision under the guidance of the eye; there is no blind groping with the needle for tissues not seen and perhaps not felt. (5) Each of the layers of the perineal floor—vaginal wall, submucosa, muscular supports, fascial planes, and skin—is united seriatim in layers after the plan of the better types of herniotomy. (6) The vagina is not separated from the rectum and therefore there is no danger of wounding the bowel. For the operation Babcock claims several other advantages, among which are restoration and increase of length of the vagina instead of shortening the posterior wall of the vagina, as occurs with many of the older operations, and affording greater support to the anterior vaginal wall. The description and illustrations do not refer to the worst form of lacerations of the perineum, that is, complete tears. It is in the severer forms of lacerations, those involving the rectal mucosa, that the greatest danger arises from infection.

The Treatment of Uterine Retrodisplacements.—Baldy (*Surg., Gyn., and Obst.*, 1909, viii, 421) reports his advocacy of the method bearing his name of shortening the round ligaments in the treatment of retrodisplacements of the uterus. His present recommendation is based upon an experience of nearly two hundred operations in his clinic.

This number is apparently much lower than the real number, as others have employed it in a larger number of cases. After the operation, according to Baldy, the uterus is held forward by three forces: (1) The pressure of the intra-abdominal force on its posterior surface; (2) the resistance of the encircling band of round ligaments, and (3) the pull forward and downward of the round ligaments at their attachment at the uterine cornua. The whole structure is steadied by those portions of the round ligament running to the normal abdominal attachments. As end-results Baldy has knowledge of 20 to 30 pregnancies following the operation, and in no case has there been the slightest irregularity, although a number of the women had had trouble in former pregnancies. In no case subsequently examined could the fact of a previous operation be discovered. For this operation Baldy claims it is the only one extant that accomplishes what was contemplated by Nature—"A uterus with the fundus forward of the direct line of the axis of the pelvis, freely movable in all directions on the cervix as a movable pivot, the fundus raised up to and above the iliopectineal lines, and the ovaries swung correspondingly high and free from all attachments on the posterior side of the broad ligaments."

OPHTHALMOLOGY.

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Pathological Anatomy and Pathogenesis of Cyanosis of the Retina.—Baquis (*Graefe's Archiv*, 1909, lxviii, 177) observes that individuals affected with congenital cyanosis sometimes present ocular complications, notably cyanosis of the retina. This complication frequently passes unperceived, since it does not always occasion any diminution of the vision. In intense cyanosis, the excessive amount of carbonic acid in the blood excites the vasoconstrictor centre in the bulb, causing considerable venous stasis in the retina. In severe cases the iris also becomes congested; it is, in fact, a matter of cyanosis of the eye rather cyanosis of the retina alone. The iridocorneal angle becomes the seat of a slow inflammatory process, which finally terminates in its obliteration with consequent glaucomatous hypertension.

Remote Results of Extraction of the Crystalline Lens in High Myopia.—From 1893 to 1906 Sattler practised extraction of the transparent crystalline lens 338 times upon 242 patients. Hoepner (*Clin. ophtal.*, May 10, 1909, 258) analyzes this material. The conclusion to be derived from the analysis is that removal of the transparent crystalline lens does

not guarantee against progress of the myopia, that it does not diminish the proportion of detachments of the retina, but rather, perhaps, increases the percentage somewhat, and that it does not tend to limit chorioretinitis nor disease of the vitreous. Hoepner recommends the operation, however, for the reason that the subjects operated upon—those who have had the good fortune to have escaped without complications—are more comfortable and have better use of their vision than before. Commenting upon this, Dor justly asks whether such a result is not rather inadequate. It was precisely to avoid these very complications of myopia that most was hoped for from the operation, rather than the mere satisfaction of diminishing the strength of the correcting glass.

Miners' Nystagmus.—Romiee (*Annal. d'oculist*, June, 1909, 451) has made a study of the manner of working of miners in the mine, during the course of which he examined 600 individuals affected with nystagmus. According to certain authors, miners' nystagmus is consecutive to a myopathy of the elevator muscles of the eye. This myopathy is held to be due to the excessive demands made upon the organ of vision, favored by the anemia with which the subjects are frequently affected, the insufficiency of illumination being regarded as of quite secondary importance. Vertical nystagmus is supposed to depend upon the fact that the miner is constantly obliged to direct his gaze upward. Romiée has found that most miners when they work do not look upward at all, but either directly forward or downward; while among foresters, who have the gaze constantly directed upward, nystagmus is infrequent. Romiée concludes that the principal ccause of this affection is insufficiency of illumination. Nystagmus, in fact, only occurs in workers in coal mines where the illumination is not sufficiently intense, and not in those employed in mining other substances. It would hence seem preferable to speak of "coal" miners' nystagmus. Nystagmus was less frequent in mines where unprotected lamps were used; the safety lamps are soon covered by a layer of dust which greatly reduces the intensity of the light, even as much as 70 per cent. The affection was found to have diminished 50 per cent. in mines where electric lamps were employed. The bearing of this upon prophylaxis is obvious: amelioration of illumination in mines should be secured.

Physiological Muscæ Volitantes.—Fortin (*Rec. d'ophtal.*, February, 1909, 49) has made an entoptic study of these objects; he finds that the vitreous contains normally four kinds of elements: small granular spheres, pearl-like bodies in strings, elongated filaments, and glomerules, or muscæ volitantes par excellence. All these varieties are visible, particularly by individuals having manifest ametropia and long lashes (the latter when incurved forming a series of apertures or stenopaic slits). Whatever be the histological signification of these formations and their physiological role, they are not pathological, but are found in the healthiest eyes. Pathological muscæ undoubtedly exist, but they are usually immobile with reference to the point of fixation; they are due to opacities of the cornea or lens, and in the latter case they may be the earliest symptoms of beginning cataract. Guerin records in his treatise upon diseases of the eyes, published in 1769, that Bartolini, replying to the physician, J. L. Hahnemann, who complained of seeing spider webs

dancing before his eyes, wrote: "The spider webs of which you complain need not alarm you in the least. I have been having the same experience for more than thirty years, but I was reassured upon learning that they were the occasion of more anxiety than real trouble." What was true in 1769 is still true.

Visual Disturbances from Atoxyl.—Coppez (*Clin. ophtal.*, June 10, 1909, 273) emphasizes the untoward effects of atoxyl in internal or external use. Atoxyl even in minimum doses may cause complete and incurable blindness; practitioners who have recourse to this dangerous agent should be on their guard. Ophthalmologists ought to banish atoxyl, and should take pains to inform their colleagues, who practise dermatology or medicine in the tropics, of the dangers to the sight from this preparation.

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF
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The Fight Against Tuberculosis.—The International Conference on Tuberculosis was held this year at Stockholm, the session lasting three days, July 8 to 10. The July issue of *Tuberculosis* contains the papers, and from these the following abstracts are taken:

Widstrand tells of the work of the National Antituberculosis Association in Sweden. This body, organized in 1904, now has a membership of 22,457, equivalent to 4.16 per cent. of the total population. The revenues of the society come from three sources: (1) Members, fees, which are two crowns per year for the well-to-do and one crown for the poorer; (2) donations; and (3) the sale of charity stamps. The total for four years for the sale of stamps amounts to 263,373 crowns and is as much as the fees and donations together. The work of the league has been largely along the line of giving instructions concerning the disease and its prevention to the public. Nearly 1000 popular lectures have been given and eight pamphlets have been distributed, in editions of from 20,000 to 250,000. All schools have been supplied with manuals of instruction. In the second place, the league has devoted its energies to the construction and multiplication of sanatoriums. A Government Commission was appointed to ascertain the number of open cases of pulmonary tuberculosis in the country. This was found to be about 30,000, and it was learned that about one-third of these were in immediate need of institutional help. In 1908 the Parliament made provision for the expenditure during the following ten years of the sum of 11,042,250 crowns in the construction and maintenance of sanatoriums. The law now provides for compulsory notification of the disease and the disinfection of infected houses. Wet nurses and those who handle milk must be shown to be free from the disease, and both the teachers and pupils in the schools are inspected. Dispensaries are kept up in the larger cities and model workmen's dwellings have been

built and are rented at low rates, provided their occupants live according to certain hygienic rules. Four villages with a high death rate from tuberculosis and in unsanitary condition have been taken in charge, supplied with physicians and nurses, and special inducements made to the inhabitants to improve their manner of living. Since 1906 the league has attempted to place the healthy children of tuberculous parents in the country under the care of healthy foster parents, following the recommendation in this particular of Grancher, of France, but this has not, so far, worked well on account of the unwillingness of the parents to part with their children.

Raw, of Liverpool, holds that lupus is caused almost exclusively by the bovine bacillus, and that it is conveyed to man in milk and in other bovine products or it is spread from person to person. He treats lupus with tuberculin made from human bacilli and pulmonary tuberculosis with that made from bovine bacilli. He states that he has treated more than 100 cases of lupus with bovine tuberculin with marked benefit, some being apparently quite cured. He has autopsied 33 persons with lupus, and in only three of these did he find the lungs at all affected. He has never seen people with pulmonary tuberculosis develop lupus, but in three cases he has seen lupus of the neck extend directly to the lung apex.

Buhre, of Stockholm, tells more in detail of the attempts made in Sweden to follow out the recommendation of Grancher in removing the healthy children of tuberculosis parents. Fifty thousand kroner has been set aside for this purpose and 197 children have been cared for in this way. It is, of course, too early to tell what the results of this will be.

Pannwitz, of Germany, discusses the construction and management of a sanatorium for tuberculous children. He holds that sanatoriums represent for curable cases the quickest and surest means of effecting a cure. Forest convalescent stations cannot take the place of sanatoriums, because they have not the required facilities. The children's sanatorium should have sick pavilions, one for each sex, reception rooms, and dormitories. Play grounds free from dust must be provided, and food should be abundant, savory, easily digestible, and nutritious. In practice it has been found desirable to keep a separate colony for those children suspected of being tuberculous. The children should be prepared for healthy rural occupations. Those found to be incurable should be placed in a section equipped with hospital comforts.

Klebs, of Germany, holds that the Ehrlich theory does not hold for tuberculosis. Permanent immunity must be active, and can only be produced in livings beings who destroy newly invading germs. The antagonistic theory of immunity based on this view is supported by the fact that vaccinated individuals show partial variolous eruptions, genuine variola eruptions appearing in such cases on certain portions of the body, such as the back and the abdomen, and they are so much the rarer as the attacked child stands nearer the time of vaccination. Klebs thinks that the tubercle bacilli of the blind worm are the antagonists of the tubercle bacilli of warm-blooded animals, and he uses the former in treating disease caused by the latter.

Raw, of Liverpool, thinks that the ophthalmic and cutaneous tests

are valuable in the diagnosis of tuberculosis in man, that the subcutaneous test is good for animals, but not desirable in man. He holds that the von Pirquet test should be made with a strong tuberculin, otherwise no effect is produced. While he does not use injections of tuberculin in the diagnosis of the disease in man he does employ it in the treatment and thinks it valuable for this purpose.

On the other hand, TURBAN, of Switzerland, says that the conjunctival test must be absolutely discarded on account of its danger. The cutaneous reaction, he holds, is too delicate, showing up when there are only local and insignificant lesions. The subcutaneous test he regards as the best for diagnostic purposes, and holds that it is safe if properly applied. The opsonic index denotes tuberculosis if it is below 0.9 or above 1.1, and if the opsonins are at the same time thermostable; but the normal state of the index, between 0.9 and 1.1, does not exclude tuberculosis. If the opsonins are thermolabile, while the index is too low, no tuberculosis exists, but a reduction of the resistance against it, caused by other diseases (diabetes, syphilis, influenza), must be presumed. A strongly oscillating index denotes progression of the disease, while a gradual, uniform rise leads to the presumption that there is an improvement. Turban states that the serum of Marmorek is a specific remedy, useful in a limited number of early cases of pulmonary and other forms of tuberculosis, but that it does not immunize. The various tuberculins are also regarded as specific remedies, the best according to Turban's experience being Koch's old tuberculin. The use of tuberculins is, however, difficult and not free from danger. There is in certain individuals a limit beyond which it is dangerous to go, even when the increase is made slowly. Surest protection from danger is obtained by watching the temperature, which should be taken in the rectum every two hours, combined with exact observations of the diseased centre. There are pronounced centre reactions in the lungs without any rise in temperature which may demand a suspension of the treatment.

D'ALMEIDA, of Portugal, urges the inspection of school children, the teaching of hygiene, and the use of vacation colonies, especially on the sea shore.

ALTSCHUL, of Prag, says: The principal duty of the modern school is the hygienic education of youth, education for cleanliness, for bodily and mental ability. Such hygienic instruction in school, consistently carried through from the beginning, may and must be followed up in riper years by a methodical teaching of hygiene, when, among other subjects, the question of the combat against tuberculosis must be discussed. The compendiums serving as guides for the instruction must be written by physicians and not by pedagogues, because the knowledge of medical science rectified by medical practice is absolutely indispensable for this purpose.

FRANZ, of Vienna, makes an interesting report upon a thousand soliders discharged in 1901 and 1902 because they reacted to tuberculin. His conclusions are as follows: (1) At the age from twenty-one to twenty-three years the strongest individuals often react to diagnostic doses of tuberculin; (2) the frequency of the reaction varies with the nationality, being greatest in those among whom tuberculosis is most common, as

the Bosnians; (3) of those who reacted at that time, only a small percentage has developed clinical evidence of the disease since; (4) up to the present time no basis has been found, either in the quantity of tuberculin required to produce the reaction or in the intensity of the reaction, for drawing definite conclusions concerning the character and course of the incipient tuberculous disease; (5) a dose of less than 3 milligrams is often not sufficient to ascertain a latent tuberculous infection.

Hope, of Liverpool, tells how tuberculosis has decreased in his city under improved hygienic conditions. In 1885 the mortality rate from this disease was 2.7 per 1000 among males, and 2.2 among females; in 1907 it was 1.9 and 1.1 respectively.

Köhler, of Germany, calls attention to the fact that consumptives are often neurasthenics, and he says that hygiene of the nervous system must begin in childhood. On account of the close relation between the hygiene of the nerves and the total energy of the body, the former must be considered, with the object of creating a strong and healthy people, as a part of the prophylaxis of pulmonary infection.

Morin, of Switzerland, emphasizes the value of sunlight in both the prevention and treatment of tuberculosis.

Onodi, of Budapest, claims that the nose is often the seat of a primary tubercular infection.

Petren, of Sweden, thinks that the sanatoriums should care for consumptives in all stages. He argues with much force that it is mutually beneficial to the incipient and the advanced to be kept together. The former are impressed with the gravity of the disease and stimulated to do their best, while the latter are cheered and saved from utter discouragement.

Taussig, of Prag, dwells upon the desirability of more reliable and thorough statistics concerning tuberculosis.

Unterberger, of St. Petersburg, discusses the hereditary transmission of tuberculosis. An inflammable building and a spark are necessary to a conflagration. The building may be fireproof; it may burn slowly, or it may be a tinder box. To prevent conflagrations we should build fireproof houses first of all, and then it is well to limit the flying sparks. Too much stress has been laid on the bacillus, and the warfare is being waged against the consumptive rather than the disease. The fear of the bacillus is warranted neither by biological research nor by practical observation. We must, of course, kill the bacilli, but we must give more thought to strengthening the body.

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ORIGINAL ARTICLES.

EXOPHTHALMOS AND OTHER EYE SIGNS IN CHRONIC NEPHRITIS.

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It is the purpose of this communication to direct attention to the frequent occurrence of exophthalmos in chronic nephritis, and to advance the view that the exophthalmos of chronic nephritis is very analogous to that of exophthalmic goitre, being but one of a number of evidences of a chronic systemic intoxication.

Exophthalmos can be easily produced experimentally by stimulating the cervical chain of autonomic fibers. According to the work of Landström[1] and MacCallum and Cornell,[2] this exophthalmos is due to the contraction of a cuff of plain muscle which, attached to the septum orbitale anteriorly and to the equator of the eyeball posteriorly, acts as an opponent of the recti muscles of the eye and produces exophthalmos when it is tonically contracted. This cuff of plain muscle is under the control of the autonomic fibers passing to the eye through the cervical autonomic chain.

[1] Ueber Morbus Basedowii, Thesis, Stockholm, 1907. The Pathogenesis of the Eye Signs of Exophthalmic Goitre, Editorial Jour. Amer. Med. Assoc., 1909, lii, 476, 6.

[2] The Mechanism of Exophthalmos, Med. News, Oct. 15, 1904.

It seems unquestionably true that toxins produced by certain diseases are capable of stimulating the autonomic system and producing, among other effects, exophthalmos. This toxic effect upon the autonomic nerve fibers is most familiar to us in exophthalmic goitre. In Basedow's disease the bodily functions under the dominion of the autonomic system are profoundly affected, owing apparently to overstimulation. Thus, the tremor, tachycardia, exophthalmos, sweating, vasomotor dilatation, and the evidences of irritation of the central nervous system are most probably accounted for by the assumption of chronic systemic intoxication, the exact nature of which need not concern us here.

Undoubtedly every physician of experience has occasionally observed exophthalmos and other eye signs in cases of chronic nephritis. It is surprising, therefore, that not one of the leading systematic treatises upon nephritis which we have been able to consult, makes mention of exophthalmos as a sign of chronic nephritis. It is a not infrequent finding, as we shall presently show. Though we have been observing exophthalmos in chronic nephritis and commenting upon it at the ward visits for several years, the material upon which the conclusions of this paper are based represent the medical service of the Johns Hopkins Hospital with chronic nephritis since January 1, 1909. Our total number of cases is too small to permit of very accurate statistical deductions, but they are valuable in that the patients have been studied with especial reference to the occurrence of exophthalmos and other ocular conditions in chronic nephritis.

Among the total admissions of thirty-three cases of chronic nephritis during the first four months of 1909, 16 (48.4 per cent.) showed exophthalmos. The exophthalmos varied greatly in degree, as did the gravity of the nephritic process in the various individuals; indeed, it may be stated that those cases presenting evidences of serious intoxication (suburemic or uremic symptoms) most frequently showed exophthalmos and one or more of the allied ocular signs—anisocoria, von Graefe's, Moebius', or Stellwag's sign. Exophthalmos has been an obvious sign in all of the cases of chronic nephritis which have died in the Johns Hopkins Hospital since January 1, 1909, seven in number. We have also observed that the cases of chronic nephritis showing albuminuric retinitis during this period have invariably shown exophthalmos, with one or more of the other ocular signs to be spoken of presently. It should be emphasized that exophthalmos is but one of several ocular signs which are frequently present in chronic nephritis.

We have already referred to the work of Landström in demonstrating the presence of a cuff of plain muscle attaching the eyeball anteriorly to the septum orbitale. This author thinks that a spastic condition of this plain muscle—the opponens recti—satisfactorily accounts for the widening of the lid slits (Stellwag's sign),

dissociation of the movements of the ball and lids (von Graefe's sign), and the break of convergence of the eyes (Moebius' sign). All of these classic signs of exophthalmic goitre may be present in a maximum degree in chronic nephritis without the thyroid showing any apparent involvement. The conclusion lies very near, that in both diseases a chronic systemic intoxication affecting the autonomic system is the causative factor in the production of the ocular manifestations. In our series of 16 cases von Graefe's sign was positive in 11 (68.7 per cent.), Stellwag's was positive in 13 (81.3 per cent.), and Moebius' sign was positive in 7 (43.7 per cent.). The pupils were noted as unequal in 5 cases (31 per cent.), and albuminuric retinitis was observed in 8 cases (50 per cent.).

Exophthalmos was associated with arterial hypertension (pressure above 160 mm.) in 12 of our 16 cases, and in 2 cases which may be instanced (Ray C. and Robert W. B.) a maximum degree of exophthalmos was associated with the maximum arterial tension shown by the patient; as the blood pressure fell the eyes became less prominent.

We are not of the opinion, however, that the exophthalmos is due to chronic hypertension. They may exist quite independently, and certainly only a small percentage of cases having arterial hypertension show exophthalmos. R. Sattler[3] has described exophthalmos in some cases of hemorrhagic retinitis with hypertension due to cardiac, renal, and hepatic lesions. It is far more reasonable to assume that the arterial hypertension and the eye signs mentioned are but evidences of poisoning by perhaps separate toxins. It is well known that uremia may develop in a patient whose blood pressure is not increased, and it seems very probable that in chronic renal insufficiency several toxins are present in the blood manifesting themselves in various ways.

Illustrative Cases. Case I.—Annie P., aged thirty-five years, black, laundress. Admitted November 30, 1908; discharged improved April 12, 1909.

Complaint. Shortness of breath and swelling of feet.

Family History. Negative.

Past History. Patient had rheumatism in winter of 1906–07 for three months, which seems to have been of the acute articular type. Patient gets up two or three times each night to urinate.

Present Illness. Dates from April, 1908, when she was admitted to the Johns Hopkins Hospital with dyspnœa, cough, frothy expectoration, and swelling of feet. A diagnosis of aortic and mitral insufficiency was made. After her discharge, in 1908, she remained fairly well for a time, but recently all of her many symptoms have returned in exaggerated form.

Physical Examination. It was noted that the eyes were prominent (Fig. 1), pupils were equal, moderately contracted, reacted slug-

[3] Arch. Ophthal., New York, 1885, xiv, 190 to 195.

gishly, and lids were puffy. There was a well-marked von Graefe's sign. There were signs of aortic and mitral insufficiency in the heart and effusion in the right pleural cavity. The patient had marked anasarca.

She gradually improved, and was discharged when able to walk around without much dyspnœa.

FIG. 1.—Case I.

The urine varied in specific gravity from 1010 to 1020, and in color from pale yellow to orange. The quantity was reduced, as a rule. Albumin was constantly present, ranging from 0.5 to 10 grams per liter by Esbach. Rather numerous hyaline and finely and coarsely granular casts.

The blood pressure averaged about 120 mm. Hg., reaching 140 occasionally.

CASE II.—Ray C., aged twenty-two years, single, Russian Jewess, a shop girl. Admitted January 1, 1909, complaining of twitchings of the right arm and hand and difficulty in speaking.

The patient had been admitted to this hospital also in 1904, when a diagnosis of hemichorea was registered. At that time the urine on four examinations was as follows: Pale yellow in color, specific gravity from 1008 to 1012, trace of albumin, and upon one occasion a few finely granular and hyaline casts. No exophthalmos was noted during this admission. The patient was discharged greatly improved.

The patient's family and past histories are negative except for frequent attacks of tonsillitis which were quite severe, necessitating the removal of the tonsils one year ago.

The attack which brought the patient to the hospital this time began November 1, 1908, with inability to write due to involuntary jerkings of the right arm and hand, and this muscular twitching has

since involved the face and tongue so that speech has been difficult. The patient also complained of headache and pain over the heart.

The physical findings upon admission were as follows: The eyes were markedly protuberant and the lids and conjunctivæ slightly œdematous (Fig. 2). Well-marked Moebius, von Graefe, and Stellwag signs. The heart was slightly enlarged, extending 11.5 cm. to the left in the fourth intercostal space. There was a loud, rough, well-transmitted systolic murmur, with an accentuated pulmonic second sound. The blood pressure upon admission was 245 mm. Hg.

Dr. Bordley examined the patient's eyes and noted a marked œdema of the disk and retina, obscuring most of the vessels on the border of the disk, with several small hemorrhages and white plaques scattered over both fundi. The changes were thought to be unquestionably of nephritic orgin.

Fig. 2.—Case II.

Three weeks after admission the patient developed a severe multiform erythema, which persisted for ten days. On February 10, she complained of excruciating headache and nausea, with vomiting, and at eleven o'clock had a general convulsion. This was followed by three similar convulsions during the afternoon. The patient responded well to treatment, and gradually returned to her former condition. Since these first convulsions the patient has had two similar attacks, with four and ten convulsions, respectively, and unconsciousness lasting from six to twelve hours.

The blood upon admission showed red blood cells, 5,400,000; white blood cells, 13,300; hemoglobin, 90 per cent. The leukocytes have ranged since then from 7000 to 25,000 and the red blood cells have fallen to 4,100,000, hemoglobin to 70 per cent. The urinary picture has been as follows: Total amount from 1 to $1\frac{3}{4}$ liters; pale

yellow, clear; specific gravity ranging about 1012; no sugar; heavy cloud of albumin, ranging from 2 to 10 grams per liter by Esbach; numerous hyaline and finely and coarsely granular casts.

The blood pressure upon admission was 245 mm. Hg., and since then has been elevated, with an average of about 215, frequently reaching 230.

Exophthalmos (Fig. 2) has been a very constant and striking feature, remarked by all observers. It has varied in degree from time to time, growing definitely less as the patient's condition improved and becoming more marked with each exacerbation.

Case III.—Robert W. B., married, aged fifty-one years, white, a painter. Admitted January 15, 1909. Died January 17, 1909.

The patient had been admitted to the hospital in 1901, and a diagnosis of lead poisoning with peripheral neuritis was then registered. He complained on present admission of severe pain all over the body, and headache.

Family History. Negative.

Past History. Patient says he has had malaria several times. He is subject to severe headaches and has been as long as he can remember. Eye sight has been failing steadily for the last five years. For the last two years he had been suffering with attacks of palpitation and orthopnœa. Gastric and intestinal disorder with vomiting and diarrhœa have been rather frequent of late. He has had to get up several times each night for the past five years to urinate, and passes his urine frequently during the day. The patient has been a heavy smoker, a constant beer drinker, and he very often drinks whisky before breakfast. In 1901 he suffered from a severe peripheral neuritis due to lead poisoning, and was treated in the Johns Hopkins Hospital. At that time the specific gravity of the urine was 1010, with a trace of albumin and a few hyaline casts.

Present Illness. For the past six months the patient has been unable to work much, and has felt very feeble and weak. During this time he has had frequent attacks of abdominal pain, has been constantly constipated, short of breath on exertion, and subject to orthopnœa. One week before admission he was confined to his bed with very severe headache, constipation, and was at times irrational. He passed large amounts of urine during the day and rose from one to seven times at night to urinate.

Physical Examination. Patient in a rather lethargic, torpid condition, respirations 16 to a minute, very deep, and with groaning expirations. Muscular twitchings of the arms noted on admission. The patient was thin, poorly nourished, very anemic looking, had pale mucous membranes and a dry, harsh skin. The eyes were very prominent and staring, the scleræ were visible above and below the iris. The right eye was more prominent than the left. The pupils were equal and reacted normally. There was marked dissociation of the lids, and the eyeball convergence was very poor. The heart

was slightly enlarged, with marked arteriosclerosis of the peripheral vessels. There was a double wrist-drop present.

Upon the day following admission the patient had a general convulsion. It was followed by two convulsions a few hours later. Upon ophthalmoscopic examination there was a well-marked albuminuric retinitis, with œdema, exudate, and hemorrhages in both eyes. The retinal arteries were tortuous and small.

The blood showed 3,000,000 red cells, 11,600 white cells, 35 per cent. hemoglobin. Grawitz granules were demonstrated in the red blood cells. The blood pressure upon admission was 160 mm. Hg., and this fell to 110 at time of death. *It was noted that the exophthalmos*, so marked upon admission, was much less marked at the time of death.

The urine upon two examinations was noted as light yellow; specific gravity, 1012; albumin positive, with large numbers of granular casts, pus cells, and epithelial cells.

The diagnosis of chronic nephritis with acute exacerbation following saturnism and chronic alcoholism was made.

Autopsy, No. 3155. Anatomical diagnosis: Extreme grade of acute and chronic diffuse nephritis; œdema of the brain; concentric hypertrophy of the heart; bilateral bronchopneumonia; pleural œdema; acute pericarditis; fatty degeneration of the liver; anemia.

Case IV.—Jos. B., married, aged forty-eight years, white, a tailor. Admitted January 13, 1909. Discharged February 25, 1909, improved.

Complaint. Cough, pain in back.

Family History. Negative.

Past History. Negative except that for some years he has had to get up two or three times at night to urinate. Drinks five or six glasses of beer daily, two glasses of whisky, and gin occasionally.

Present Illness. Began four weeks ago with a cold and cough and pain in back. His head has ached violently and his eye sight has been growing worse. He has had anorexia and polyuria for the past four weeks.

Physical Examination. Patient was very pale and drowsy. Slight dyspnœa, 28 to minute. The eyes were noted as very prominent and staring, lids somewhat œdematous and inflamed. Right pupil was larger than the left, both reacted to light and accommodation. The heart was slightly enlarged; sounds clear. During the examination the patient became violently delirious and had to be placed in a restraining jacket.

Dr. Randolph, upon ophthalmoscopic examination, noted a high grade of arteriosclerosis of the retinal vessels with several hemorrhages in both fundi; also marked exophthalmos, the left eye being more prominent than the right, and a high grade of peripheral arteriosclerosis.

There were 5,200,000 red blood cells, 11,200 white blood cells,

80 per cent. hemoglobin. The blood pressure upon admission was 240; fell to 140 under continuous use of sodium nitrite.

The urine upon admission was pale yellow, clear, increased in quantity; specific gravity, 1012; marked trace of albumin with moderate number of finely granular and hyaline casts.

As the blood pressure fell the urinary output became greatly decreased, being as low as 150 c.c. in twenty-four hours. The specific gravity averaged 1020, going as high as 1032. The hyaline and granular casts persisted.

The patient left the hospital greatly improved, and a diagnosis of chronic nephritis with uremia was registered.

CASE V.—Oscar T. H., married, aged forty-six years, white. A musician. Admitted March 2, 1909. Died March 5, 1909.

Complaint. Shortness of breath.

Family History. Unimportant.

Past History. The only item of importance is that the patient says he has to get up every hour during the night to urinate. Voids a large quantity during the night, two quarts he thinks. He does not urinate very much during the day. Of late he has had to get up only three or four times during the night. Patient has drunk beer and whiskey to excess, and has been a heavy smoker.

Present Illness. Began two years ago, when patient became short of breath while blowing a cornet as he was marching up hill. This shortness of breath has grown markedly worse, and he had to give up blowing musical instruments. About a month ago he began to be giddy and dizzy upon attempting to work, and this symptom has persisted. His feet have been swollen for the past two weeks.

Physical Examination. Revealed a pallid, dyspnœic man with typical Cheyne-Stokes respiration. Dr. Thayer noted that the radials were thickened, feeling like small cords. The temporals were markedly arteriosclerotic. The eyes were distinctly exophthalmic, and in both fundi there were exudate and hemorrhages together with neuroretinal œdema, presenting a picture of albuminuric retinitis. The heart was enlarged 15.5 cm. to the left and 5 cm. to the right. There was a gallop rhythm to the heart, but no distinct murmurs were audible. The blood pressure ranged from 230 to 200 mm. Hg. There were numerous fine rales at the bases of both lungs. The liver was easily palpable, and there was shifting dulness in both flanks. Dr. Thayer summed up the case as follows: Marked Cheyne-Stokes respiration; œdema of lungs; enlarged heart from high blood pressure; large liver; œdema of back and legs; albuminuric retinitis, and the case was diagnosticated as chronic interstitial nephritis.

There were 5,800,000 red blood cells, 8600 white blood cells, 87 per cent. hemoglobin. The urine was not increased in amount. It was pale yellow and clear; specific gravity, 1010; heavy cloud of albumin; numerous coarsely granular and hyaline casts.

Patient sank into coma and died three days after admission.

Autopsy, No. 3179. March 5, 1909. Anatomical diagnosis: Extreme arteriosclerosis with almost complete obliteration of many of the principal arteries, including the renals, mesenteric, hepatic, coronaries, cœliac, etc.; extreme hypertrophy of the heart; fibrous myocarditis; atrophied kidneys; degenerative changes of the kidneys; chronic passive congestion of the lungs.

Case VI.—J. J. McF., male, single, aged thirty-two years, white. A bridge worker, using lead paints a great deal. Admitted February 24, 1909. Died March 3, 1909.

Complaint. Pain in the left side and shortness of breath.

Family History. Negative.

Past History. Unimportant, except that the patient has been a worker in lead paints all his life, has drunk freely of beer and whiskey, and has smoked moderately.

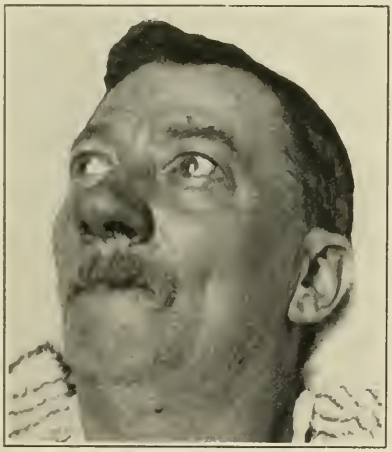

Fig. 3.—Case VI.

Present Illness. Patient says he was well until four days before admission. Went to bed at 10 P.M., and as soon as he got into bed became suddenly short of breath. He was unable to breathe lying down. This attack lasted for half an hour. Pain then developed in the left chest, with a cough. Pain in the left side and cough have continued for the past four days and the patient's bowels have not moved during this time.

Physical Examination. There was marked staring exophthalmos of both eyes (Fig. 3). Von Graefe's and Moebius' signs were well marked. The pupils were equal and reacted normally. The heart presented evidence of a pericardial effusion; a systolic murmur at the apex was heard in addition to a pericardial friction rub. There was a pleural friction rub with slight dulness at each base behind.

There was a tophus in the right ear, which contained sodium biurate crystals, and the patient gave a history of arthritis of the big toes, so that he certainly had gout. Dr. Randolph examined the fundi oculi and noted the presence of albuminuric retinitis in both.

On March 1 the pericardium was tapped and about 30 c.c. of hemorrhagic fluid removed.

Blood Examination. 2,900,000 red blood cells, 18,000 white blood cells, and 48 per cent. hemoglobin. The blood pressure was 170 mm. Hg., and fell to 150 at time of death.

The urine was constantly increased in amount, pale yellow; specific gravity, 1010 upon four examniations; albumin 1.5 to 2 grams per liter by Esbach, and there were hyaline, granular, and waxy casts.

Autopsy, No. 3176. March 3, 1909. Anatomical diagnosis: Chronic diffuse nephritis, small granular kidney; lead poisoning; cardiac dilatation and hypertrophy; arteriosclerosis; organizing hemorrhagic pericarditis with effusion; serofibrinous pleuritis, bilateral with effusion; hemorrhagic bronchopneumonia; œdema and atelectasis of the lungs; gouty arthritis; tophi; encapsulated typical tuberculous foci.

Case VII.—William B., aged forty-six years, white. A metal polisher. Admitted March 4, 1909. Discharged April 5, 1909, improved.

Complaint. Shortness of breath and pain in back and left side.

Family History. Negative.

Past History. Without bearing upon the present illness.

Present Illness. Began four weeks before his admission with shortness of breath and palpitation. He has been troubled pretty constantly of late with attacks of dizziness. He is dyspnœic upon slight exertion. He is up six or eight times during each night to void. Urine is pale, clear, and greatly increased in quantity. He is troubled with cramps in the calves of his legs at night. He occasionally has numbness of the right arm and side, which he has noticed during the past year. The legs are sometimes swollen in the morning.

Physical Examination. Revealed a well-nourished man, slightly dyspnœic. *The eyes were markedly exophthalmic* (Fig. 4). The conjunctivæ were slightly œdematous. The pupils were small, tending to myosis, reacting to light. There was a positive Moebius' sign. No von Graefe's or Joffroy's signs. The lid slits were widened. Dr. Bordley, upon ophthalmoscopic examination, noted that there was marked retinal arteriosclerosis and typical albuminuric retinitis, as evidenced by neuroretinal œdema, white plaques, and numerous hemorrhages in both fundi. The heart was enlarged, extending 14.5 cm. to left in fifth interspace and 4 cm. to right. The heart was irregular in protodiastolic gallop rhythm, and a fine systolic murmur at apex. There was marked general arteriosclerosis.

Dr. Barker summarized the case as follows: "History of alcohol-

ism, acne rosacea, dizziness, palpitation, dyspnœa, polyuria, and pollakiuria, albuminuria, cylindruria, exophthalmos, gallop rhythm, marked arteriosclerosis. The patient probably has a contracted arteriosclerotic kidney with enlarged heart due to the renal and vascular changes. Quite possibly he has a beginning cerebral arteriosclerosis. The heart is beginning to show mitral insufficiency—probably relative."

Blood. Red blood cells, 4,400,000; white blood cells; 8600; hemoglobin, 70 to 80 per cent.

Fig. 4.—Case VII.

The urine was increased in amount, pale yellow; specific gravity averaged 1012; albumin 0.5 to 2 grams per liter; moderate number of hyaline and finely granular casts.

There was chronic arterial hypertension, the pressure seldom falling below 200 and rising as high as 230 on several occasions. The patient's family (wife and daughter) state that the eyes have grown prominent during the past eight months. The diagnosis of arteriosclerosis, chronic nephritis, and myocardial disease was registered.

Case VIII.—Jeremiah F., married, aged sixty-four years, black. A cook. Admitted April 1, 1909. Discharged April 28, 1909, improved.

Family History. Negative.

Past History. Without bearing upon present illness. For about a year patient has had frequency of micturition, up three or four times at night to void. The same number of times during the day.

Present Illness. Began March 25, 1909, with an attack of shortness of breath. He had pain in his lungs of an acute cramp-like character, and had a severe cough and orthopnœa. His legs began to swell a week before admission.

Physical Examination. The patient was poorly nourished. The skin was dry and harsh. The eyes showed well-marked exophthalmos and positive Stellwag's, von Graefe's, and Moebius' signs. The right pupil was larger than the left. The pupils reacted very sluggishly to light and accommodation. The heart was slightly enlarged, extending 12 cm. to left in fifth intercostal space, and 4.5 cm. to right. The action was irregular with extra systoles. No murmur. There was marked general arteriosclerosis.

Red blood cells, 3,530,000; white blood cells, 4900; hemoglobin, 60 per cent. The blood pressure was normal.

The urine was pale yellow in color, from 1012 to 1020 in specific gravity, with a trace of albumin and a few hyaline casts. The total daily amount of urine was rather small.

Case IX.—George H. E., widower, aged fifty-three years. A painter. Admitted March 8, 1909. Died forty-eight hours later.

Complained of pain in stomach.

The *family and past history* contain nothing of importance.

The *present illness* began about January 1, 1909. Patient began to lose his appetite and had dull burning pain in the abdomen. This pain bore no reference to food taken. No vomiting. For the past five months the patient has had to get up several times each night to urinate.

Physical Examination. Patient presented a haggard, pallid, emaciated appearance, with a marked dyspnœa. The eyes were strikingly prominent, giving a staring expression to the face. Stellwag's and von Graefe's signs were present. The left pupil was slightly larger than the right; both reacted to light and accommodation. Both parotid glands were swollen and tender. The mucous membranes were pale, and the skin was harsh and dry. The heart was enlarged, extending 12.5 cm. to left. No murmurs. There was diffuse arteriosclerosis in peripheral vessels. The epigastrium was filled with a tumor mass, which extended 8 cm. below costal margin in right mammary line, 3 cm. above umbilicus in mid line.

The red blood cells were 3,260,000; white blood cells, 7600; hemoglobin, 60 per cent. The blood pressure was 110 upon single examination.

Two urinary examinations were made. The urine was greatly reduced in amount, catheterization being necessary to obtain a specimen. The urine was yellow, specific gravity was 1020, distinct trace of albumin; and contained hyaline finely and coarsely granular casts.

The patient died rather suddenly on the second day in the hospital. No autopsy obtained. The tumor mass in the epigastrium was thought to have been carcinoma.

Case X.—Herman G. M., aged fifty-two years, white. A bricklayer. Admitted April 5, 1909. Discharged improved April 19, 1909.

Complaint. Shortness of breath.

Family History. Negative.

Past History. Patient had malaria thirty years ago and pleurisy twenty-two years ago. Eight years ago, in April, 1901, patient had a paralytic stroke of the right side, involving face, arm, and leg. He recovered almost completely from this attack. The patient drinks gin and whiskey to excess and chews a plug of tobacco a day. His occupation exposes him to inclement weather and subjects him to hard work.

Present Illness. Began two weeks ago, March 23, 1909, with drowsiness, weakness, and shortness of breath. A week later patient had pain in epigastrium just above umbilicus. Appetite became poor and bowels constipated. For the past three days he has vomited two or three times a day. Patient has had to get up eight or nine times at night to void, and about nine or ten times during the day. His ankles and legs are sometimes swollen.

Physical Examination. Revealed a short plethoric individual, dyspnœic, breathing 36 to minute. The eyes were markedly prominent, producing a distinct stare. There was a fairly well-marked von Graefe's sign. The right pupil was slightly larger than the left, both reacted normally. The heart was enlarged 16 cm. to left in fifth intercostal space, and 4.5 cm. to right. No murmurs. There was a well-marked protodiastolic gallop rhythm. There was very slight œdema of the feet.

No ophthalmoscopic examination was made.

Red blood cells, 5,168,000; white blood cells, 14,200; hemoglobin, 95 per cent. The blood pressure ranged from 200 to 130, remaining most of the time between 180 and 200 mm. Hg.

The urine was reduced in amount to 500 c.c. or less in twenty-four hours, which was probably due to the free purgation and sweating. The urine was reddish yellow in color, averaged 1022 in specific gravity; albumin, 0.25 grams per liter by Esbach on five examinations. There was a moderate number of finely granular and hyaline casts.

Case XI.—Henry R., aged sixty-one years, black. Admitted April 28, 1909.

Complaint. Pain in back.

Family History. Unimportant.

Past History. Patient had diphtheria at fourteen, malaria six years ago, and had cough with shortness of breath for past two years. Chronically constipated. The patient is up three times each night to urinate. Has some difficulty in starting stream. Patient has been a heavy drinker for past ten years, taking eight glasses of beer and two or three glasses of gin or whiskey a day. Usually drinks a glass of beer every morning before breakfast. Has worked hard all his life, and is exposed to the weather a great deal. Thinks he has lost 18 pounds in the past year.

Present Illness. In February, 1909, the patient was taken with sudden sharp pain in the small of his back and became very short of breath. The pain in his back was so severe that he came to the Dispensary and was put in a cast two weeks ago, but without relief. This pain has been present for last two months and is worse on coughing and when he stoops over. Has been constantly dyspnœic and orthopnœic. About a week ago his stomach and legs began to swell.

Physical Examination. Patient is irrational, though not violently delirious. Breathing, 32 to minute; breath, urinous. The eyes are moderately exophthalmotic; lid slits wide; pupils very small and react sluggishly. Von Graefe's sign is positive and convergence is very poor. The mucous membranes are pale, and skin is harsh and dry. The heart is enlarged, extending 15.5 cm. to left in fifth intercostal space, 3.5 cm. to right in fourth intercostal space. There is a loud transmitted systolic murmur at apex and a diastolic murmur in aortic area. The liver is somewhat enlarged. Movable dulness in the flanks and definite œdema of the ankles.

Red blood cells, 2,880,000; white blood cells, 6,800; hemoglobin, 50 per cent. Blood pressure is 140 mm. Hg.

Urine is decreased in amount, orange yellow, specific gravity 1014, and contains hyaline and finely and coarsely granular casts.

Case XII.—George L. P., married, aged thirty-three years. Admitted to the hospital April 25, 1909, and died the same day. Of importance in the family history is the fact that the patient's father and his sister died of tuberculosis, and both grandparents died with Bright's disease.

Past History. Had scarlet fever at eleven without apparent sequels. History otherwise negative.

Present Illness. Began five months ago. Patient noticed he was passing much more urine than normal, up four or five times each night to urinate. Two months ago patient's eyes began to fail him, objects were blurred, and at times he was almost completely blind. Severe headaches have been present for the last two months. The patient grew gradually weaker, and he suffered a great deal with nausea and vomiting. His condition grew gradually worse until nine days before admission, when he took to his bed and vomited constantly. Since this time his urine has been very scanty.

Physical Examination. Positive findings were as follows: Patient was thin and anemic in appearance. The eyes were very prominent, giving a markedly staring appearance to the face. Stellwag's and von Graefe's signs were well marked. Pupils were equal and reacted to light and accommodation. The mucous membranes and skin were very dry. The heart was enlarged, extending 11.5 cm. to left in fifth intercostal space and 3.5 cm. to right in fourth intercostal space. There was a short systolic murmur at apex. The aortic

second sound was louder than the pulmonary second. There was moderate arteriosclerosis.

Upon ophthalmoscopic examination a marked albuminuric retinitis was revealed, with neuroretinal œdema and many white patches of exudate. Several large hemorrhages in both fundi.

The blood picture was as follows: 4,192,000 red cells, 21,800 white cells, 60 per cent. hemoglobin. The blood pressure was 120 mm. Hg.

The urine was decreased in amount; specific gravity, 1012; pale yellow in color; albumin in large quantity, Esbach, 4 grams per liter, and there were fairly numerous granular casts.

Case XIII.—Daniel H., aged fifty-five years, married. A farmer. Admitted April 29, 1909, complaining of weakness. Died in coma May 1, 1909.

The *family* and *past history* are without bearing upon the present illness. The patient gave a history of rising once each night to urinate.

The *present illness* began a year ago. The patient noticed that he was gradually growing weaker. Exertion caused him shortness of breath, which symptom has grown constantly more marked during the past year. About one month ago he noticed that his sight was failing, and since then has been unable to read ordinary print. There has been no œdema of the feet and ankles. He has been getting up two or three times at night to urinate, but thinks the total quantity voided has not been greatly increased. Ten days before admission the patient sank into a coma from which he could be easily aroused. Four days ago the patient had a general convulsion, since when he has been unconscious, and was brought into the hospital in a comatose condition.

The positive physical findings were as follows: Patient comatose, and at times there are rapid twitchings of the right arm. There is a coarse tremor of the hand. Breath is urinous. Mucous membranes are pale. The right pupil is larger than the left. Both react normally. The eyeballs are markedly prominent and there is a well-marked von Graefe's sign. The right eyeball seems more prominent than the left. Upon ophthalmoscopic examination there was a most marked neuroretinal œdema, arteries tortuous, and scattered throughout both fundi numerous hemorrhages and white plaques, these being especially numerous around the macular region. An albuminuric retinitis was diagnosticated.

The blood showed 3,920,000 red blood cells, 10,680 white blood cells, 73 per cent. hemoglobin. The blood pressure upon admission was 210 mm. Hg., and sank gradually to 130 at time of death, four days after admission.

The urine was noted as light yellow, clear; specific gravity, 1010; heavy cloud of albumin with a few granular casts.

The patient did not respond to treatment, and died four days after admission.

Case XIV.—Annie J., aged fifty years, widow.

Diagnosis. Chronic nephritis; arteriosclerosis; had four former admissions, with diagnosis of chronic nephritis in each. The eyes were staringly prominent, marked Stellwag's and von Graefe's signs.

Heart 13.5 cm. to the left in the fifth intercostal space. No murmurs.

Urine, 1005 to 1020; pale yellow; contains albumin and a few granular casts. Blood pressure from 240 to 180 mm. Hg.

Autopsy, February, 1909. Anatomical Diagnosis: Arteriosclerosis; obesity; chronic diffuse nephritis; chronic fibrous myocarditis; cardiac hypertrophy and dilatation; chronic passive congestion of viscera.

Case XV.—Henry P., married, aged 55 years.

Diagnosis. Arteriosclerosis; myocarditis; chronic nephritis; mitral insufficiency. Eyes noted as exophthalmic. No mention of other ocular signs.

Heart 14 cm. to the left in fifth intercostal space. The first sound at the apex is followed by a rough transmitted murmur. The patient is very dyspnœic.

Urine, 1010 to 1020; albumin, 1 gram per liter by Esbach; a few hyaline and granular casts; pale yellow is the color. Blood pressure, 160 to 190 mm. Hg. No ophthalmoscopic examination. Discharged improved.

Case XVI.—Henry P., aged fifty-four years.

Diagnosis. Chronic nephritis; cirrhosis of the liver. Death and autopsy.

The patient gave history very typical of nephritis. Up three or four times each night to urinate; anemic.

The eyes were noted as quite prominent; ocular movements were good.

Urine, light yellow; specific gravity, 1010 to 1012; contains albumin and many granular casts. Blood pressure, 160 mm. Hg.

Autopsy. Anatomical diagnosis: General arteriosclerosis; chronic diffuse nephritis; pulmonary œdema; cirrhosis of liver.

THE NATURE OF THE ARTERIOSCLEROTIC PROCESS.[1]

By J. George Adami, M.D., F.R.S.,
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Surely all of us who are active pathologists have been impressed by the fact that the more autopsies we perform the larger does arteriosclerosis loom as the fundamental morbid process in the majority of deaths after the age of forty years. If trauma, malignant disease, and infections, such as pneumonia and the rarer tuberculosis, be eliminated, we are left with that vast mass of cases of cardiac incompetence, aneurysm, chronic bronchitis and emphysema, cerebral apoplexy, and chronic Bright's disease; and the more we study these cases the more it is borne in upon us that in these the terminal event is but an outcome of the one common underlying process of arterial disease. At the same time, the more carefully we study the arteries in these cases the more we recognize that we deal with an almost protean series of disturbances: now it is the upper portion of the aorta, the ascending limb, or the arch that shows the most pronounced and obvious lesions; now the abdominal aorta; in other cases the aorta is relatively free from sclerotic change, but its larger branches show irregularities, this with or without pronounced involvement of the smaller arteries, which in their turn are not equally affected; at times the renal, at times the cerebral, at times the arteries of the extremities are the seats of most advanced change. In the second place we find that the lesions are not all of one order, so much so that it may well be debated, as it is debated, whether one common name should be applied to the series. I am free to acknowledge that this name arteriosclerosis is not wholly satisfactory. Marchand[2] has recently recommended that of atherosclerosis, but this, like the older atheroma, is inadequate, in that it implies that the development of ἀθήρα, or a porridgy degeneration, is inevitable. Something might be said for this suggestion had we to deal only with the aorta and the larger vessels, although even in these there are developed sclerotic states—I refer to those of syphilitic origin—which do not by any means necessarily pass on to an atheromatous stage. (These Marchand would place in a separate class.) It is, however, inadmissible for in the condition as found in the smaller sized arteries the sclerotic change is predominant, and the atheromatous or degenerative is characteristically absent. Again, it may rightly be objected that the atheroma is a secondary and not a primary condition; it is always the secondary outcome of fibrosis. "Arteriocapillary fibrosis," save for its flatulency, has more in its favor

[1] The annual address delivered at a meeting of the Pathological Society of Philadelphia, April 24, 1909.

[2] Verhandl. Deut. path. Gesellsch., 1906.

than atherosclerosis, but again is incorrect, for the capillary changes are not obvious, and, as I shall point out, when present must be regarded as secondary. We come back, therefore, to this, that the term "arteriosclerosis" is the most satisfactory name that has so far been afforded for this series of arterial changes, since sclerosis, which is equivalent to fibrosis, is the condition of commonest occurrence in the series of included conditions.

What, then, are the different forms that we have to take into account? So much has been written upon the subject of late years that I need only refer to the salient points, for doubtless the majority here present have as full a knowledge of this matter as have I who speak. And first as to the main orders of disturbance seen in the aorta and its larger branches—in the vessels, as we may term them, of the elastic type:

1. Most common is the ordinary nodose arteriosclerosis. This shows itself in the slightest cases as a thickening and sclerosis, more particularly at or around the origins of the side arteries, as yellowish, white thickenings notably affecting the origins of the series of intercostal arteries. Later these have grown in size, and some may have coalesced, forming in the opened aorta projecting flattened nodes, and now these may be scattered somewhat irregularly through the aorta, tending to be more abundant and more advanced in the abdominal region. It is this form that, more particularly, passes into the condition of atheroma, and of calcification of the intimal plaques.

2. Almost as frequent is a contrasted order of disturbances, in which clinically we have the most obvious proof of the existence of arteriosclerosis, in the presence of hardened and at times pipe-stem radials; but when we come to examine the aorta we may in the most typical cases find a complete absence of nodose thickening of the intima. In its place there is a diffuse dilatation of the aorta, affecting more particularly the thoracic portion. Instead of being thickened, the aortic wall appears to be thinned and the increased volume of the tube tends to show itself not only in breadth, but also in length, the vessel becoming somewhat tortuous; the arch takes a wider sweep, the abdominal section is curved. This I am inclined to regard as the uncomplicated senile type of the disorder. It is further characterized by the presence, in the common iliacs and the carotids, of slight depressions, tending to have their long axis situated transversely, in fact, by the very reverse of the sclerotic nodosities, a giving way taking the place of thickening of the wall. And the question may well be asked: Should this be included? It is not a sclerosis, but merely a degeneration. This I freely admit, but would urge that the condition of the whole arterial tree has to be taken into consideration in making our diagnosis, and if we do this we find that in these cases the smaller arteries are markedly sclerotic, with localized areas of intimal fibrosis, with hypertrophy and fibrosis

of the middle coat and often with well pronounced peri-arterial fibrosis. The sclerosis of the radials and other middle-sized arteries, often so prominent in these cases, is paradoxically not a sclerosis proper; it is not necessarily due to fibrosis of the intima or other coats. Sometimes there is a diffuse thickening of the intima of moderate grade, but that is not the cause of the hardening. As Dr. Klotz[3] has most serviceably pointed out, the essential change in vessels of this type is a calcification of the media, which I may add, following Russell,[4] is apt to be preceded by a marked hypertrophy of the middle-coat. For convenience, I would speak of this order of arteriosclerosis as Moenckeberg's sclerosis, Moenckeberg[5] having been the first to make a full study of what is the most striking feature, namely, the areas of giving way and slight sacculation seen in the main branches of the aorta due to degeneration of the media. But, doing this, I would impress upon you that uncomplicated examples of this order of disturbance are relatively uncommon. Quite the commonest condition in elderly people is a combination of these two types, having occasional nodes or plaques of sclerosis or atheroma in the aorta, more particularly in its abdominal portion, though by no means necessarily confined to this region, with this faint saccular giving way in the common iliacs and carotids, suggesting strongly that the same order of events leads now to the one, now to the other development; that, in fact, they are diverse manifestations of a common state.

3. The third great type is the syphilitic. The aortic manifestations of this form are in the broad features so much of the same order as those of ordinary nodose arteriosclerosis that it is only during the last generation and, indeed, during the last seven years that the distinguishing features have attained general recognition. Thanks to the labors of Döhle[6] of Kiel and other pupils of Heller, and of Chiari,[7] we now have no difficulty in making the distinction, if not always by the naked eye, then surely under the microscope. The seat of election in the first place is in the ascending aorta and arch, and here the nodes are liable to lie in groups. These in their earlier stage are large and, if I may so express it, succulent, with a semi-translucent or hyaline appearance; characteristically they have little tendency to atheromatous and calcareous change, but, on the contrary, exhibit a later scarring or central depression with some puckering. The reason for this is found upon microscopic

[3] Calcification of the Aorta in Rabbits after Injection of Adrenalin, Jour. Exp. Med., 1906, viii, 325; Calcification of the Media in Arteries of the Elastic Tissue Type, Jour. Exp. Med., 1906, viii, 330; The Relationship of Experimental Arterial Disease in Animals to Arteriosclerosis in Man, Jour. Exp. Med., 1906, viii, 304; Experimental Arteriosclerosis, Brit. Med. Jour., December, 1906; Experimental Work-Arteriosclerosis, Montreal Med. Jour., March, 1908, and Centralbl. f. allg. Pathol., 1908, xix, 535.

[4] Arterial Hypertonus, Sclerosis, and Blood Pressure, Edinburgh, 1907.

[5] Virchow's Archiv, 1903, clxxi, 141.

[6] Inaug. Diss., Kiel, 1885.

[7] Präger med. Woch., 1806, xxxi, No. 12; Verhandl. Deut. path. Gesellsch., 1904.

examination. The primary disturbance here is subacute mesaortitis, with small-celled infiltration around branches of the vasa vasorum and absorption of the elements proper of the media. Coincidently there is overgrowth of the intimal tissues, and when, as a result, the deeper portions of the overgrowth exhibit necrotic change and degeneration, then the underlying inflammatory granulation tissue advances new capillaries into the necrotic area. The result is an absorption of the degenerated material, replacement by cicatricial tissue, shrinkage, and scarring. So far as my observations go, there is a striking lack of vascularization and replacement in the ordinary nodose arteriosclerosis. It does not occur during the necrotic atheromatous stage; I have encountered it when there is already pronounced intimal calcification and the necrotic change is extending into the inner layers of the media.

Here in connection with syphilis, and yet more markedly, there is to be observed that same paradox seen in connection with the Moenckeberg type; while the syphilitic virus leads to the more pronounced intimal overgrowth, at the same time it leads characteristically to the opposite condition of lack of intimal overgrowth, thinning of all the coats, and aneurysm production. It will be familiar to you that modern workers, with the exception of von Hansemann, ascribe from 60 to 85 per cent. of aneurysms to syphilis. How are we to reconcile these opposing results?

These are the main types. Acute changes do not come under the heading of arteriosclerosis, nor do simple degenerative changes without proliferation. The fatty streaks of the intima seen often after acute infection, are not here included, although these and certain other intimal changes may throw light on certain phases of the process. To them I shall refer later.

Turning now to the other arteries, we may, as regards arteriosclerotic change, divide them into the muscular arteries and the arterioles. As regards the former, the same changes, the ordinary thickening of the intima, the giving way of the Moenckeberg type, and the syphilitic changes may be seen as in the aorta, with, however, one or two modifications which would seem to be a function of relative size. Thus we do not encounter anything like the same extent of atheromatous and degenerative change. Compared with the size of the artery, the areas of intimal thickening may be extreme; nevertheless, in absolute size they do not compare with the plaques seen in the aorta. And it may be reasonably suggested that degeneration when it occurs is due to cutting off of the nutritive lymph supply by the development superficially of layer after layer of dense new connective tissue, and that in general the size of those plaques in the arteries (save in the larger cerebral vessels and the coronaries) is not sufficient to inhibit the percolation of lymph and arrest of nutrition of the more central areas. This does not, however, mean that there may not be extreme calcification, more especially in the

middle sized arteries; only that calcification affects, more particularly not the intima, but the media.

This brings us to the second point of difference, namely, that in these arteries the media exhibits much more obvious alteration. Where there are relatively large plaques of intimal thickening there beneath them, even with the naked eye, the media is seen to be notably thinned. But, on the other hand, it may exhibit diffuse thickening from hypertrophy of its muscle tissue, with little obvious intimal change. On the other hand, there may be degeneration of the muscle and replacement by connective tissue; in some regions this may be associated with giving way and dilatation of the arterial lumen with associated intimal thinning; in others there is seen a more or less diffuse thickening and overgrowth of the intima, suggesting compensatory change. Passing to the arterioles, we find, it is true, the same series of changes, but here the relationships are significantly altered. There has been active debate as to their significance and relative frequency. In one order of cases the muscular hypertrophy of the middle coat has been the dominating change. This is especially well seen in certain kidneys, so much so that sundry observers have laid stress upon this as the cardinal change. In another series fibrosis is the most evident alteration—fibrosis of the *intima*, fibrosis replacing largely the *muscular* tissue of the *media*, fibrosis and pronounced overdevelopment of the *adventitia*. So that other observers have waved the muscular change to one side and regarded the fibrotic change as the all-important condition.

Yet another state has also been dwelt upon, particularly in association with the infectious type, as seen in tuberculosis, syphilis, and chronic glanders, namely, the active cellular proliferation of the intimal endothelium leading to an obliterative endarteritis. It is still unsettled whether this proliferation is directly connected—as Baumgarten believes it to be—with the production of the intimal fibrosis—whether, that is, the endothelium directly gives origin to the connective tissue overgrowth of these arterioles, or whether the processes are distinct. Provisionally I incline to Baumgarten's opinion.

How now are we to harmonize and bring into a common scheme these various lesions? What, in short, is the fundamental underlying change—if there be any—common to be different conditions? The answer to this question can be approached in two ways—either by an exhaustive analysis of the factors seen to be in action in the various types of arteriosclerosis; or by taking one form in which the causative factors have been most fully determined and applying the results gained from the study of this form to the other cases, to observe whether any common general principles are capable of application to all. Both, I believe, lead surely to the same end result, and let me confess that I have been puzzled as to which method to employ. It seems to me, however, that the former would con-

sume too much time; it would demand a preliminary discussion of the minute anatomy and of the physiology of the arterial tree, of the regulation of the blood pressure, and the influence of contraction of the arterioles upon the aorta and the aortic conditions. It is true that, employing the other method, reference must be made to most if not all of these matters, but I imagine that it will be less wearisome if I take up a concrete case and then proceed to apply the results gained from that to a consideration of the other cases.

Now undoubtedly we possess the clearest and most indubitable data regarding the syphilitic form. There is no question nowadays that the primary lesion in syphilitic aortitis is a granulomatous condition or small-cell infiltration immediately surrounding the terminal vasa vasorum in the media; the primary condition is, as Chiari describes it, a syphilitic mesaortitis, and this involves in the first place the outer half or two-thirds of the aortic media. Certain of the vasa only are affected. Here, just as in acute mycotic aneurysm, as shown by Dr. J. McCrae's[8] study of a case published from our laboratory, the infective agent gains entrance into and lodgment in particular vasa. And here it may be noted that several observers have discovered the spirochete in the aortic lesions. With this infiltration there is, as Dr. Klotz points out in an article shortly to be published, a remarkable dissolution of the elements of the media. They seem literally to melt away. This explains the thinning and depressions which may occur, so that, as Aschoff points out, placed against the light these areas are semitranslucent. To what an extent this process may extend is well shown in the accompanying specimen (Fig. 1) which Dr. Klotz has been so good as to permit me to demonstrate to you. This has been stained to demonstrate the elastic tissue. You will observe that in the region where there is the most extensive small-cell infiltration the elastica has completely disappeared.

I will not say that this dissolution is specific. I do not believe that it is. I am inclined to think that it may be the outcome of local inflammation, due to more than one cause, with swelling, degeneration, and necrosis of the cells of the involved area. But undoubtedly the most frequent cause is syphilis. Here, therefore, is the onset of the syphilitic or mycotic type. It will be readily understood how such extreme destruction of the media, the main sustaining coat of the aorta, leads on to aneurysm. In fact, we may say that it is universally acknowledged nowadays that degeneration of the middle coat is the dominant cause of aortic aneurysm. But such extreme destruction of the middle coat as here shown is the exception and not the rule; so again in syphilitic aortitis aneurysm production is the exception; what is far more common and widespread is the pro-

[8] Jour. Path. and Bact., 1905, x, 373.

duction of intimal thickening. How is this to be explained? I doubt if there be any more obvious example of compensation in the whole of pathology; degeneration of the one coat, the media, is accompanied or followed by overgrowth of the other, the intima; nay, more, as Dr. Klotz's example illustrates very beautifully, and as I confess I had not previously realized, in connection with the aorta there may[9] be a coincident and very pronounced hypertrophy of the adventitia (Fig. 1).

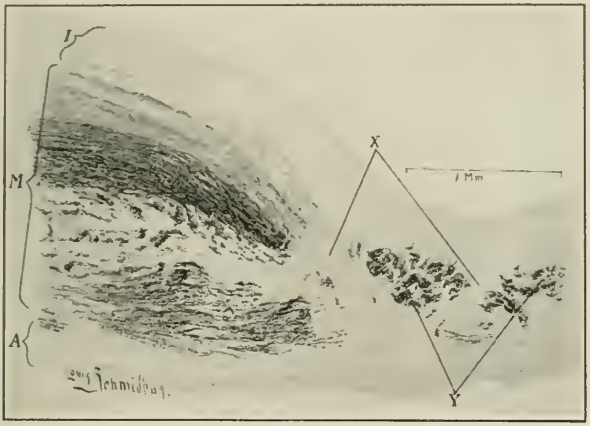

FIG. 1.—Section from a case of syphilitic mesaortitis stained by the elastic-tissue stain to demonstrate the extraordinarily degenerated and diseased appearance of the elastic tissue through syphilitic mesaortitis. It will be seen that at *X* the elastic fibres have completely disappeared; at *Y* they are reduced to irregular masses. There is some definite hypertrophy, both of the intima, *I*, and of the adventitia, *A*, but this has been insufficient to prevent a certain giving way of the wall. (From a specimen prepared by Dr. Klotz).

Certain points stand out very prominently regarding this intimal thickening. In the first place, it exhibits none of the ordinary earmarks of an inflammation. There is no formation of granulation tissue properly so-called; no new vessels; no small-cell infiltration, and, notwithstanding this, the new connective tissue formation attains to large proportions. The intima, it is true, is in itself non-vascular; it receives its nourishment by diffusion from the plasma within the aorta, as, indeed, would seem to be the case with the innermost layers of the media. But if we take any other non-vascular region, the cornea for example, and induce in that an inflammation by any of the accepted methods—by the injection of bacteria or the employment of caustic agents—within a short time new vessels make their way to the injured area from the periphery. Save as a late event, as already noted, we do not encounter this in the intimal thickening in syphilis

[9] Since this address was delivered, J. H. Wright and O. Richardson have also called attention to this thickening of the adventitia with extensive necrosis of the media. Boston Med. and Surg. Jour., 1909, clx, 539.

even when there is an active granulation tissue formation in the media. This is a very remarkable fact; the intimal overgrowth is essentially and purely hypertrophic. The internal elastic lamina constitutes as it were a barrier. We do not deal with an endarteritis, and Virchow was wrong in labelling it with this name; as again have been Thoma and other later writers (Fig. 2).

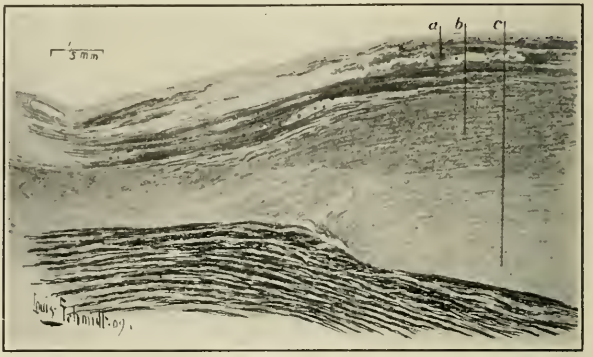

Fig. 2.—Section of an aorta from syphilitic mesaortitis with some narrowing of the media and great overgrowth of the intima. It will be observed that the layers of new tissue of the intima show a regular arrangement with absence of vessels and of small-cell infiltration. The outer layers, at *a*, in this case exhibited pronounced fatty change. The intermediate layer, *b*, took on a slight stain with the elastic tissue reagent, and under the high power this was seen to be due to the fine fibrils of what, from their arrangement, were young connective tissues, taking on the elastic tissue stain. Deeper down, at *c*, there was a complete absence of this reaction. The media, on the contrary, while narrowed, stained deeply with this stain. (From a specimen prepared by Dr. Klotz.)

But if an hypertrophy, how is it to be explained? We owe to Thoma,[10] in connection with ordinary arteriosclerosis, the first clear recognition that this is a compensatory hypertrophy—that in ordinary arteriosclerosis there is first a giving way of the media, and that the intima proliferates as a consequence. Thoma sought to explain this overgrowth as of nutritive origin, as due to alteration in the rate of blood flow at the region of bulging of the arterial wall. For myself, I must confess that I have never been able to grasp his explanation; what is more, I have never met anyone who has pretended to do so. Thirteen years ago, in an address before the New York Pathological Society, I afforded an explanation.[11] That also was not comprehended at the time. For example, Professor Mott[12] wholly mistook its drift. But I am more and more convinced that that explanation was rational, and, what is more, recent studies of a different nature have afforded a brilliant demonstration of its

[10] Virchow's Archiv, civ, and subsequent volumes.

[11] On the Relationship between Inflammation and Sundry Forms of Fibrosis, Middleton Goldsmith Lecture, Med. Rec., 1896, pp. 469 and 505; see also Discussion upon Arteriosclerosis, Brit. Med. Jour., December, 1906, ii.

[12] Allbutt's System of Medicine.

correctness. I laid down then that this regular development of layer after layer of new connective tissue was non-inflammatory, but was of the nature of *strain hypertrophy*—that just as increased pull or strain of the muscles upon their bony insertions leads in athletes to an increased development of the bony ridges, to a localized hypertrophy of bone, so within certain limits, increased tension or pull upon fibrous connective tissue favors its overgrowth, provided that adequate nourishment be afforded at the same time. It is but another example of the law, wholly misunderstood by Weigert, that increased functional activity within certain limits favors growth. I am glad to see that Professor Aschoff[13] explains the progressive increase in the elastic tissue of the aorta as being due to a like work hypertrophy.

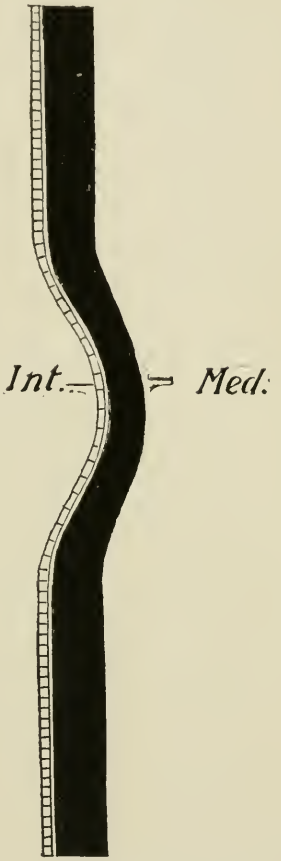

Fig. 3.—Schematic representation of the increased strain brought to bear upon the cells of the intima, *Int.*, when the media, *Med.*, undergoes a localized expansion through relative weakness.

I held, and so I still hold, that a moderate local giving way of the media under the pressure to which it is subjected by the blood within an artery causes an increased strain upon the overlying intima, which must undergo a certain amount of stretching, as it now becomes pressed outward to expand over the concavity afforded by the bulging of the media (Fig. 3). Herein is the stimulus to growth, and the growth continues, layer after layer, until not, as Thoma held, the concavity is accurately filled and the lumen restored to the status quo ante, but until the volume of the new tissue and the resistance that this affords to the mean distending force balances the loss sustained by the weakened media. In other words, when the resistance offered by the whole arterial wall at such a locality is equivalent to that of the rest of the aortic or arterial wall, then the strain is reduced and the process of hypertrophy becomes arrested. In these cases the youngest tissue is situated immediately beneath the endo-

[13] Beihefte zur med. Klinik, 1908, iv.

thelium, the oldest most remote and nearest to the deeper musculoelastic layer. Unlike what occurs in the ordinary form of arteriosclerosis, in syphilis this latter layer, so far as I have seen, shows comparatively little hypertrophy.

The convincing demonstration of the accuracy of this view has been afforded by one of Carrel's remarkable transplantation experiments. Carrel,[14] it will be remembered, removed a length of the cats carotid and replaced it by a length of vein from the same animal and of approximately the same caliber; under the increased blood pressure the vein exhibited dilatation, but left in situ for many weeks it presented at the end of the period a remarkable connective tissue hypertrophy of all its coats, so that its eventual thickness was distinctly greater than that of the artery at either end of the transplanted piece. The adequate and, in fact, as I freely admit, improved nutrition to which the vein is subjected, might by some be held to explain the hypertrophy of the intima, but at the same time the more that becomes sclerosed, the less becomes the nutrition of the deeper layers; but all the coats showed the connective tissue overgrowth. Add to this that cases in which increased nutrition, pure and simple, leads to hypertrophy are almost if not entirely wanting. I can bring to mind no single incontrovertible example. There is, I hold, no other satisfactory conclusion than that in this case it is the increased strain thrown upon the venous wall (coupled with adequate nutrition) that leads to the profound connective tissue hypertrophy.

When, on the other hand, the destruction of the media is more widespread and, we must presume, more acute, then, as in aneurysm formation, we encounter not intimal overgrowth, but the reverse. In other words, the expansion of the intima which has wholly lost its main support, the media, is now so great that we pass from the state of *strain* to that of *overstrain*, and now with the overstrain in place of hypertrophy we tend to get atrophy.

We thus arrive at the following conclusions regarding the effects of syphilis upon the aorta: (1) The primary disturbance is a granulomatous, inflammatory degeneration of the media. (2) This leads to a local giving way of the aorta. (3) If this be moderate it results in a strain hypertrophy of the intima and of the adventitia, with the development of a nodose intimal sclerosis. (4) If it be extreme, there results on the contrary an overstrain atrophy of the intima and aneurysm formation. (5) The intimal nodosities are here not of inflammatory type and are non-vascular, although, with the progresive laying down of layer upon layer of connective tissue on the more internal aspect of the intima, the earlier and deeper placed layers of new tissue gain less and less nourishment, and so are liable to exhibit fatty degeneration and necrosis. (6) These products of necrosis exert a chemiotactic influence upon the nearby

[14] Jour. Exp. Med., 1908, x, 130.

vessels of the medial granulation tissue, with, as a result, (*a*) a secondary and late entrance of new vessels into the early and deeply placed atheromatous area; (*b*) absorption of the necrotic products; (*c*) replacement by granulation tissue; (*d*) contraction of the granulation tissue; and (*e*) depression and scarring of the sclerotic nodules so characteristic of syphilitic sclerosis.

Can we apply these deductions to ordinary non-syphilitic nodose arteriosclerosis and to the Moenckeberg's type already noted? I think we can, or otherwise, and this is the crucial point, we can demonstrate that the ordinary arteriosclerosis of advancing life exhibits a primary giving way of the media, and we can do this both histologically and by experiment. First, as regards the histology, a most interesting study has been published recently by Professor Aschoff regarding the progressive changes that are undergone by the aortic media in the course of life. I will not go into all the details, but merely epitomize his main conclusions. In infancy the elastic laminæ of the media stand out sharply defined, well separated from each other by the muscle layers, which are well developed. There is, that is to say, sharp alternation of muscle and elastic layers. From childhood there is to be observed a slowly progressive increase in the elastic elements of the media. Not only do the individual lamellæ seen in cross-sections become thicker, but also they afford an increasing number of fine secondary filaments feathering off from these and crossing the muscle layer, so that now they are no longer sharply defined, but more ragged upon cross-section. This progressive increase attains its maximum at or about the age of thirty-five, and from now on for the next fifteen years the condition is relatively stationary. After fifty, according to Aschoff, there is to be observed a slowly progressive atrophy of the elastica. The media becomes obviously thinner and presumably weaker.

Independently, Dr. Klotz has been studying the same subject for some years and for the last twelve months Mr. Foster, under his direction, has examined a long series of aortas, from those of infancy up to those of advanced age. His work is on the eve of publication. Again, not to enter into details, I may say that these observers are wholly in agreement with Professor Aschoff's findings up to the age of thirty-five and in practical agreement up to that of fifty. But, as Klotz has already pointed out on more than one occasion, the subsequent degenerative process is one showing itself histologically not so much in connection with the elastic lamellæ as with the intervening muscle. This from the age of thirty-five onward begins to exhibit fatty degeneration, although it is only after fifty that, as a rule, this becomes at all marked. And now one or both of two processes may occur. Either, as shown by Klotz's admirable studies, the fatty gives place to a calcareous infiltration, fine calcareous granules appearing in the course of the muscle fibers—a sign of necrosis of these fibers—or they undergo complete absorption. Per-

haps most frequently both processes are to be made out. As a result the intervening spaces between the elastic lamellæ become smaller and smaller, and often there is afforded the appearance of thick lamellæ due to the approximation of two or more, which can be resolved under higher magnification. With this, whereas in the young aorta the lamellæ are wavy, due to the contraction of the intervening muscles fibers, now they are relatively straight. It is this muscular atrophy rather than atrophy of the elastic tissue that thus would seem to be the main cause of the thinning of the aortic media. I do not in the least mean to say that the elastica is unaffected in this process. At times the calcareous degeneration affects it also. Everything indicates that coincidently it *loses its elasticity*, becomes more rigid and more expanded as the result of internal pressure; it is unapt to return to its former length.

It is further to be noted that the calcification is unequally distributed. Frequently in Dr. Klotz's specimens of the Moenckeberg type it is most marked, and the thinning of the media is coincidently most pronounced, in the walls of the shallow sacculations already noted. The presence of the sacculations show that here are the regions of greatest weakening. But it is frequent to find that when there is marked nodose thickening of the intima then also the underlying aortic media is thinned. This, it is true, is most evident not in the aorta, but in its branches—in the carotids, the splenic artery, the coronaries, etc. In all of these there may be an extraordinary overdevelopment of the fibroid intima and an equally striking expansion and giving way of the media. All must be familiar with specimens showing this condition. On the other hand, it has to be admitted freely that there may be well-marked intimal nodes of the aorta when the underlying media shows no recognizable thinning. Apart from possible exceptions to be subsequently noted there is an explanation to be afforded for this state of affairs—an explanation along the lines of Thoma's well-known observations upon the disappearance of the projecting intimal nodosities when the aorta is filled with melted lard under pressure. This is most rapidly afforded by the accompanying diagram (Fig. 4), which represents a length of aortic wall, in which the midportion has become weaker than the rest. This portion under internal pressure will be liable to give way, and, while thus bulging, will of necessity exhibit a definite grade of thinning, with compensation or strain overgrowth of the intima tending to fill the concavity. If, as occurs postmortem, the internal pressure be removed, then the natural elasticity of this midportion of the wall, which has not been wholly lost, will result in a disappearance of the concavity, the wall now appearing to be of normal thickness of media with a projecting nodosity of the intima. The same will naturally appear in sections prepared under the same conditions.

Let us now turn to the experimental evidence. The active dis-

cussion as to whether the lesions caused by adrenalin, barium chloride, nicotine, and other drugs in the rabbit are to be regarded as in any way equivalent to arteriosclerosis as seen in man, will be familiar to all of you, and many of you may have come to very definite conclusions upon the matter. Certainly these lesions do not resemble the ordinary nodose arteriosclerosis, but, as all must admit, the constant feature is a giving way of the middle coat, and this to such an extent that at times definite aneurysmal pouches are produced, at others and frequently those more shallow sacculations, scarce to be designated as aneurysms proper. As Klotz and K. Ziegler[15] independently pointed out, the appearance is identical with that noted by Moenckeberg in his type of arterial change. Medial degeneration, and giving way is the dominant lesion of adrenalin disturbances. I took occasion to point out at the discussion on Arteriosclerosis, at the Toronto meeting of the British Medical Association in 1907, that the reason for the lack of intimal overgrowth might well be the acute development of the medial degeneration, resulting in rapid expansion of the intimal sheath and overstrain rather than strain, and I suggested that less extreme degeneration would be found to result in a more typical arteriosclerosis with intimal hypertrophy. Since then Dr. Klotz, by a different method, has demonstrated the truth of this surmise on my part. He has set up arterial disease in the rabbit without the use of any drug whatsoever, and that by merely suspending a (previously) healthy well-grown buck for three minutes daily by the hind legs—head downward. This naturally caused *pro tem.* an unaccustomed blood pressure in the thoracic, cervical, and cranial vessels, a reduced pressure in the abdominal and other arteries of the hinder end of the body. After this is continued for one hundred to one hundred and twenty days the heart is found greatly hypertrophied, the arch and the thoracic aorta are dilated, with very distinct thinning of their walls; the abdominal aorta, on the other hand, is unaffected; the carotids are

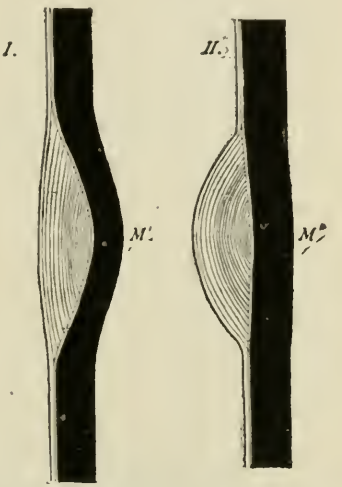

FIG. 4.—*I*, media weakened at *M'* with overgrowth of intima filling in the depression. *II*, with postmortem rigor and contraction of the muscles of the media and removal of the blood pressure from within, the stretched media at *M''* contracts; the intimal thickening thus projects into the arterial lumen.

[15] Ziegler's Beiträge, 1905, xxxviii, 229.

larger than normal, and—this is the important point—they exhibit a typical irregular intimal sclerosis. There is pronounced connective tissue thickening of that intima.

These results can only be explained in one way, namely, as the effects of the daily temporary suddenly increased blood pressure brought to bear upon the vessels of the anterior half of the body.

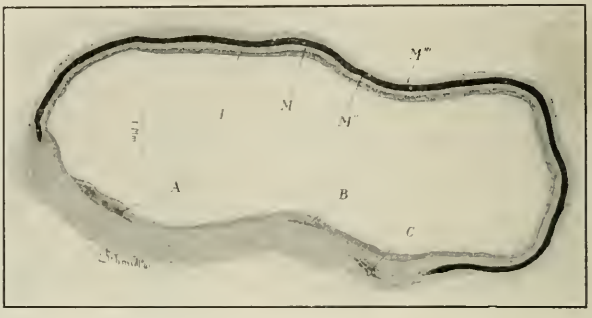

FIG. 5.—Section through the aorta of a rabbit that had been suspended for three minutes daily by the hind legs, for one hundred and thirty days, to demonstrate dilatation and relative absence of intimal proliferation. Around the greater part of the organ there is pronounced thinning of the media with calcareous degeneration of the middle layer (M''). This dilatation and thinning are less marked round about one-third of the organ, and here there is a slight grade of patchy hypertrophy of the intima at *A* and *B*. This hypertrophy here particularly involves the deeper musculo-elastic layer. At *C* there is beginning calcification of the media. (From a specimen stained with Sudan III and silver nitrate by Dr. Klotz.)

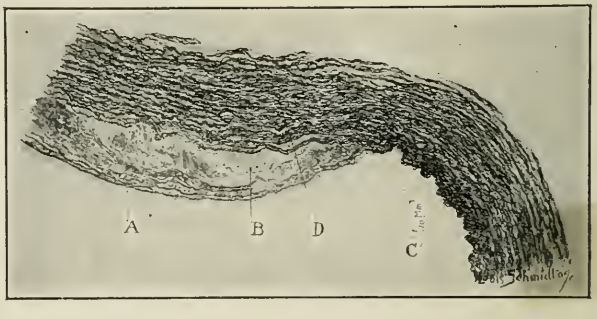

FIG. 6.—Section from the carotid of the same rabbit (higher magnification) exhibiting at *A* well marked nodular sclerosis of the intima. At *B* the deeper layers of the hypertrophied intima are already showing degenerative changes. *C*, normal intima. *D*, media. (From a specimen by Dr. Klotz).

That has resulted in diffuse dilatation of the larger vessel, the aorta, without intimal overgrowth, and in pronounced intimal overgrowth of the smaller vessels, the carotids. Here for the first time, I think, we have an absolute demonstration that one and the same cause is responsible for the two orders of conditions, the demonstration that the arterial thinning of the Moenckeberg type and the intimal thickening of ordinary (senile) arteriosclerosis are diverse mani-

festations of a common process. The greater circumference and the relatively slighter resisting or accommodative force of the aortic wall due, I may say, to its anatomical structure, has led to overstrain and dilatation, and as a consequence absence of intimal proliferation; the lesser circumference of the carotids and their proportionately greater resisting strength has been followed by a relatively slighter dilatation, tending to be localized to particular areas, and here as a result there has been strain, not overstrain, of the intima and a resultant connective tissue hypertrophy. I must add in justice that another Canadian worker, Dr. Harvey, of Toronto, continuing work begun under Professor J. J. MacKenzie, has independently, in the laboratory of Professor Dixon, of Cambridge, England, brought about the same diffuse dilatation of the aorta and cardiac hypertrophy by temporal digital compression of the rabbit's aorta extending over long periods. He does not, however, in his communication call attention to the coincident sclerotic change in the smaller vessels.[16]

Here then we arrive at a common order of events in the two great types of aortic sclerosis—the syphilitic and the ordinary nodose forms. In both we find weakening of the media as the primary disturbance; in both, if this be above a certain grade, there is pronounced giving way without intimal compensation; in both, if below this grade, a strain hypertrophy of the intima is manifested, leading to the production of intimal thickening. Only, in syphilis the giving way of the media is the more extreme, and so we more frequently encounter advanced aneurysmal formations as contrasted with the shallower sacculations of the Moenckeberg type, and in this, also, occurring as a rule earlier in life, there is a more exuberant regenerative power exhibited by the intima.

This view, it will be seen, demands that strain or pressure is the cause of the intimal overgrowth in both orders of disturbance; it demands also that the giving way of the media in the first place is due to a local weakening sufficient to render the arterial wall unable to resist the outward thrust of the column of blood. Nay, more, Klotz's experiment shows that increased intravascular pressure alone may be the cause of the medial degeneration and weakening in the first place, of the giving way of the arterial wall in the second, and of the intimal hypertrophy in the third. We are brought thus face to face with the time honored problem—to what extent is heightened arterial pressure—or, as Sir Clifford Allbutt would seek to have us call it, *hyperpiesis*—a factor in arteriosclerosis?

From the data already afforded, it is evidently not an essential factor in the syphilitic cases. Undoubtedly when present it may

[16] Personal communication from Professor MacKenzie and Sir Clifford Allbutt. I was of the impression that Harvey's results had been published, but can find no reference in the Index Medicus or elsewhere.

accelerate the process in these, but infection is here the primary agent in causing the atrophy and absorption of the media.

As regards senile arteriosclerosis, the observations of Aschoff, Klotz, and Foster show that after fifty there is to be determined a physiological atrophy of the media; obviously the degeneration of the muscular layers and the further indications of change in the elastic lamellæ indicate a weakened media. It is not necessary in these cases to demand an increased blood pressure. The normal blood pressure must eventually be adequate to cause giving way of the media with or without consequent intimal hypertrophy.

Granting this for senile cases, then for the presenile arteriosclerosis, we are, from Klotz's experiment, forced to recognize that high intra-arterial pressure, and this alone, is sufficient to bring about a nodose arteriosclerosis with all its possible dangers. That experiment affords the experimental proof of the danger of heightened blood pressure, which from practical experience clinicians have so learnt to fear—and this in a perfectly clear manner, with no ambiguities or possibilities of lack of appreciation of other factors. High pressure, and high pressure only, can be invoked to explain the results which I have demonstrated to you.

We then reach the following conclusions, namely: That *like results may ensue when, on the one hand, the artery has undergone weakening and the blood pressure is normal, and when, on the other hand, the artery has no preliminary degeneration of its media and the blood pressure is above the normal.* What is of importance is that, as shown by this experiment, it is not necessary to have a blood pressure which is permanently above the normal; an acute rise for a short period frequently repeated will bring about the result.

I would greatly like to enter into a discussion of the mechanism of increased arterial pressure in relationship to peripheral arterial sclerosis, but have already taken so much of your time that I can venture at most to suggest the headings. I would call your attention to the fact that it is the arterioles through the body and their tonus that determine the existence of the normal blood pressure; that heightened blood pressure in the larger vessels means increased contraction of the arterioles of important areas; that thus to changes in the arterioles and smaller arteries we must eventually look for the explanation of hyperpiesis; that it is by contraction of the arterioles that adrenalin, digitalin, barium chloride and the other raisers of blood pressure produce their effects; that in this contraction we have the elements necessary for the establishment of a vicious cycle; that, as shown by Leonard Hill, the arteries react normally to increased internal pressure not by dilatation, but by contraction; that thus the higher the blood pressure the more contracted do the smaller arteries tend to become; and that, if the heart responds to the increased work thrown upon it, the higher still becomes the blood pressure. Along these lines it will be seen that rise of blood pressure tends in the

first place to throw increased work upon the musculature of the smaller arteries, tends, that is, to cause their hypertrophy; if the rise is progressive, then this hypertrophy gives place to exhaustion, muscular degeneration, and dilatation of the arteries (and arterioles). As a result strain hypertrophy of the intima shows itself with thickening, and it may also be of the adventitia, resulting in chronic periarteritis. And now, with continued degenertion of the medial muscle in those muscular arteries, fibrosis of the media may also show itself. I would thus regard muscular hypertrophy of the arteries and fibrosis of the different coats as different stages in one and the same process. Whether these peripheral changes are the more marked, or the central, depends, I would suggest, upon the relative resisting power of the elastic and the muscular arteries of the individual respectively.

Lastly, it may be asked, have I included here all the forms of arteriosclerosis and intimal hypertrophy? I certainly believe that I have referred to the more important, though I have not exhausted the forms. There is, for example, a form which Aschoff would term functional sclerosis, seen in the ovaries and uterus, in the ovaries after menstruation and ovulation, in the uterus after menstruation and placentation. The sclerotic change here may be extreme, and again is of a hypertrophic and regenerative type—so much so that in the uterus there may be encountered, as shown by Sohma,[17] the development of what is practically a new artery within the old, with endothelial, intimal, muscular, and even what corresponds to an adventitial coat. Here, I would hazard, the same prinicple is at work. The extreme congestion and dilatation of these arteries in menstruation and placentation leads to an overstrain of their walls with consequent overexpansion and degenerative weakening, to such an extent that at the conclusions of these physiological events, when there is no longer a call for dilatation of the arteries, they still remain expanded. Nor is there any adequate mechanism on the proximal side (toward the aorta) to cut off fully the blood supply. There is still, therefore, a certain amount of strain exerted upon the arterial walls from within, and this leads now to secondary overgrowth.

Secondly, Dr. Klotz has shown experimentally, and Saltykow[18] has confirmed, that certain bacterial toxins, *e.g.*, the diphtheritic, lead to medial degeneration, others, like typhoid toxins, have no recognizable effect upon this coat, but induce a primary intimal proliferation. I freely accept the existence of such primary intimal overgrowth. I have to confess, however, that I have some doubts as to its frequency as a cause of extensive arteriosclerosis in man. When it is at all well developed, then I would point out, the superficial intimal overgrowth

[17] Archiv f. Gynäk u. Obst., 1908, lxxxiv, 84.
[18] Ziegler's Beiträge, 1908, xlii, 187.

cuts off the due nutrition of the musculo-elastic layer, and, indeed, of the innermost layer of the media; degeneration and weakening of these is the natural result, and there is established a vicious circle; the greater the intimal thickening the greater becomes the weakening of the lower intimal layer, and so of the arterial wall; nor is the full picture of arteriosclerosis attained until both events are present.

The same picture is afforded in the arterioles. My former colleague, Dr. Duval,[19] now professor of pathology in Tulane University, then pathologist to the Montreal General Hospital, published certain most interesting studies upon the effects of attenuated glanders bacilli upon the vascular system of the rabbit. In these he demonstrated most clearly that these bacilli induce an active proliferation of the endothelium of the smaller arteries, and he gives a figure of a later stage in which a well-marked sclerotic node of the intima has associated with it a bulging and thinning of the media. If these two pictures represent successive phases of one process—and of that I am not wholly convinced—it must still be admitted that medial weakening, whether by the direct action of the glanders toxin or by arrest of nourishment of the media through the intimal hypertrophy, is an essential part of the process. Endothelial proliferation alone does not constitute arteriosclerosis.

Lastly, and perhaps of the greatest present importance, there is the consideration of the part played by the deeper or musculo-elastic layer of the aortic intima in the arteriosclerotic process. Following the lead of Jores,[20] the majority of the recent German writers on the subject lay stress upon the degeneration and hypertrophic processes found in this as being the fundamental change in arteriosclerosis. Here I have an objection to make of the same order as my criticism of the use of the term atherosclerosis, namely, that aortic disturbances are dwelt upon to the exclusion of the concomitant changes in the arteries of muscular type. It is only in the larger branches of the aorta—those of the intermediate type—that this layer continues to be a factor of any importance. As we pass into the smaller arteries its development becomes so slight that it cannot possibly be a factor of the first order.

In the aorta its fully developed structure—of longitudinally arranged plain muscle fibers and abundant elastic fibrillæ—renders it akin to the media. At most, the longitudinal arrangement of its elements suggests that its function is to oppose elongation rather than dilatation of the vessels in which it is present. Simple hypertrophy must connote increased strength of the arterial wall; degeneration must signify weakening of the wall as a whole. Now, significantly this layer is curiously apt to exhibit degeneration. Why this should be so is not wholly understood. I would suggest that its position relative to sources of nutrition is largely at fault. The

[19] Jour. Exp. Med., 1907, ix, 241.

[20] Ziegler's Beiträge, 1902, xxxii, 146.

internal elastic lamellæ of the media largely cut it off from nutrition from without; the plasma from the interior of the vessel has to pass through and be acted upon by the more superficial layers of the intima before reaching it. It has no independent source of nutrition, and more particularly if the superficial layers of the intima have undergone thickening, the factors favoring degeneration are clearly greatly increased. The striking picture that this layer presents in the earlier stages of degeneration—the swollen condition of its cell elements, the œdema of its interstitial substances, the diffuse soapy and fatty change that it undergoes—all, I am inclined to lay down, tend to arrest the attention and tend thus to a neglect of the concomitant medial and superficial intimal changes. And this point of view is supported by the existence of degenerative changes in the layer unaccompanied by any other vascular disturbance. In cases of burns and of acute infections, and that even in the young, it is common to meet with fine fatty streaks in the aorta. And these streaks indicate a fatty degeneration of, in the main, this musculo-elastic layer. It is natural to imagine that here we have the first stage in the arteriosclerotic process. I am, however, convinced that the two have no necessary association. I would ask anyone who doubts this to follow the example of Drs. J. McCrae and Klotz in our service at the Royal Victoria Hospital, and make a note upon the postmortem report every time that these fatty streaks are present. It will surprise him to find how common they are; how frequently they are present in the quite young; how little obvious relationship they have to the arteriosclerotic state.

I do not deny that a weakening of the musculo-elastic layer weakens the whole arterial wall, and thus may be a factor favoring subsequent intimal hypertrophy. I only urge that this is not nearly so frequent or so important as is a weakening of the media.

Thus we revert to our main conclusions that the dominant primary event in the arteriosclerotic process—syphilitic, senile, or functional—is a localized or it may be a diffuse weakening of the arterial wall and especially of the media. This induces increased strain upon the remaining coats; and if this be not excessive, that strain leads more especially to connective tissue overgrowth and the development of the characteristic lesions of arteriosclerosis.

In the foregoing remarks I have repeatedly referred to the work accomplished in our Montreal laboratories, and more particularly to the studies of Dr. Klotz. When the chief of a laboratory quotes the work of those under him, it is usual to infer that the work is his under another name—that he has inspired the researches. In justice to my colleague, I may say that in this instance this most decidedly is not the case. I have quoted Dr. Klotz's work so frequently because I have followed it with very deep interest and know intimately its progressive developments, and further because

each step has, to my mind, confirmed and expanded the conclusions I reached long years ago regarding the essential nature of the arteriosclerotic progress. I appreciate too highly the originality of Dr. Klotz to claim more than having been fortunate in suggesting to him that line of inquiry into the nature of calcification which he carried out with so much distinction and of which much of his later work is, as it were, a natural evolution. I should be glad to regard this address, and to have this address regarded, essentially as a vehicle for bringing forward in a connected manner the observations of my colleague and my interpretation of them, which must not necessarily be thought to be his interpretation.

SOME CONDITIONS AFFECTING THE DISCHARGE OF FOOD FROM THE STOMACH.

By Carl A. Hedblom,

and

Walter B. Cannon, M.D.,

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The purpose of this investigation was to determine as accurately as possible, under otherwise normal conditions, the influence of some of the commoner physical, chemical, and biological factors on the movements of the stomach and on the rate at which it discharges food into the intestine. The method employed was that used by one of us in studying the passage of different foodstuffs from the stomach.[1] The food used, except when otherwise specified, was 25 c.c. of mashed potato, with which was mixed 5 grams of subnitrate of bismuth. The consistency, which depends upon the amount of water added, was always as nearly uniform as could be judged by the eye and by manipulation. It will appear, however, from observations described below, that within the limits involved variations of consistency have no appreciable effect on the rate of gastric discharge of this food. Full-grown normal cats, deprived of food for twenty-four hours previous to the experiment, served for observation. In all the experiments, except those with fluid food, the animals were placed on the holder and fed from a spoon with no special difficulty. At regular intervals after feeding, observations were made by means of a fluorescent screen illuminated by the x-rays; and the dark shadows made by the food were traced in outline on transparent paper laid over the screen. The aggregate

[1] Cannon, Amer. Jour. Med. Phys., 1904, xii, 387.

length of these shadows measured the relative amount of food in the intestine at the different times of observation.

The method has been demonstrated to be above serious criticism.[2] During the first two hours, when the aggregate length of the food-masses in the intestine is most significant as an indication of the rate of gastric discharge, the intestinal content is usually not great, and can therefore be traced with only slight liability to error.

In establishing the normal rate of discharge as a basis for comparisons, the possibility of subjective differences, the personal equation, is involved. That Magnus[3] has recently used the method above described, with no essential variations from the original results, indicates that the personal equation need not be great. In order to estimate the extent of a possible deviation in our work, however, the original observations on mashed potato were repeated by one of us (Hedblom). The two tables which follow show a close agreement in the results independently obtained.

Hours after feeding	½	1	2	3	4
Aggregate length, in cm., of food masses in small intestine (Cannon)	9	30	43.5	28	22
	10	39	53.5	28.5	26
	9.5	22	36	36.5	30
	9	30.5	39	8	7
Average (4 cases)	9.5	30.5	43	25	21

Hours after feeding	½	1	2	3	4
Aggregate length, in cm., of food masses in small intestine (Hedblom)	10	32	42	31	31
	7	24	43	34	26
	11	29	43	33	23
	13	27	38	33	30
Average (4 cases)	10	28	41.5	32.5	27.5

After thus determining that a resonably close agreement existed between the results secured by the use of the method by different persons, we undertook a study of the effects on the rate of gastric discharge, of variations in the consistency of the food, the presence of gas in the stomach, extremes of temperature, different degrees of acidity, massage, irritation of the intestines, and fatigue.

The Influence of Consistency. A number of investigators have studied the effect of varying the water content of the food, as well as the rate of discharge of various liquids. In some of these researches animals with duodenal fistulas were used; in others the stomach tube was relied upon to determine the rate of discharge. Moritz,[4] who used the fistula method, found that water begins to leave the dog's stomach immediately after being swallowed, and that the stomach is almost emptied in fifteen to thirty minutes. In one case milk was discharged with similar rapidity; in another case, when the milk coagulated, the stomach was not emptied for

[2] Cannon, loc. cit., p. 391.

[3] Archiv f. die ges. Phys., 1908, cxxii, 210.

[4] Ztschr. f. Biol., 1901, xlii, 572

one and a half hours. These results Moritz confirmed on man. He states that water leaves the stomach of man as quickly as it leaves the stomach of the lower animals; but that beer, milk, bouillon, and thin soup remain longer than water. Even with the least fluid of the substances he used—a thick soup—the stomach was practically empty in an hour. These observations, that liquids leave the stomach rapidly, are confirmed by the work of Hirsch,[5] Penzoldt,[6] Schüle,[7] and Roux and Balthazard.[8] These studies, however, are concerned almost entirely with liquids and not with the discharge of a certain food in various consistencies. To obtain information regarding the effects of varying consistency and other mechanical factors on the gastric discharge, observations were made on more or less viscous samples of potato, on hard particles mixed with the food, and on coarse graham bread.

1. *The Influence of Variations in the Viscosity of the Food.*—For this study potato was used. It was baked in order to drive off most of the water. Two series of observations were made. In the first series no water was added; the potato when mixed with bismuth subnitrate and ready for feeding was very thick and doughy. In the second series water was added until the mixture was of the consistency of thin gruel. The volume fed in all cases was 25 c.c. The results with these extremes of consistency should be compared with the results when potato of the standard consistency (intermediate between the extremes) is fed. The following were the figures obtained.

Potato, thick doughy consistency.

Hours after feeding	½	1	2
Aggregate length, in cm., of food masses in small intestine	5	11	28
	16	28	49
	17	28	41
	13	40	54
	5	17	35
Average (5 cases)	11	25	41.5

Potato, thin gruelly consistency.

Hours after feeding	½	1	2
Aggregate length, in cm., of food masses in small intestine	14	20	35
	9	17	31
	15	22	40
	17	20	40
	18	37	42
Average (5 cases)	14.5	23	37.5

As the curves in Fig. 1 show graphically, the rates of discharge of the same kind of food with fairly widely varying consistencies are nearly the same. Indeed, the rates of discharge do not differ among themselves enough to permit any noteworthy significance to be attributed to the differences in consistency.

[5] Centralbl. f. klin. Med., 1893, xiv, 75.
[6] Deut. Archiv f. klin. Med., 1893. li, 567.
[7] Ztschr. f. klin. Med., 1896, xxix, 49.
[8] Arch. de phys., 1898, xxx, 90.

Further evidence that consistency alone is not a prominent factor in determining the rate of gastric discharge is furnished by Moritz,[9] who observed wide differences in this rate when various fluids of practically the same consistency were given. Thus, water passed from the stomach much faster than beer, and considerably faster than bouillon.[10] On the whole, the conclusion seems warranted that while liquids leave the stomach very rapidly, more or less dilution of a food predominantly carbohydrate has but slight effect in modifying the rate of gastric discharge.

Protein food leaves the stomach more slowly than carbohydrate. The explanation for this phenomenon is that the union of protein with the acid of the gastric juice delays the appearance of a sufficiently strong acid reaction of the contents near the pylorus and thereby delays the first opening of the pylorus.[11] If protein food is diluted with water there is evidently, in a given amount of the food, less protein to unite with the acid than would be present if the same amount were given undiluted.

Lean beef of standard consistency gives the following average figures:

Hours after feeding	½	1	2	3	4
Cm. in small intestine	1.5	2.5	16	22	24.5

When lean beef, shredded and mixed with water to a thin gruelly consistency, was fed, the following figures resulted:

Hours after feeding	½	1	2	3	4
Aggregate length, in cm., of food masses in the small intestine	0	11	20	23	29
	7	9	30	30	28
	0	20	32	33	32
	5	8	22	32	30
	6	22	35	38	37
	0	8	29	39	32
	8	21	33	38	36
Average (7 cases)	3.5	14	28.5	33	32

A comparison of the figures in the foregoing tables and the curves in Fig. 2 shows that the dilution of the protein food, and the reduction thereby of the material uniting with the acid of the gastric juice, tends toward a more rapid discharge of the protein from the stomach.

Schüle[12] states in connection with his investigation of the discharge of liquids that fluid foods seem to increase the motility of the stomach. An inference to the same effect can be drawn from

[9] Loc. cit., p. 590.

[10] The slow emergence of beer and bouillon, compared with water, can be explained by the effect of these fluids in stimulating the flow of gastric juice (see Pawlow, The Work of the Digestive Glands, London, 1902, p. 138 et seq.) and the resultant closure of the pylorus through the acid reflex from the duodenum.

[11] Cannon, Amer. Jour. Phys., 1907, xx, 292.

[12] Ztsch. f. klin. Med., 1896, xxix, 73.

Moritz's statement: "Solid food causes a checking of gastric peristalsis, which overcomes the stimulating effect otherwise exerted upon it by water." We have never noted any significant changes in peristalsis due to variations in consistency.

2. *The Influence of Hard Particles in the Food.* There are numerous clinical reports of foreign bodies swallowed accidentally or wilfully by children and demented persons, and in some instances by normal adults. Eckold[13] reviews more than seventy such cases in which objects of the most varied size and character were swallowed. When they remained long in the stomach more or less serious derangement of gastric functions resulted, followed by malnutrition and in a large percentage, by death. The stomach may evidently function, though imperfectly, while containing a foreign body. But these cases represent decidedly abnormal conditions, and do not throw much light on conditions that may be classed as more or less common, such as the presence in the stomach of bone, seeds, and poorly masticated food.

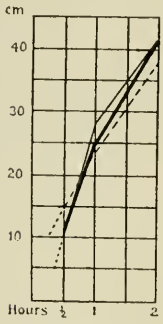

FIG. 1

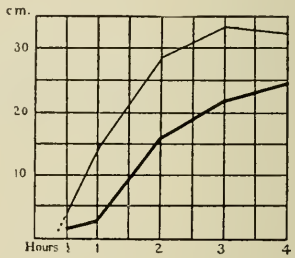

FIG. 2

FIG. 1.—These and the following curves show the average aggregate length of the food masses in the small intestine at designated intervals after feeding. The light continuous line is the curve for potato of standard consistency; the heavy continuous line, for thick doughy consistency; the broken line, for thin gruelly consistency.

FIG. 2.—The heavy line is the curve for lean beef of standard consistency, the light line that for lean beef of thin gruelly consistency.

Few observations as to the relation between hard food masses and gastric discharge have been reported. Schüle[14] states that coarse food remains in the stomach longer than food of smoother consistency. Moritz[15] has described experiments on a dog with a duodenal fistula, in which finely chopped sausage began to leave the stomach in forty-five minutes, whereas coarse unchopped sausage did not begin to leave for two hours, and then appeared at the fistula in a "soaked slippery condition." Cannon[16] reported in his

[13] Inaugural Dissertation, Greifswald, 1896.

[14] Loc. cit., p. 75.

[15] Verhandl. der deut. Naturforscher und Aerzte, 1893, p. 25.

[16] Amer. Jour. of Phys., 1898, i, 359.

first paper on the stomach that hard particles repeatedly pushed up to the pylorus checked the outgo of food from the stomach. The method used in the present investigation permits a more careful testing of this conclusion.

As hard particles, small irregular pieces of starch paste were used. Ten per cent. starch paste, mixed with subnitrate of bismuth, was cut into cubes and set aside to dry. From fifteen to twenty of these dried cubes, 1 to 5 mm. in dimension, were given mixed with the standard potato. In the first four cases reported below the the particles, which had dried for two weeks, were very hard. In the other cases the particles dried only two to four days.

Hours after feeding	½	1	2
Aggregate length, in cm., of the food masses in the small intestine[17]	4	5	14
	0	3	9
	2	5	7
	0	0	0
	7	13	23
	0	8	22
	0	8	25
	4	13	20
	2	16	21
	1	13	23
Average (10 cases)	2	8.5	16.5

In Fig. 3 the normal discharge is compared graphically with the discharge when the same food, with hard particles added, is fed. As the figures and the curves show, there is a marked retardation of the outgo of food from the stomach when hard particles are present. In the first four cases the outgo was clearly slower than in the others, as if the harder particles had had an inhibitory effect until softened. The checking action of the softer particles, however, shows likewise the working of a mechanical factor in modifying the normal mechanism at the pylorus.

The hard particles with a large bismuth content could be seen on the fluorescent screen as small black shadows against the less dark background of the shadow of the stomach. The to-and-fro oscillations of the small masses in the antrum already described[18] were repeatedly observed. There is a discrepancy between the immobility of these particles in the cardiac end of the stomach, as described in the earlier paper, and Beaumont's report that food circulates down along the greater and up along the lesser curvature. It is conceivable that the difference of observation might be due to a difference in the consistency of the food—that circulation might occur in a fluid gastric content, but not in a semisolid mass.

Starch paste of the consistency of thin gruel, with bismuth sub-

[17] In several cases, notably Case 4, there was an accumulation of gas in the stomach. The presence of the gas tended still farther to check the rate of the discharge.

[18] See Cannon, Amer. Jour. of Phys., 1908, i, 369.

nitrate added, was given. Several (3 or 4) of the hardened starch particles were then swallowed by the animal. In all the experiments the particles lay along the lesser curvature, until gradually carried toward the pylorus. There was no evidence of a circulation of the gastric contents such as Beaumont described.

3. *The Influence of Coarse Graham Bread.* There is evidence that food containing spicules of bran, such as coarse graham bread, may stimulate peristalsis in the large bowel and thus prevent constipation. It was of interest to determine whether gastric peristalsis and the discharge from the stomach are affected by food of this character. There are statements in the literature that no appreciable difference exists between whole meal and white bread in the duration of their stay in the stomach. Penzoldt[19] remarks, however, that light bread remains in the stomach as long as black bread and longer than bread made of coarse meal.

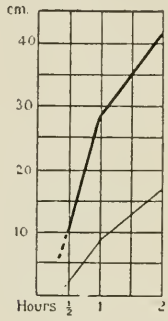

FIG. 3

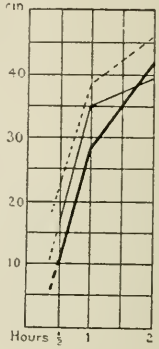

FIG. 4

FIG. 3.—The heavy line is the normal curve for potato; the light line, the curve when hard particles are present in the food (10 cases).

FIG. 4.—The heavy line is the normal curve for potato, the light line that for white bread (7 cases), the broken line that for graham bread (11 cases).

In the observations on the rate of discharge of bread, common white bread obtained at a bakery was used as a control instead of potato. The bread was prepared, however, just as in the observations with potato, and the same proportion of bismuth subnitrate was added. The following are the figures for white bread:

Hours after feeding	½	1	2
Aggregate length, in cm., of the food masses in the small intestine	10	30	41
	26	40	33
	15	33	30
	20	35	46
	19	42	41
	10	30	36
	16	36	49
Average (7 cases)	16.5	35	39.5

[19] Deut. Archiv. f. klin. Med., 1893, li, 560.

The graham bread fed in the first eight of the following cases was not so coarse as that fed in the last three cases:

Hours after feeding	½	1	2
Aggregate length, in cm., of the food masses in the small intestine	23	41	48
	30	45	49
	35	42	33
	24	47	48
	16	45	49
	25	24	46
	25	37	41
	19	40	50
	10	33	51
	16	25	44
	25	34	45
Average (11 cases)	22.5	38	46

A graphic comparison of these results with the results when potato is fed is shown in Fig. 4. The curves and the tables reveal a slightly slower rate of discharge for white bread than for potato, and a more rapid discharge for graham bread than for either of the other foods.

In summarizing the effects of the consistency of food on its discharge from the stomach, it may be fairly stated that within limits more or less water added to carbohydrate food does not change the rate of outgo, though dilution hastens the outgo when protein is fed; that hard particles in the food distinctly hinder the discharge from the stomach; and that coarse branny food is forced through the pylorus at a slightly more rapid rate than are similar foods finer in texture.

The Influence of Gas in the Stomach. The common use of carbonated water has led to attention being paid to the effects of carbon dioxide on the functions of the stomach. There is fair unanimity of testimony that this gas stimulates both secretion of hydrochloric acid and gastric peristalsis.[20] That the presence of a body of gas in the stomach might affect the exit of food has apparently not been much considered. Yet with the *x*-rays gastric peristalsis may be seen moving over an accumulation of gas without either churning the gastric contents or propelling them onward. The gas acts as a shield, keeping the walls of the stomach away from the food. We desired to learn what might be the effect of a considerable amount of gas in the stomach on the discharge.

The animals were fed the standard amount of food, and then air was blown into the stomach. In the first experiments the volume of air introduced was measured, but this procedure proved to be of little use, for eructations soon changed the original quantity. In all but the first four of the following cases the air was introduced

[20] See Jaworski, Ztschr. f. Biol., 1883, xix, 443; 1884, xx, 234; Gillespie, Deut. med. Woch., 1887, p. 836; Penzoldt, Deut. Archiv f. klin. Med., 1903, p. 567.

while the animal was under x-ray observation; in these latter cases the distention of the stomach walls, which could be clearly seen, could be easily regulated. In a few instances eructations nearly emptied the stomach of air during the first hour; more air was then introduced until approximately the original volume was restored.

The following results were obtained:

Hours after feeding	½	1	2	3
Aggregate length, in cm., of the food masses in the small intestine	0	6	29	37
	9	34	44	44
	13	27	33	35
	14	24	34	40
	0	25	30	27
	0	30	33	24
	0	17	13	38
	0	17	31	33
	0	0	33	40
	14	26	36	38
	0	10	32	32
	3	6	22	29
	0	11	27	35
	0	10	24	32
Average (14 cases)	4	17.5	30	34.5

These average figures are compared with the average figures for normal conditions in Fig. 5. Examination of the table shows that there is, as was to be expected, a wide variation in the effects produced by the presence of gas in the stomach. In few cases, however, is there any effect except a retardation of the discharge into

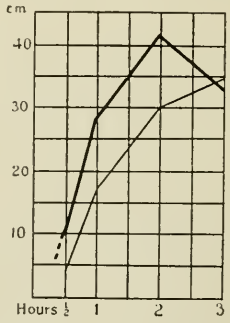

Fig. 5.—The heavy line is the normal curve for potato, the light line that for potato when gas is present in the stomach.

the intestine. This result has been noted repeatedly in other instances in which gas appeared in the stomach spontaneously. Thus, in one case in which fibrin was fed and in which the peristaltic waves could be clearly seen passing over the gas in the stomach the discharge was as follows:

Hours after feeding	½	1	2	3	4	5
Cm. fibrin, when gas present	0	0	0	10	17	22
Cm. fibrin, average of 4 normal cases	4	8	21	29.5	32.5	32

Such cases of spontaneous accumulation of gas seemed to be associated with gastric atony and enfeebled peristalsis. When the air was experimentally introduced, however, peristalsis, when observed, was normal in rate and intensity.

With peristalsis normal, how may the retardation of the discharge from the stomach, noted in the above experiments, be explained? That the distention of the gastric walls prevented them from exerting a direct propelling action on the food mass was distinctly to be seen. In a number of the observations of this series the food occupied relatively the position represented in Figure 6. There was direct contact between the stomach wall and the food only at one surface. Thus the gas prevented the churning of the food by the peristaltic waves and the gradual propulsion of the food through the pylorus.

FIG. 6

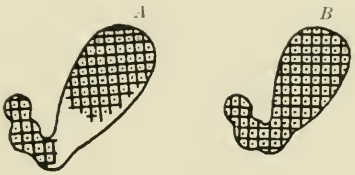

FIG. 7

FIG. 6.—Tracing of the outline of a stomach showing the separation between the walls and the food, caused by gas.

FIG. 7.—*A*. Tracing of the outline of a stomach showing a collection of gas in the pre-antral region. *B*. The same stomach after removal of gas.

The retardation of gastric discharge through the accumulation of gas in the stomach is a result which evidently might be different in man and in the cat. In the upright position of man any gas in the stomach would naturally rise to the fundus, and the food would then be in the region of active peristalsis. Furthermore, the gas might thus be readily voided by eructation. But in the prone position of man gas in the stomach may render peristaltic activities quite as ineffective as it does in the cat.

In connection with these observations there has been observed repeatedly in the pre-antral region an apparently empty space at about the position indicated in Fig. 6. Magnus[21] described a similar appearance in the stomachs of morphinized cats. He attributed the appearance to a persistent constriction of the gastric wall in this region. On two different occasions when we noted this appearance (Fig. 7, *A*) we passed a tube down the œsophagus and into the stomach, watching by means of the fluorescent screen its exact course. When the tube reached the light area, the area disappeared, and when the tube was withdrawn the shadow of the stomach presented a uniform dark appearance (Fig. 7, *B*). The light area was apparently due to gas which was carried off by the tube.

[21] Archiv f. die ges. Phys., 1908, cxxii, 254.

The Influence of Heat and Cold. The possible effects of heat and cold on the secretory and motor functions of the alimentary canal have been studied by various methods. Lüderitz[22] exposed the stomach and intestines of rabbits in a bath of normal salt solution which was gradually cooled. He saw no change in the motor activity until a temperature of 28° to 30° C. was reached. In one case very vigorous peristalsis was seen at 29° C. Below 28° the movements ceased. Oser[23] states that low temperatures close the pylorus, but that higher temperatures, up to 37° C., have no such effect. According to Müller,[24] low temperatures have a quieting, even a paralysing effect on the movements of the stomach, whereas high temperatures increase gastric peristalsis. These observations are in accord with those of Schüle,[25] who found that water at 45° left the stomach much faster than water at 0° or 28° C., but they do not seem to accord with Müller's own results that hot and cold fluids leave the stomach more slowly than fluids at body temperature.

The effect of temperature variations on secretion has been little studied. Cahn[36] reports that when he fed powdered meat with water at 15°, 1 to 1.3 p.m. of HCl was secreted during the first hour, and that with the water at 45° the acid was up to 1.8 p.m. Both these results, however, are lower than the results when temperature is normal. Micheli[27] in a study of the gastric secretion in ninety patients found that with water at 35° to 37° the secretion was greatest, at 45° to 50° least, and at 2° to 4° greater than at room temperature. A similar stimulating effect of cold water was noted by Jaworski.[28]

In such studies as are above cited the time required for the equalization of the temperature of the body and of the ingested food is especially significant, for it is probable that the temperature effects diminish as the equalization takes place. By use of maximum thermometers, Winternitz[29] observed that thirty minutes after drinking 500 c.c. of water at 5° to 7° the temperature of the gastric contents was only 0.6° C. lower than general bodily temperature. On a patient with gastric fistula Quincke[30] obtained similar results when cold water was taken, and further found that water at 40° C. reached body temperature within ten minutes. According to Quincke, hot or cold water reaches body temperature sooner than lukewarm milk. As Müller points out, the stomach is in a high degree able to bring food of widely differing temperature quickly to the tem-

[22] Virchow's Archiv, cxvi, 53.
[23] Ztschr. f. klin. Med., 1892, xx, 287.
[24] Ztschr. f. diät. und physikal. Ther., 1904, viii, 587.
[25] Ztschr. f. klin. Med., 1896, xxix, 81.
[26] Ztschr. f. klin. Med., 1887, xii, 36.
[27] Archiv ital. di clin. med., 1896. Referat, Archiv f. Verdauungs-Krankheiten, 1897, iii, 244.
[28] Deut. Archiv f. klin. Med., 1884, xxxv, 76.
[29] Physiologic Bases of Hydrotherapy, in A System of Physiologic Therapeutics, Philadelphia, 1902, ix, 41.
[30] Archiv f. exper. Path. und Pharm., 1888, xxv, 380.

perature of the body, a function doubtless dependent on the central position of the organ in the body and to the rich blood supply in its walls and in the surrounding structures.

Since the stimulating influence due to variations of temperature is present for only a comparatively short interval, the influence exerted might be correspondingly short; but the possibility of the effect outlasting for some time the period of stimulation must be considered. In the following experiments to determine the rate of discharge of hot and cold solid foods, the conditions of experimentation were quite normal. Care was taken to keep the food at the temperature stated until all had been fed.

In two cases in which the hot food was given the potato was kept in a dish surrounded by a large quantity of water at 50° to 55° C. during the period of feeding, and the animals were fed from a spoon. In the other cases the food was given by means of a syringe, and was delivered into the stomach at a temperature of approximately 60°.

The cold food was fed in a frozen condition, and reached the stomach in frozen lumps.

The following results were obtained:

HOT FOOD, 50° to 60°.			
Hours after feeding	$\frac{1}{2}$	1	2
Aggregate length, in cm., of the food masses in the small intestine	13	27	48
	10	38	45
	14	31	43
	12	31	45
	18	38	45
Average (5 cases)	13.5	33	45
COLD FOOD, FROZEN.			
Hours after feeding	$\frac{1}{2}$	1	2
Aggregate length, in cm., of the food masses in the small intestine	10	29	50
	18	43	49
	19	38	39
	14	34	44
	12	38	44
	12	25	52
Average (6 cases)	14	34.5	46.5

As the tables and the curves (Fig. 8) indicate, the only change from the normal in the rate of discharge of food, hot or cold, is a slight acceleration, but this change is so slight as to be inconsiderable. In none of the cases was there observed any notable variation from the usual peristalsis.

In a series of observations made by Mr. C. R. Metcalf in this laboratory, hot and cold applications applied from 1 to 40 minutes to the abdomen of healthy cats produced no appreciable alteration in gastric peristalsis. It continued without interruption and without evident change of rate as measured by a stop watch. On the other hand, as already reported,[31] excessive cooling of the stomach

[31] Cannon and Murphy, Annals of Surgery, 1906, xliii, 531.

and intestines, by introducing cold sterile salt solution into the abdominal cavity, may be followed by increased activity of intestinal peristalsis. But this is a procedure causing changes of temperature in the bowel too great to be produced by any external applications.

The conclusion seems justified that changes in the temperature of the food do not influence for any length of time either gastric peristalsis or the rate of discharge of solid food from the stomach.

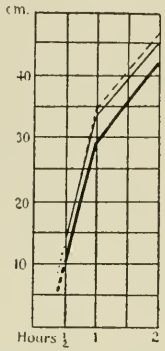

FIG. 8.—The heavy line is the normal curve for potato, the light line that for potato fed hot and the broken line that for potato fed cold.

THE INFLUENCE OF HYPERACIDITY. The earlier literature contains many conflicting statements regarding the relation between gastric motility and the secretion of HCl. Reigel,[32] Kussmaul,[33] and others express the idea that through abnormal acidity the pylorus is kept spastically closed. Similarly, Moritz[34] found in experimenting on himself that 500 c.c. of 0.95 per cent. HCl was more slowly expelled from the stomach than the same amount of water. Some of the earlier investigators regarded the acid of the gastric juice as the real stimulus to peristalsis. Later observations, however, have shown that peristalsis may be present even when the gastric contents are neutral or even alkaline.

The part played by HCl in controlling the discharge of food from the stomach and in stimulating the flow of pancreatic juice has recently been emphasized. The pylorus remains closed until an acid reaction appears in the chyme on the gastric side of the sphincter; it then opens to permit the exit of some of the chyme. Thereupon the acid chyme in the duodenum closes the sphincter, and keeps it closed until the pancreatic juice and bile, caused to flow by the presence of acid in the duodenum, have neutralized the chyme. Then and only then does the pylorus open again and let out more of the stomach contents.[35] In accordance with this explanation

[32] Ztschr. für klin. Med., 1886, xi, 17.
[33] Deut. Archiv für klin. Med., 1869, vi, 460.
[34] Loc. cit., p. 570.
[35] Cannon, Amer. Jour. Phys., 1907, xx, 293.

of the control of the pylorus, hyperacidity might very well cause a retardation of gastric discharge, but it would do so because longer time would be required for neutralization in the duodenum, and the pylorus would therefore be held closed for longer periods. It is of interest to inquire as to the effects of hyperacidity on the rate at which food leaves the stomach. To obtain evidence on this question cats were fed potato with which had been mixed a known percentage of HCl.

In determining the acidity boiled potato was mashed without the addition of water and then concentrated acid was added. Thus, for 1 per cent. acidity, 4.2 c.c. HCl with specific gravity 1.185 (*i. e.*, about 34 per cent.) was added to 100 c.c. potato, weight 140 grams. For the other percentages of acidity the corresponding fraction of this amount of acid was accurately measured and mixed with the potato. The following are the results obtained:

0.25 PER CENT. HCL.

Hours after feeding	½	1	2
Aggregate length, in cm., of the food masses in the small intestine	24	47	72
	25	35	48
	28	40	55
	34	63	64
	22	40	60
	40	52	61
Average (6 cases)	29	46	60

0.5 PER CENT. HCL.

Hours after feeding	½	1	2
Aggregate length, in cm., of the food masses in the small intestine	25	40	58
	25	41	58
	17	34	46
	20	38	49
	20	35	56
	18	30	47
Average (6 cases)	21	36.5	52

1 PER CENT. HCL.

Hours after feeding	½	1	2
Aggregate length, in cm., of the food masses in the small intestine	8	20	34
	9	16	46
	13	35	43
	9	30	72
	14	27	50
	15	19	40
Average (6 cases)	11.5	24.5	47.5

In comparing with the normal conditions these results of feeding acid food, it is fairer to use the second rather than the first half-hour of the normal curve as a standard, since at the beginning of the first half-hour digestion has not begun and no acid has yet appeared at the pylorus, while at the beginning of the second half-hour acid chyme is being discharged. As the curves (Fig. 9) and as

the tables indicate, the rate of exit is faster than normal when the potato has an acidity of 0.25 per cent., and slower than normal when it has an acidity of 1 per cent. Potato with an acidity of 0.5 per cent. is discharged during the first half-hour about as rapidly as the food is normally discharged. The difference between the outgo of the weakly acid (0.25 per cent.) and the strongly acid (1 per cent.) potato is remarkable. Note that at the end of the first half-hour there was in the intestine more than two and a half times as much, and at the end of an hour about two times as much of the weakly acid potato as of the strongly acid. The rapid exit

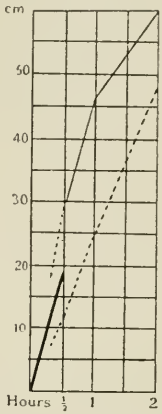

FIG. 9.—The heavy line is the curve (for the second half-hour) when potato is fed normally; the light line, when fed with 0.25 per cent. acidity (HCl); and the dotted line, when fed with 1 per cent. acidity.

was confirmed by the appearance of the stomach: in several instances the stomach was practically empty at the second observation—a condition normally never seen. The appearance of the food masses in the intestine was also characteristic, for long, continuous coils of food were regularly observed.

In all the experiments on the feeding of acid food gastric peristalsis was especially deep and rapid. The rate was usually slightly faster than six waves per minute. In one instance, at the half-hour observation, the waves passed so rapidly as to attract attention. On timing them with a stop-watch twenty waves were seen to move over the antrum in sixty-six seconds, more than eighteen per minute. This rate was so much faster than normal that the counting was repeated, with the same results. At the one-hour observation and thereafter, however, the waves passed at the usual rate.[36]

[36] Opportunity was offered to note the rate of gastric peristalsis in four kittens about six weeks old. The number of waves per minute was within the limits observed in adult animals. The rate of discharge from the stomach was likewise not remarkably different from the rate observed in adults.

The Influence of Massage. The animals selected for observations on the effect of massage were especially docile; they were thus selected in order that the manipulation might not enrage them and thereby introduce as a complicating factor the inhibitory influence of a strong emotional state. Most of the time purring indicated a peaceful tranquillity. Massage was applied by gently kneading the abdomen, especially under the last ribs, as the animal was lying comfortably on its right side. None of the animals was massaged all the time between observations, but of six, four were massaged one-fourth of the time and two one-half of the time. In spite of this extensive manipulation there was no considerable change in the rate of gastric discharge. The average figures for the first three regular observations were 12, 25, and 36 cm., instead of the normal 10, 28, and 41.5 cm. The gastric peristaltic waves, which passed at the usual rate, were notably deep. Segmentation in the small intestine was also quite marked.

The Influence of Irritation of the Colon. It has recently been shown that resection of the small intestine some distance below the pylorus results in such strong and continuous contraction of the pyloric sphincter that, in spite of vigorous peristalsis, food may not begin to leave the stomach for four or five hours after feeding.[37] It is of interest to know how far along the alimentary canal such irritation may reflect its influence on the stomach. The clinical observation that inflammation of the appendix may disturb gastric digestion indicates the possibility of effects on one part of the canal from abnormal conditions in a relatively remote region. The following experiments were tried with the purpose of determining whether irritation of the colon would influence the exit of food from the stomach. In these experiments croton oil was used as an irritant. In no case did the animal show signs of discomfort from the procedure.

Five cats, a few hours after receiving a cleansing enema, were fed 25 c.c. potato and were then injected per rectum with 50 c.c. sweet oil containing 1.25 c.c. croton oil. When observed, the peristalsis of both the stomach and intestine seemed unusually vigorous. The stomach was peculiarly elongated. The rate of discharge may be judged from the following table:

Hours after feeding	½	1	2
Aggregate length, in cm., of the food masses in the small intestine	23	39	39
	0	1	16
	16	17	33
	0	5	19
	0	12	22

The irregularities of the figures in the above table can perhaps be explained on the impossibility of clearing the colon and the

[37] Cannon and Murphy, loc. cit., p. 516.

consequent uncertainty as to the degree of dilution of the irritant by the contents of the bowel. Three of the five cases, however, showed a marked slowing of the discharge.

In order to control conditions more exactly, four cats were etherized, and, under aseptic precautions, a few drops of croton oil injected into the cecum through a small median incision in the abdominal wall. The next day they were fed the standard potato and observed. In all the animals gastric peristalsis was vigorous, but in none of them was there any discharge into the intestine during the first half-hour. In two cases gas appeared in the stomach at the end of the fourth hour, and in one of these cases so much gas accumulated that the distended stomach caused stretching of the abdominal wall and some respiratory difficulty. In another case the first food to enter the colon passed into it during one of the observations. The small mass was forced into the large intestine and pushed upward a short distance, whereupon it was violently segmented for a few moments. Thirty minutes later no more food had entered the large intestine, but a large solid coil of food, the shadow of which was about 17 cm. long, seemed to be blocked immediately before the ileocolic valve. The colon was filled with gas so that its outline was clearly visible. The following table shows the results of these experiments:

Hours after feeding	½	1	2	3	4	5	6	7	8
Aggregate length, in cm., of the food masses in the small intestine	0	0	9	25	34	34	37	45	40
	0	6	32	44	45	33	47	44	49
	0	0	6	18	21	39	41	45	51
	0	8	8	30	29	33	32	41	37
Average (4 cases)	0	3.5	13.5	29	32	35	39	43.5	44

Comparison of these figures with the figures from normal cases (Fig. 10) shows at once remarkable differences. Not only is the gastric discharge much slower when the colon is irritated, but the passage of the food through the small intestine is greatly retarded, The curve drops mainly because of the passage of material into the large intestine. Note that when the colon is irritated the curve fails to drop throughout eight hours, whereas the normal curve begins to drop at the end of two hours. Normally potato begins to appear in the colon at the end of two or three hours; under the condition of the present experiment, however, it did not appear in the colon until six or seven hours had elapsed. In all cases food was still present in the stomach at the end of seven hours, though normally the stomach is emptied of this food in about three hours.

To prove that the effects noted above were not due to the operative procedure alone, four cats were operated upon as before, and, instead of croton oil, a few drops of water were injected into the colon. In two of the cases the gastric discharge on the next

day was somewhat delayed, but not nearly so much delayed as when croton oil was injected; the other two cases were close to the normal.

The conclusion is justified that irritation of the colon can cause marked retardation in the rate of exit of food from the stomach and in the passage of food through the small intestine.

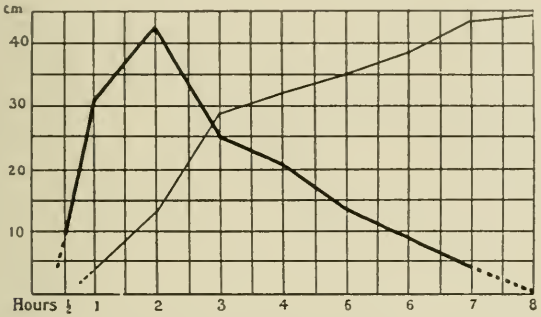

FIG. 10.—The heavy line is the normal curve for potato, the light line the curve when croton oil has been injected into the colon.

SUMMARY. If carbohydrate food is thinned by adding water, there is, within limits, very little change in the rate of exit from the stomach; but adding water to protein food tends to make the discharge more rapid. When hard particles are present in the food the rate of outgo from the stomach is notably retarded. Coarse branny food leaves the stomach slightly faster than similar foods of finer texture. The presence of gas in the stomach delays gastric discharge, an effect due to the gas preventing the walls of the stomach from exerting the normal mixing and propelling action on the food. No considerable variation from the normal rate of exit from the stomach is observed when the food is fed very hot or very cold. Food with approximately normal acidity leaves the stomach much faster than food which is hyperacid (1 per cent.), a result in harmony with other observations on the acid control of the pylorus. Feeding acid food is followed by deep and rapid peristalsis. Massage of the stomach, even when extensive, has very slight influence on the passage of food through the pylorus. Irritation of the colon (with croton oil) notably retards gastric discharge and delays the movements of food through the small intestine.

THE RELATION OF THE FOODSTUFFS TO ALIMENTARY FUNCTIONS.

By Lafayette B. Mendel,
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The great development of experimental research which has taken place in recent years in the study of the digestive functions has inaugurated a variety of new viewpoints in this department of physiology. In the earlier days attention was concentrated upon the processes carried out in the stomach, and the activities of the remainder of the alimentary tract failed to be drawn into consideration. The recognition of the importance of the changes which have their seat in the intestines was the outcome primarily of biochemical investigation. It is not long since we were accustomed and content solely to analyze the changes which the alimentary processes bring about in the foodstuffs—to study the effect of the digestive enzymes upon them, and to investigate the disappearance of the products from the digestive tube. Considerations such as these comprised practically the sum total of alimentation as it was then appreciated.

But instead of recounting the transformations which the foods experience in the digestive processes, instead of debating where and whether fats, carbohydrates, and proteins are completely disintegrated into simpler molecules before they are reconstructed into forms of flesh and blood, another aspect of the alimentary functions has been proposed for this discussion. The familiar query regarding the changes which digestion imparts to the foodstuffs will for once give way to a newer and reversed point of view. Let us inquire what influence the foods have on the alimentary processes. It will appear that, aside from their value as nutrients, the components of the diet exercise specific influences upon the alimentary organs. Are not such physiological agencies an appropriate theme for consideration in the field of therapeutics?

So long as the secretion of the digestive juices and the regulation of the muscular mechanisms of the gastro-intestinal tract were assumed to be under the sole dominance of a nervous apparatus, the *character* of food eaten was destined to play an insignificant part in the secretory and muscular responses. Fat and starch alike were food; and the physiologist was not inclined to discriminate between them in their action on secretion. The gush of chyme in the duodenum, like the bolus of food in the stomach, provoked a flow of digestive secretion by some obscure mechanical agency or some nervous reflex. We have become emancipated from these broad generalizations. Not all foodstuffs provoke secretion in equal degree. Not all constipation is due primarily to failure of peristalsis. The skilled

therapeutist of today can well afford to devote to the specific action of the diet the same detailed consideration which he accords to his armamentarium of drugs.

Perhaps the most fruitful and suggestive of the more recent ideas has been that of the *hormones*, or chemical excitants, which travel about the organism. Ever since Bayliss and Starling demonstrated that the flow of the pancreatic juice and the bile can be incited not only by nervous reflexes, but also by the absorption of definite chemical substances which are despatched through the blood stream to the pancreas and liver respectively, and there set up increased secretory activity, speculation has been rife as to the extent to which such chemical regulation of our functions is maintained. We have been taught to recognize the humoral paths of stimulating influences—to appreciate that active chemical agents may travel about the body from part to part, and awaken responses through the circulation as well as through the less direct nervous paths.

One of the newest of these hormones, or chemical excitants, to deserve attention in this connection has lately been brought to notice by Edkins. He has shown that the contact of certain food products with the pyloric end of the stomach gives rise to some chemical substance—a gastric hormone or secretagogue—which acts as a powerful stimulus to gastric secretion when it is introduced into the circulation. In the case of the pancreas, as is well known, the presence of acid in the duodenum awakens a speedy response: in part, this may be due to a nervous reflex; in part, probably also to the formation or liberation of a specific substance (a secretin) in the walls of the intestines, which travels directly to the glands in the blood current and provokes secretion. Now, in the stomach, acid alone is at best a slight stimulus; but dextrins, maltose and dextrose, proteoses, and, above all, extract of meat, are pronounced gastric secretagogues. They do not act from the fundus; but are absorbed in part and act from the pyloric end of the stomach and the duodenum. The first products of digestion, therefore, acting on the pyloric membrane, liberate a substance—the hormone, gastrin—which is absorbed into the blood stream and carried to all the cells of the stomach where it functions as a specific excitant of their secretory activity. Here then is furnished a definite illustration of certain food products arousing the gastric cells. We learn a dietetic method of inducing secretion in the stomach. The psychic flow which comes at the outset of the meal and has been made familiar through the researches of Pawlow is nervous in origin. It can, accordingly, be provoked, modified, or inhibited through purely nervous channels. The later and more prolonged flow of gastric juice is associated with the food derivatives. This food factor in gastric secretion is, by no means, an entirely new conception. The germ of the modern work may be found in the older observations by Schiff. But the work of Edkins and others has emphasized the specific nature of the

excitants and the fact that they act directly through absorption and not primarily on a central nervous mechanism. Bouillon or meat soup taken early in the meal soon reaches the pylorus and duodenum and incites a flow of gastric juice. The dextrins and maltose of toasted bread and zwieback perform the same function. We have thus learned an application of dietotherapy to awaken the dormant secretory glands. Abundance of secretion in the stomach in turn promises abundance of pancreatic juice and bile, because of the excitatory function of the gastric hydrochloric acid when it reaches the duodenum. The intestinal secretion is likewise said to be facilitated through the excitation by hormones.

On the other hand, fats are well known to *inhibit* gastric secretion in a striking manner. Thus, the nature of the food intake may materially modify the secretory responses of the alimentary tract and its appendages. The better appreciation and extension of the laboratory studies on the responses of the secretory glands to specific food products already give promise of useful application in the field of clinical medicine. When drugs still largely fail, a carefully selected dietetic regime may succeed in awakening or repressing organs upon the proper secretory function of which our nutritive welfare depends.

There are, furthermore, numerous and significant indirect or secondary influences, which arise from the regulatory function exercised by certain foods upon the alimentary processes. Whatever promotes an abundance of HCl in the stomach and insures its discharge through the pylorus, provokes a liberal flow of bile, and a consequent ready absorption of the digestion products of fats. Again, high or low gastric acidity markedly modifies the extent of the putrefactive changes in the bowel. The relaxation and closure of the stomach sphincters at the cardia and the pylorus are under the control (in large measure) of the reaction which prevails there. Foods which tend (like proteins) to combine with acid and diminish the "free" HCl, or (like fats) to repress acid secretion, will delay the discharge from the stomach. Whatever facilitates speedy accumulation of acid in the pyloric region tends toward an earlier emptying of the organ into the parts beyond. Newer investigations suggest that bile stimulates peristalsis in the intestines. Enough has been said to indicate how intimately and intricately the food influences may involve the ensemble of digestive phenomena.

In this connection, mention may be made of the interesting discovery, by Boldyreff, of the regurgitation of duodenal contents into the stomach under appropriate conditions. This is most likely to occur when the stomach is empty or acid secretion has been repressed to a minimum by ingestion of fats. Pancreatic juice may thus be obtained from the stomach, and the method has already been introduced into clinical usage for the diagnosis of pancreatic conditions. Migaj has suggested that strong acids in the stomach are neutralized

by such regurgitations of alkaline duodenal contents. It should be noted, finally, that even peristalsis has been brought under the alleged domination of hormones: Zuelzer claims that at the height of digestion the mucosa of the stomach yields a substance which causes energetic but natural peristalsis extending throughout the intestinal tube. This, like many of the interesting speculations which the introduction of a new idea into physiology has evolved, demands a further critical examination before it deserves to be incorporated with the more permanent acquisitions of our science.

Another and somewhat different conception of the influence of the foodstuffs upon digestive phenomena owes its prominence to the authority of Pawlow. He has taught that the secretory glands may respond in a unique way to variations in the type of foodstuffs ingested. According to him there may be a specific adaptation of the secretions, such as that of the pancreas, so that the richness of the juice in proteolytic, amylolytic, or lipolytic enzyme varies with the nature of the diet. We are taught to speak of "meat" juice, or "bread" juice, or "milk" juice, each varying noticeably in its relative digestive power. It must be admitted that an adaptive response of this sort by which more enzyme of one type or another is produced specifically as it is needed, in reply to the stimulus of the individual foodstuffs, presents a remarkable specificity of physiological function. However, the consensus of opinion at the present moment speaks against such a specific sensibility of the mucous membrane of the alimentary tract, alluring as the idea may be. One of the critics of the adaptation hypothesis (Popielski) naïvely remarks that the welfare of the organism will be better assured if we ourselves conduct the analysis of our foods than if the task is left to the digestive glands. Despite contradictory statements it appears in the case of the saliva that the texture, more than the composition of the food, is a decisive factor in awakening the secretory response.

In addition to this there is, however, a further type of claim which postulates even a more radical adaptation of secretions to the diet. The feeding of specific foods is assumed to induce the formation of desired enzymes, in some cases even when they are never found at all under usual conditions. Much more satisfactory evidence than has yet been offered will be needed to convince the unbiassed physiologist that cells can suddenly assume new roles with the ease which has been claimed—that a few days' feeding with bread or milk sugar, for example, will elicit the production of starch-digesting or sugar-inverting enzymes in secretions in which they are normally wanting. These problems of adaptation are still in the realm of the unproved. As Starling says: "There are riddles enough in physiology without conjuring up a teleological adaptation for which the experimental evidence is inadequate, the conception of the mechanism impossible, and which is not necessary for the well-being of the animal."

Examined from a still different point of view, the food factor is of

preëminent significance in relation to alimentary changes. It modifies the extent of the bacterial transformations which go on there; while Metchnikoff and his followers would have us believe that it is possible by dietetic regulation to alter decidedly the bacterial flora of the alimentary tract. The usefulness of lactic acid, sour milk, and lactic acid ferments as curative agents is nothing less than problematical at the present moment. But the very principles underlying their use emphasize the possibility of regulating alimentary changes most profoundly by the character of the food intake and its natural concomitants. The dietotherapy of infants has lately witnessed the advent of a new theory for the etiology of the alimentary intoxications (so-called) of children. Whereas, for a long time attention has primarily been directed to the dangers of the products of the intestinal flora, Finkelstein maintains most emphatically that these familiar disturbances are ofttimes nutritive (metabolic) and digestive in character, rather than more specifically bacterial. He directs attention to the food rather than the ferments in the treatment of these infantile disorders. "Food intoxication," if we may coin the expression, is by no means a novelty; nor are we prepared as yet to realize its possible consequences. A secretory function fails; fats are improperly absorbed; the fatty acids form soaps of calcium, robbing the growing organism of its supply of lime, which is wasted with the stools; acidosis intervenes. What a serious train of symptoms can here be avoided by a timely consideration of the relations of food to the alimentary processes. Drug medication is of insignificant moment amid such complications. The possibilities of the newer knowledge of the laboratory must be translated into practice and there made effective. As Professor Ewing has appropriately remarked in another connection: "The feeding of healthy man, as well as the diet of the sick, cannot be left to chance, guided by appetite, or ruled by tradition, but can be safely directed only according to the laws of digestion and metabolism."

DIET AND THE CARE OF THE BOWELS IN TYPHOID FEVER.

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"Lack of perception is the devil's best friend."

Typhoid fever is characterized by infection, and in the vast majority of cases by local ulceration in the ileum and the cecum. The prognosis in normal individuals depends upon the degree of the infection and the extent of the local ulceration in the intestine.

The former can be fairly well measured by the fever, the nervous symptoms, and the degree of prostration present; the latter is practically unknown. Notoriously we have as yet no efficient, specific treatment. The well-being of the patient depends upon the care he receives as to diet, bowel movements, and rest.

Diet in typhoid fever must be prescribed with two objects in view —maintenance of the nutrition and the avoidance of gastro-intestinal disturbances. It is a well-known fact that until the time of Graves, in 1840, patients with any form of fever were depleted, according to the teaching of Sydenham. Since Graves' teaching, patients with fever, notably typhoid fever, have been fed with the view of limiting the nitrogen loss to a minimum.

The diet in typhoid fever until the last fifteen years was liquid, with milk as a basis. Within the last fifteen years, however, a diet much more varied has been recommended by eminent men, notably, Shattuck, Barr, and Bushuyev. Any recommendation coming from Frederick Shattuck compels attention He cites 241 cases treated over a period of eleven years, on a most liberal diet, with great comfort to the patient and with no increase in mortality. The claims made by the advocates of a liberal diet may be tabulated as follows:

Liberal diet.	Liquid diet.
1. Satisfies the patient.	1. Disgusts the patient.
2. Provides sufficient calories.	2. Provides insufficient calories.
3. Does not produce a culture medium in the bowel.	3. Produces an excellent culture medium in the bowel.
4. Does not cause intestinal irritation.	4. Causes intestinal disturbances by curds and distention.
5. Does not increase the mortality.	
6. Does not increase perforation and hemorrhage, but rather diminishes them.	6. Increases perforation and hemorrhage, because of curds.

Taking these claims up seratim:

1. The comfort of the patient is unquestionably greater when he is given food other than milk and water. Three or four weeks of an absolute milk diet is most trying to the patient if he be not very ill, and is perhaps still more trying during convalesence. No one who has seen the change in mental attitude of a patient on milk when he is ordered milk in the form of junket, is given ice cream, or thin cornstarch, egg, or other suitable diet, can doubt its good effect, mentally, at least. Those who are in the habit of giving the diet suggested by Shattuck claim still greater improvement of the moral.

2. The usual amount of milk given in the course of a liquid diet, maintained at St. Timothy's Hospital, Roxborough, Philadelphia, varied from 48 to 72 ounces in twenty-four hours, in each of 514 cases, and this I think agrees well with the usual amount given. According to Chittenden, 2 quarts of milk supply 1300 calories. To supply 2700 or 2800 calories, which in health is Chittenden's necessary amount, each patient should receive 4 quarts of milk. Our

patients had no more than 2½ quarts, or 1625 calories. We must acknowledge that, theoretically at least, these 514 patients were decidedly underfed. The result of these cases, however, which agrees fairly closely with other groups of cases, would go to show, that however much more comfort can be obtained from a fuller diet, so far as recovery is concerned, it is not a necessity. A large number of metabolism experiments made by Puritz,[1] in 1890 to 1892, in which typhoid fever patients were fed on meat, bread, milk, eggs, tea, coffee, and port wine, shows that from 79 to 82 per cent. of the nitrogen consumed was assimilated. These experiments go to strengthen the position of the adherents of a more liberal diet, for if 80 per cent. of the nitrogen consumed can be assimilated, certainly it is common sense to give it to the patient, provided it does not otherwise harm him.

3. The claim that milk as compared with solid foods causes a good culture medium in the bowel, is not well founded. Certainly the entire intestinal canal is swarming with all sorts of bacteria. It matters not, it seems to me, whether the food be taken as milk or more solid food, that which remains must provide the culture medium. There is no more refuse in the bowels after a diet of milk than after other diets. In many autopsies, made by myself in St. Timothy's Hopsital, I have failed to find the lower bowel overfull, or containing a large number of curds, though the patients had consumed nothing but milk during the course of the illness.

4. Does milk cause more or less intestinal irritation than the same quantity of other food? One of the chief objections to a liberal diet, in the minds of those who use a liquid diet, is the fear that the solid food will leave so much residue in the intestine that the ever-present ulcerations will be irritated, and the intestinal accidents, perforation, and hemorrhage increased. It must be remembered, in this connection, that milk on entering the stomach at once becomes a solid food, although easily digested.

Statistics published by Kinnicutt go to show that perforation and hemorrhage are slightly less in patients on a free than in those on a milk diet, but aside from this, which will be considered later, the tympany and diarrhœa which some writers attribute to milk diet are more in the imagination than in reality. When tympany does occur with a milk diet, when diarrhœa is excessive, the milk may be withdrawn; it may be substituted by peptonized milk, junket, or albumen water. In the series of cases from St. Timothy's Hospital diarrhœa occurred 113 times, but it was rarely excessive; when more than four stools occurred daily, it was considered diarrhœa. It would appear, therefore, and this has been my personal experience, that milk diet does not, as a rule, cause undue distention or diarrhœa; when it does the diet may be controlled, so that the

[1] Quoted by Atwater and Langworthy.

difficulty may be obviated. We may well judge the effect of a liberal diet and a liquid diet upon mortality, perforation, hemorrhage, and relapse, by a collection of a large number of cases. It must be remembered, however, that statistics alone are not to be depended upon for information, however great their help. To help decide this question, Kinnicutt has published some remarkable statistics collected from different hospitals. I have another series to submit from St. Timothy's Hospital.

KINNICUTT'S TABLES. Free Diet.

Number of cases.	Relapse.		Hemorrhage.		Perforation.		Mortality.	
	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.
733	48	15.2	35	4.77	10	1.36	60	9.47
Basis of	315 cases		733 cases		733 cases		633 cases	

Liquid Diet.

Number of cases.	Relapse.		Hemorrhage.		Perforation.		Mortality.	
	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.
4654	507	10.89	411	8.83	111	2.38	491	10.55

ST. TIMOTHY'S HOSPITAL TABLES. Liquid Diet.

Number of cases.	Relapse.		Hemorrhage.		Perforation.		Mortality.	
	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.
514	43	8.36	54	10.50	21	4.00	69	13.42

COMBINED TABLES. Liquid Diet.

Number of cases.	Relapse.		Hemorrhage.		Perforation.		Mortality.	
	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.
5168	550	10.64	465	8.99	132	2.74	560	10.83

Dr. J. C. Wilson[2] has published a series of 1904 cases of typhoid fever treated by the method of Brand with a mortality of 7.5 per cent. No mention is made in the article as to the diet, and certainly the cases were not reported with that end in view. As all the cases were treated in Philadelphia, it is presumed that the cases were given a liquid diet. The series shows, however, that the diet alone is not the only factor which affects the mortality.

These tables, so far as they are of value, show the following. In the first place the mortality is somewhat less in the series on a liberal diet. In 633 cases fed on liberal diet, there was a mortality of 9.47 per cent. In 5168 cases on milk diet, the mortality was 10.83 per cent.—a difference of a little over 1.75 per cent. in favor of a free diet. Relapse was present 550 times, or 10.64 per cent., in liquid diet as against 15.2 per cent. in free diet. Hemorrhage was present in 8.99 per cent. as against 4.77 per cent. in liberal diet. Perforation was present in 2.74 per cent. as against 1.36 per cent. in liberal diet. These differences are certainly negligible. For instance, of my own cases, which are all classed under liquid diet, and were treated other-

[2] Phila. Med. Jour., March 3, 1900.

wise entirely differently, several died in a few hours after their admission. The diet had nothing to do with their demise. In one year at St. Timothy's Hospital there were 12 deaths. Every death was due to perforation; certainly two patients were admitted with a perforation, and one died of this accident one week after the patient was on soft diet. If these cases were excluded, the statistics would vary decidedly. In that particular year there would have been the difference between 9 and 12 per cent.

We, who have seen the stools of normal adults and children after eating immoderately, and know how they are filled with curds if given large quantities of milk, undigested particles of meat, etc., can realize the foolhardiness of using any diet in typhoid fever except under strict watchfulness. Any physician of experience will be certain, without statistics, that gross errors in diet do cause both relapses and intestinal accidents in typhoid fever. Two such cases come to mind as I write; they could be multiplied many times. One, a case in St. Timothy's Hospital, which was convalescent without any symptoms of any kind, at the end of two weeks, while on soft diet developed, a sudden rise in temperature, and in two days after, a perforation. At the operation and subsequent autopsy the intestine was found fairly well filled with fecal matter, which had perforated an ulcer. It would appear that this particular perforation occurred because of irritation of an unhealed ulcer by a large fecal mass. A second case, a girl, aged twelve years, had passed through a severe attack of typhoid fever. She had a normal temperature for ten days. The family was told that her milk might be increased. They at once gave her six quarts in twenty-four hours, with an immediate result of a severe relapse. Certainly, it appears that both of these results were due to excess in diet, one of milk, the other of a liberal diet.

If hemorrhage occurs, all food must be withdrawn. Water may be given in small quantities; in twenty-four hours albumen water, then peptonized milk. After one week the usual diet may be resumed. If perforation occurs, there is no question of diet. The case must at once be handed to the surgeon for operation. Thromboses are said to be much less frequent in cases well fed than in cases fed on liquid diet. There are no statistics known to me to prove this point. My own cases cannot be used for this. I believe there is such a thing as fever of malnutrition following a liquid diet. Patients may run a temperature of one or two degrees above normal for days and weeks after a normal temperature. In these cases I think food must be given. It is claimed that they do not occur with a full diet.

Shattuck, in discussing Kinnicutt's paper in 1906, said: "For more than ten years I have been feeding my patients according to their digestive power, rather than according to the name of the disease, avoiding such articles of diet as can leave an irritating residue. I am convinced that comfort is promoted and convalescence shortened."

Bushuyev treated 398 patients in the Military Hospital in 1895 to 1897. He[3] is most enthusiastic, saying: "Under this regimen the general condition of the patients is incomparably better than when kept upon an exclusively liquid diet. The common complaints are scarcely ever heard. At breakfast, dinner, and supper the patients are uncommonly wide awake. Even those who are very ill sit up in bed, beg for food, and eat with much satisfaction; only a few have to be fed by nurses. If one observes the patients at meal times he wholly forgets that these individuals are seriously ill with temperature above 39° C. During the first hours in the ward the patient lies in a motionless condition, failing to answer questions and refusing food. But if one succeeds in some way or another in persuading him to eat a bit of meat or cutlet, or an egg, he immediately begins to show some interest in his surroundings."

These are certainly commendatory sentences. In this country we would doubt the advisability of patients sitting up in bed with typhoid fever, even though they were able. Bushuyev treated 318 cases on the diet described, with a mortality of 8.2 per cent. as compared with a mortality of 12.4 per cent. in 2887 patients in the same hospital during the ten preceding years.

If one compares the diet list of Shattuck with that of Bushuyev, both of which are appended, it is easy to choose between the two. Bushuyev gives no directions as to methods of feeding, nor preparations of foods. Shattuck's is the more rational.

My advice as to diet for typhoid fever cases is to study each patient well, and feed him according to his needs. First, put the patient on a liquid diet, with milk and egg albumen as a basis. Increase the diet up to a limit of 2500 calories, by the addition of starches, minced chicken, or meat, chopped soft parts of oysters, soups, after the diet of Shattuck. Keep a careful watch upon the stools and on the digestive functions, to see that neither undigested curds or other particles pass, and that tympany and diarrhœa and constipation do not occur. The presence of particles in the stool, whether milk curds or other matter, is at once to be taken as an indication to remove the offending article of food from the diet list, or change its character. If diarrhœa or tympany continue, stop all food and begin again with digested milk, eggs, etc.

To paraphrase Kinnicutt, the employment of a fixed diet in all cases of typhoid fever would be as irrational as a decided limitation of diet in many cases of typhoid fever.

The care of the bowel movements is akin to that of the diet. The keynote must be the avoidance of routine. It would appear certainly unscientific to give salines constantly to a patient already suffering from an irritated and ulcerated intestinal mucous membrane. On the other hand, it is irrational to allow a patient to go several days

[3] I quote from a translation by Kinnicutt.

without a bowel movement. While the first practice irritates the bowels by undue action, retention of fecal matter may allow the patient to absorb deleterious substances, or the retained fecal matter may irritate the ulcers by its presence. The safe course is to give the patient divided doses of calomel, if seen in the first week, in order to remove any irritating substance that may remain in the bowel. After that, a daily evacuation must be secured by a glycerin or a simple enema, if there is a tendency to constipation. If diarrhœa occurs, food should be stopped or regulated. If it then persists, a mixture of salol and bismuth should be given, or an opiate if excessive.

To conclude: Clinical and autopsy evidence proves that a regulated milk diet does not irritate the bowel, neither does a diet of milk re-inforced by carefully selected carbohydrate and nitrogenous diet other than milk. Common sense must be applied to both, and both may be used, but in semiconscious and unconscious patients the diet is perforce liquid. The excessive excavations should be controlled if diarrhœa is present. One bowel movement should be obtained by daily enema if there is constipation.

Shattuck's dietary in the Massachusetts General Hospital consists of:

1. Milk, hot or cold, with or without salt, diluted with lime water, soda water, Apollinaris, Vichy; peptogenic and peptonized milk; cream and water; milk with white of egg; slip buttermilk, koumyss, matzoon, milk whey, milk with tea, coffee, cocoa.

2. Soups: Beef, veal, chicken, tomato, potato, oyster, mutton, pea, bean, squash; carefully strained and thickened with rice (powdered), arrowroot, flour, milk or cream, egg, barley.

3. Horlick's food, Mellin's food, malted milk, carnipeptone, bovinine, somatose.

4. Beef juice.

5. Gruels: Strained cornmeal, crackers, flour, barley water, toast water, albumen water with lemon juice.

6. Ice cream.

7. Eggs: Soft boiled or raw; eggnog.

8. Finely minced lean meat, scraped beef. The soft part of raw oysters. Soft crackers with milk or broth. Soft puddings without raisins. Soft toast without crust. Blanc mange, wine jelly, apple sauce, and macaroni.

The total number of cases treated by Shattuck on this principle is 241, extended over the period from 1893 to 1904.

Bushuyev's daily menu in the Kief Military Hospital, Russia, is as follows:

7 A.M. Tea with roll.

8 A.M. 400 c.c. of soft (liquid) oatmeal, barley, or wheat porridge, with butter.

9 A.M. One or two boiled eggs, soft or hard, as the patient desires.

10 to 11 A.M. A glass (200 to 220 c.c.) of milk with a roll, one-half a cutlet, and a bit of boiled meat (160 to 168 grams).

12 to 12.30 P.M. A plate (220 c.c.) of chicken soup or a bowl of ordinary soup, sometimes with a bit of chicken from the soup, and a small cup of "kisel" (a sort of sour jelly); rarely, a little preserved fruit.

3 P.M. Tea with roll.

6 P.M. A cup of chicken or beef soup; semolina pudding or milk, a bit of chicken.

8 P.M. Milk with roll.

During the night: Coffee or tea with milk, two or four times; coffee with cognac.

In addition, the patients receive from one to three ounces of wine in the morning, and every two hours half an ounce of Stokes' mixture.

THE ETIOLOGY OF LOOSE BOWEL MOVEMENTS.

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Before considering from a clinical standpoint the etiology of loose movements of the bowels, a brief statement of the physiological facts which have a bearing on the condition may be of service. In the intestinal tract we have to deal with a canal which is lined with various forms of glandular epithelium, and which has to perform the important functions of digestion, absorption, and, finally, elimination. Its activities are presided over by a sensitive nervous system energized from, and sympathetic with, the nervous centres in the cerebrum and cord. For its nutrition it is dependent upon the general blood and lymph stream, and is an early sufferer when these are at fault. Moreover, it is in direct communication with the external air, and becomes a culture ground for bacteria, some harmless, possibly even beneficial, while others are potentially dangerous and on occasions may be the cause of much disturbance and may even bring death to the whole organism. These functions and conditions are all mutually complimentary, so that disorder in one promptly leads to disturbance in the others.

As one of the by-results of the effective co-working of these functions, the majority of individuals secure one daily evacuation of fecal material of a soft-solid consistence, from which nutritive material has been almost entirely abstracted. In a few, evacuations may number two or three per day, and in another small group of individuals the frequency of the evacuations may be reduced to one every second

or third day. Both these conditions verge on the pathological, but so long as the general health in no way suffers and the movements are moderately firm in consistence, they can scarcely be said to demand interference on the part of the physician. An alteration in the consistence of the evacuations, however, must be regarded as more important. The contents of the small bowel are normally fluid and pass in from four to six hours from the stomach to the cecum. Considerable inspissation takes place in the ascending colon, and the now slowly moving contents of the transverse and descending colon acquire, under normal conditions, a distinctly firm consistence. The presence of an undue amount of water in the evacuations must, therefore, be regarded as indicative of some disordered function, the importance of which will vary according to the underlying cause, and to the systemic effects produced.

Looseness of the bowels, as it is popularly termed, may be dependent primarily upon: (1) Excessive propulsive peristaltic action; (2) an abnormal increase in the amount of secretion from the mucous membrane and secreting glands of the canal; and (3) defective absorption. These may work in a vicious circle. Increased peristalsis prevents absorption, and is often associated with increased secretion; increased secretion stimulates peristalsis and interferes with absorption; and defective absorption stimulates peristalsis, and is generally associated with some fault in secretion.

Although some physiologists regard true peristalsis as, in part, of myogenic origin, experiments indicate that it is, for the greater part at least, reflex in character, and that the stimuli which evoke it arise almost entirely from the bowel contents; these stimuli are directed and energized by the plexuses of Meissner and Auerbach, but are modified and controlled by impulses from the central nervous system through the vagus and splanchnic nerves.

Such stimuli may be the result of mechanical or of chemical irritation. The bowel contents act mechanically as stimulants to peristalsis by their bulk and by the coarseness and indigestibility of their particles. Chemical irritation arises for the most part from acidity or acridity in the contents; such acidity or acridity may develop as a result: (1) Of irritants introduced with the food; (2) from irritants the product of faulty digestion; or (3) from irritants developed from abnormal fermentation or other undue bacterial activity in the intestines.

The action of these stimuli will be increased by any abnormal irritability of the bowel wall including any inflammatory condition of the mucosa, or any excess of irritability in muscle fibers or in the nervous centres. Still further the influence of mental fatigue and mental emotions over the activities of both stomach and intestines is well recognized, especially in the hysterical and neurasthenic.

Secondly, undue fluidity of the evacuations may have as their primary cause excessive intestinal secretion. The secretions from the

stomach, the liver, the pancreas, and from the intestinal glands proper are, as we all know, very abundant, but in normal conditions much of their watery, and some of their solid, constituents are re-absorbed into the general circulation. Should absorption be impaired and true peristalsis be unduly active, this re-absorption takes place imperfectly and fluid movements will result.

All local irritants also to some extent stimulate secretion; some bacterial toxins appear to act in this way, producing excessive secretion of watery fluid, followed by copious evacuations, as for example, the rice water discharges of cholera. The presence of certain salines in the bowel contents also stimulates secretion and by osmotic action checks absorption. It must not be forgotten also that the large epithelial cells of the colon act as eliminating organs and that the toxins eliminated by them not infrequently stimulate peristalsis.

Loose evacuations may be primarily dependent upon defective absorption. Defective absorption may in some measure be due to imperfect digestion and preparation of the food for absorption. More important, however, is the interference due to conditions which interfere with the vital activity of the columnar cells lining the mucosa, to degenerative changes affecting the permeability of the walls of the intestinal blood vessels, and to any stasis of the portal or lymphatic circulation, all of which conditions may have an important place in the etiology of loose movements.

Closely associated with these conditions, sometimes as a cause and sometimes as a sequence, is an abnormal development of bacteria in the intestinal tract. Although the infant is reported to come into this world with sterile intestines, contamination of them quickly occurs, and the canal of an infant a few days old is said to contain a numerous flora. In the adult stomach numerous microörganisms, derived in part from the ingested food and in part from the air, are found. The intestines also, normally, contain many forms of bacteria to which the system has grown accustomed, and which are said to favor the natural digestive changes taking place in the intestine. In the small intestine no one form appears to be constantly present, but the flora varies from time to time according to the character of the diet. Most of the bacteria found in this portion of the canal appear to act on the carbohydrates in preference to the proteids. In the large intestine Bacillus coli is constantly present, and although ordinarily harmless, may at times vary much in virulence and in the toxicity of its products. To what extent these normal bacteria may induce disorder has not been fully determined. Their undue growth is held in check in the stomach by the presence of free acid and by the process of digestion proceeding with due rapidity; in the intestine, by the biliary acids, by the acids resulting from the decomposition of fats, and possibly by other digestive secretions, and also by the normal rapidity of intestinal

digestion and absorption. Disturbances of digestion and motility may antagonize or inhibit these preventive measures and either favor an excessive growth of such bacteria as may exist in the canal, or bring about a marked increase in their virulence. Under these conditions irritative conditions of the mucosa will surely develop.

With the introduction of new forms, and especially with the introduction of pathogenic bacteria, much disturbance often arises; but not always, for such may be either destroyed or rendered inert by the same natural defences which check the undue development of the normal flora. There is also considerable experimental and clinical evidence to prove that the normal bacteria of the intestine are distinctly useful in protecting the individual against new invaders.

In considering the etiology of loose evacuations from a clinical standpoint, we note first that loose evacuations may be occasioned by the mechanically irritating character of the food. It is generally recognized that coarse foods, fruits and vegetables containing much cellulose, berries and small fruits containing numerous seeds, by mechanical irritation of the mucosa stimulate peristalsis, and may, under conditions of increased susceptibility, lead to loose movements. The ingestion also of an unduly large amount of fatty food, such as oil and butter, may lead to increased peristalsis with the appearance of the unabsorbed fat in the motions. More important, however, as an etiological factor in the production of loose movements, is a faulty performance of the gastric functions. Both defective secretion and defective motility favor fermentation in the gastric contents and lead to the development of irritating organic acids and gases, and also to a great increase in bacterial growth. Hypersecretion with marked hyperacidity of the gastric contents may, by the discharge of extremely acid chyme into the duodenum, neutralize the normal alkalinity of the contents of the small intestine, and by so doing inhibit the action of the pancreatic enzymes, irritate the intestinal mucosa, and interfere with absorption.

The exact effect on peristalsis of intestinal indigestion due to deficiency in the secretion of the bile and pancreatic juice cannot be stated positively. Clinical and experimental studies have given contradictory results in the experience of different observers. Under normal conditions both appear to be secreted in quantities much in excess of actual requirements. A marked deficiency in the pancreatic secretion undoubtedly interferes with the splitting up of the fatty elements of the food, and consequently with their absorption and assimilation; it also interferes with the necessary changes which the proteid elements have to undergo to fit them for absorption. Any deficiency in the bile intensifies this condition as regards the fats. Experiments show that when the bile is absent, from 60 to 80 per cent. of the fats escape absorption, but digestion of the carbohydrates and proteids is only slightly interfered with. Of the succus entericus we know little.

In all conditions of impaired functional activity of the digestive secretions the development of a large bacterial flora promptly occurs, in part owing to the tardiness of the digestive process, and in part to the increased amount of nutritive material escaping unabsorbed into the lower portions of the intestine and affording a richer culture medium. Such development generally leads to loose movements associated with a varying group of other symptoms, but in the present state of our knowledge, we cannot definitely state that any one form of bacterial intoxication is the specific and only cause of any one particular group of symptoms.

In these conditions of impaired intestinal digestion as the result, in part, of defective absorption, in part, of the irritating changes taking place in the bowel contents reacting on a mucosa rendered unduly sensitive, and in part, of the direct action of bacteria or their toxins, peristalsis is generally unduly stimulated and the semifluid contents are hurried along without due inpissation.

Again, the clinical investigator must always bear in mind that in some individuals the intrinsic nervous mechanism of the intestines is particularly susceptible to impulses received from the large nervous centres in the cord and cerebrum. Emotion, worry, dread of any kind, and especially sudden fright, may evoke imperative loose movements: in some by increasing motility, in others by leading to profuse watery secretion, which by some physiologists has been likened to the sudden increase in the amount of saliva secreted under nervous influences. Chilling of the lower extremities may act in the same way. The morning diarrhœa of neurasthenic individuals is due apparently to some temporary irritable condition of the lower bowel, inducing one or two early morning evacuations which empty the lower bowel, and then do not recur for the day. Diarrhœas attributable to this excessive nervous irritability are not infrequently observed in Graves' disease, in hysteria, and in neurasthenia.

Loose evacuations are occasionally due to a vicarious elimination of toxic products accumulating in the blood. It has been recently shown that the toxin of Bacillus dysenteriæ injected hypodermically is eliminated by the large epithelial cells of the colon and is a potent cause of the diarrhœa. A diarrhœa of elimination may occur in many infectious fevers, in septicemia, in pneumonia, and in malaria. The diarrhœa of Bright's disease is well known to all. In gouty attacks any checking of the arthritic symptoms may induce diarrhœa. Sudden alteration of temperature may also induce diarrhœa, due in some measure to a sudden checking of the perspiration, and in part to reflex nervous influences.

Lastly, we may meet with loose evacuations due to disturbances of the blood or lymph circulations, such as the congestion associated with a failing right ventricle, or with a blocked thoracic duct.

CHRONIC CONSTIPATION CLINICALLY CONSIDERED.[1]

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There is no single condition that is more often the cause of semi-invalidism than chronic constipation; no organ in the body is exempt from its far-reaching influence, although the effects produced are mainly concentrated on the nervous system and demonstrated in certain disturbances of general metabolism. No attempt will be made to enumerate the manifold symptoms due to constipation; but a few of the changes which take place and are observed clinically may be worthy of emphasis. These may present themselves as purely local conditions or as systemic or reflex disturbances. Patients often come complaining of vague digestive troubles, such as pain, pressure, or discomfort in some particular region or over the entire abdomen. On careful questioning the clinician soon learns that these symptoms entirely disappear after a thorough movement of the bowels nas taken place. In this class of cases the nervous phenomena are so pronounced that, despite the existence of organic changes, which may perhaps be overlooked, these patients are often called neurasthenics; and they drift from one physician to another, soon falling an easy prey to the quack and patent medicine man.

What is meant by chronic constipation? A well-known dictionary defines constipation as "a state of the bowels in which the evacuations do not take place as frequently as usual, or are inordinately hard and expelled with difficulty." The study of a number of text-books, with a view to determining the factor involved in the first part of this definition, namely, the normal period after which the food residues should be evacuated in a healthy individual, failed to disclose any satisfactory data. The writer, therefore, concluded to obtain first-hand evidence on this important physiological problem, in the following way:

Three capsules each containing 10 grains of lampblack were given to various individuals at different times during the day, and the number of hours required for the appearance of the black in the stools was noted. The results of observations on thirty individuals indicate an interval varying from twelve to fifteen hours until the lampblack could be identified in the feces. The following table is appended to show a few of the results obtained.:

[1] Read at a meeting of the American Therapeutic Society, New Haven, May 7, 1909.

Sex.	Age.	Occupation.	Habits.	Capsules taken.	Black in stool.	Time. Hours.
Male	36	Physician	Active	8.00 A.M.	7 00 P.M.	11
Male	32	Merchant	Active	8.00 A.M.	9.00 P.M.	13
Male	28	Student	Active	6.00 P.M.	9.00 A.M.	15
Male	27	Student	Active	7.00 P.M.	10.30 A.M.	15½
Male	24	Student	Active	9.00 P.M.	10.30 A.M.	13½
Male	23	Student	Active	7.30 P.M.	9.30 A.M.	14
Male	24	Student	Active	7.00 P.M.	8.30 A.M.	14½
Male	22	Student	Active	7.00 A.M.	8.00 P.M.	13
Male	24	Student	Active	8.00 A.M.	9.30 P.M.	13½
Male	24	Student	Active	10.00 P.M.	12.30 A.M.	14½

All of the persons examined were accustomed to have a movement of the bowels in the morning. The experiment shows that, without exception, lampblack taken with the evening meal had reached the end of the alimentary tract by the following morning. Black taken with breakfast usually reappeared the following morning; but in individuals accustomed to defecate later in the day, it not infrequently made its appearance in the evening hours. From this it becomes evident that the common practice of civilized man to discharge the feces in the morning is based on the physiological experience that the food and digestives residues of the previous day's intake have had abundant opportunity to traverse the alimentary tract.

Three clinical varieties of constipation are familiarily described, namely, the atonic, the spastic, and the fragmentary. I shall discuss only the first two of these, believing, as I do, that the fragmentary variety is merely a manifestation of the spastic form.

The atonic type is the first stage of chronic constipation, and is due to an atony of the muscles of the large intestine, usually induced by the use of purgatives. There is no doubt that lack of tone of the intestinal musculature plays a prominent part in this form of constipation, but it has been pointed out by Schmidt and Strassburger that a very complete digestion and absorption of the food in the intestine is also a prominent factor, in that it tends to deprive the alimentary residues of their usual bulk and semisolid texture. In the atonic form of constipation the stool is hard and dry; the caliber is usually very large. No specific symptoms are in evidence. There is no pain or flatulence. After this manifestation has existed for a number of years, there results a catarrhal condition due to the irritating effect of the impacted fecal masses upon the intestinal mucosa.

The conditions here outlined are frequently succeeded by a hypertonicity of the musculature of the intestine, giving rise to the variety known as spastic constipation. This form is usually accompanied by flatulence and a catarrhal condition of the intestine. Spasm of the transverse colon occurs, causing a retention of firm masses of feces, with resulting fermentation. Colic is a frequent symptom, and in severe cases large amounts of mucus are passed. In spastic consti-

pation the rectum is usually empty, while in the atonic variety the opposite condition prevails. On examination the transverse colon can be palpated, rolling beneath the fingers as a hard cord. Patients often complain of pain in this situation. In contradistinction to the atonic form, the stool is of small caliber, and is at times even ribbon-shaped. In many cases small, hard, dry segments are passed, initiating the so-called "fragmentary type." Oftentimes the organism attempts to get rid of the retained feces by causing violent contractions of the intestines, with a resulting mucous colic or diarrhœa. This may alternate with constipation.

It may not be easy to understand how temporary loss of memory, albuminuria, convulsions, numerous skin affections, and many other functional and organic changes can be caused by chronic constipation, but that they do occur is the common experience of the observing clinician. Many believe that the absorption of toxic products, the formation of which is facilitated by the fecal retention, is the cause of these numerous symptoms. This theory, however, is by no means universally accepted. Numerous cases are cited in which healthy individuals rarely have a movement of the bowels more than once in forty-eight hours, or even longer, yet suffer no unpleasant symptoms. While this may be true, it is an accepted clinical assumption that oftentimes an intestinal absorption of toxic compounds, formed in the gastro-enteric tract, originate changes in the liver, kidneys, brain, and bloodvessels.

In a number of patients whose only obvious ailment was chronic constipation, I have repeatedly observed an excretion of albumin, which promptly disappeared as soon as regular movements of the bowels were initiated. A few cases have come under my observation in which temporary disturbance of psychic functions has occurred, due, no doubt, to habitual constipation. It is a common experience to see various skin affections disappear after proper diet and a daily movement of the bowels. It is not unreasonable to suppose that chronic constipation is often the cause of arteriosclerosis. The constant absorption of gastro-intestinal toxins introduces an irritant for the bloodvessels, and thereby may cause an abnormal growth of fibrous tissue. I believe, therefore, that the auto-intoxication theory is one that deserves a great deal of consideration. In localities where malaria is prevalent this disease is often regarded as responsible for the malaise, headache, giddiness, chills, fever, etc., in patients whose only ailment is that affliction of modern civilization, chronic constipation.

For the successful treatment of constipation it is exceedingly important to ascertain the factors producing the condition. A few of the most frequent causes of constipation are the following: (1) Dietetic errors, a too perfect absorption of food substances; (2) gastrogenic constipation, increased acidity of the gastric juice; (3) irregular habits, the outcome of carelessness; (4) insufficient

exercise, sedentary habits, and obesity; (5) nervous influences, hysteria, diseases of the nervous system; (6) drugs; and (7) local obstructions, stenosis of the intestinal tract due to neoplasms, etc.

Hyperacidity of the gastric juice is an important etiological factor, resulting in the so-called cases of gastrogenic constipation. In a series of 100 cases of habitual constipation, taken from my records, without any attempt at selection, 87 cases showed an increased acidity, after an Ewald-Boas test breakfast. On the other hand, in diarrhœa, an analysis of the stomach contents regularly showed a decreased acidity. Constipation due to hyperacidity has been explained by Strassburger, who believes that the strong disinfectant influence of the increased hydrochloric acid causes a more perfect disintegration and sterilization of the food, thereby diminishing the number and retarding the growth of intestinal bacteria. Thus, the putrefactive changes in the bowels are decreased to such an extent that the natural irritation is removed, causing a lessened peristalsis, with resulting constipation. The microscopic examination of the excrement usually discloses the residue to be mostly detritus.

This brings me to the consideration of diet as a cause of constipation. The unusual dryness of the stools has been explained by a too perfect absorption of the foodstuffs and water. The chemical investigations of Schmidt confirm this condition, as he found that the fat, cellulose, and even nitrogen content were markedly diminished.

Studies made in the laboratory of Prof. Lafayette B. Mendel, of Yale University, have shown that whereas ordinary carbohydrates (starches and sugars) are very perfectly utilized in the alimentary tract of man, a considerable number of unusual carbohydrates, such as occur in many seaweeds, etc., are not attacked by the digestive enzymes. Experimenting with agar agar, for example, he found that the greater part was excreted in the feces unchanged. As agar agar absorbs water readily and retains it, and as it is able to resist the action of intestinal bacteria, as well as the enzymes, its value in the treatment of chronic constipation was suggested to Prof. Mendel. Such compounds might give texture to the stools without introducing any objectionable products of decomposition. In Mendel's experiments in which agar agar was fed, it could be recovered readily from the stools. The effect on the total mass of feces passed is noteworthy. The agar easily retains water in the alimentary residue and prevents the formation of the scybalous masses so characteristic of spastic constipation. It was this property of giving a soft consistency to the rectal contents, together with the resistance of the agar to bacterial decomposition, with the production of gases or other noxious products, which led to the suggestion of its use in the dietotherapy of constipation.

In order to test Mendel's theory clinically, I have systematically given agar agar to patients suffering from chronic constipation. Thirty such patients have been under observation. These were

from all walks of life; their ages varied from fifteen to eighty-three years. The majority of them were unable to have a movement of the bowels without the aid of medicine. The agar was taken morning and evening, beginning with 15-gram doses, and in most cases was eaten with milk or cream, the same as a modern breakfast food. In all but two of the cases, regular movements of the bowels began after the agar had been taken for from one to three days. In the successful cases after regular movements of the bowels had commenced, the agar was gradually reduced. The duration of treatment varied from one to three months. The following will serve as an illustration:

A clerk, aged thirty-one years, consulted me for the first time on March 8, 1909. His most prominent symptoms were headaches, accompanied by temporary lapses of memory, when bowel movements failed to occur. The clinical diagnosis was spastic constipation. The patient was given agar agar, and after three days' treatment regular movements of the bowels began. As improvement continued the amount of agar agar was gradually reduced to 5 grams per day, which is now sufficient for daily evacuations. During the course of treatment, he was unable to procure the agar for four days, but in this interval normal bowel movements occurred for two days.

Another case illustrates the results achieved in the atonic variety The patient, aged eighty-three years, has not had a normal movement of the bowels in twenty-five years without the use of drugs. He has been taking agar agar for six weeks, beginning with 15-gram doses twice daily. After three days' treatment regular bowel movements occurred. The dose has been gradually reduced to 10 grams daily. A further reduction of the amount of agar in this case failed to produce results.

The effect of the agar agar on the stool in the illustrated cases confirms Prof. Mendel's theory. In all cases there was a pronounced increase in the volume of feces passed, and instead of the small, hard, dry scybalous masses, the evacuations were well formed and of a dough-like consistency. In a few patients the agar in 15-gram doses produced a diarrhœal stool. When the dose was reduced normal evacuations returned. Clinical experience with agar agar tends to show that the rational treatment of constipation resulting from a too complete absorption of food substances and water is one of diet rather than of drugs. It is well known that the management of constipation by the use of drugs affords only temporary relief, and many times induces an impaired tonicity of the intestinal musculature, thereby aggravating the condition. The important factor in dietary treatment is to bring about a condition in which a perfect selection of foods would avoid the hardness and dryness of the feces in the intestines.

The foods to be selected and the combinations necessary for

individual requirements are so varying and manifold that usually only a long series of experiments, differing in each case, can possibly bring about ideal results. Here by the use of agar agar, in itself a simple carbohydrate, taken from seaweed, clinical as well as laboratory experiments have demonstrated the possibility of transforming the hard and dry solidity of the feces into a condition of softer consistency, so that the intestinal contents develop their own agency necessary to produce a healthy, normal evacuation.

My experiments may not be extensive enough to arrive at final conclusions, but the fact that the results obtained are consistent and continuous, unlike the diminishing and aggravating effects of drug treatment, leads us to look for great possibilities from the use of agar agar or similar substances in the treatment of habitual constipation. No claim is made that it will produce permanent cures, but surely no objection from any professional standpoint can be urged against the use of something, harmless in itself, which brings about a condition that approximates natural functions.

THE LOCATION OF THE CARDIAC APEX BEAT.

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The easy recognition of certain auscultatory signs naturally distracts from other methods of physical examination of the heart. The beginner is known by the zeal with which he employs the stethoscope and dwells upon murmurs and rales; unfortunately, many never outgrow this stage, but use auscultation as a short cut to diagnosis. While recognizing the value of auscultation, we should not be led or misled by it and underestimate that of other diagnostic procedures. I desire to call attention to certain points in regard to the so-called apex beat of the heart.

The size and position of the heart are determined by: (1) The location of the apex as ascertained by inspection, palpation, or approximately, when these fail, by auscultation; (2) the area of relative cardiac dulness; (3) orthodiagraphy,[1] or radiography of the heart by a special apparatus, which employs only perpendicular rays, and so does not distort the cardiac outline. In describing the physical signs of the heart, no point is more loosely dealt with than the so-called apex beat. In some of the standard text-books one finds this term applied to the point of maximum visible pulsation in the

[1] Moritz, Münch. med. Woch., xlvii, 992.

lower portion of the precordium; in others, to that of maximum intensity of palpable impulse; while a few emphasize the most distant point of pulsation. In the records made by hospital internes one frequently finds noted P. M. I. (point of maximum intensity) so near the median line in a case of cardiac hypertrophy, especially of the right ventricle, as to show that the point mentioned is no indication of the situation of the apex. That many hospital internes seem so devoted to the term "P.M.I." shows that they have either been taught to regard the point of maximum impulse as the important point to localize or have so interpreted their instruction. Having noticed the emphasis laid upon the point of maximum intensity by medical students, hospital internes, and practitioners in New York City, I have investigated this point in all the text-books and manuals of medical and physical diagnosis and diseases of the circulatory system published since 1895, and some of earlier date, and found in the library of the New York Academy of Medicine. The contrast between the views of foreign and of the majority of these American writers is very marked.

In the text-book of A. L. Loomis,[2] we read: "When from any cause the impulse cannot be seen, its position must be determined by palpation." From this one would infer that seeing an impulse would obviate the necessity for palpation. As the visible impulse of the normal heart is not infrequently produced by a portion of the cardiac wall some distance internal to the apex, the impulse so spoken of is evidently the "point of maximum intensity," and this author, therefore, seems not to lay stress upon the observation of the apex beat as important in determining the size of the heart. That Hare[3] applies the term apex beat to the point of maximum visible pulsation is evident, for, after speaking of the apex beat as located two inches to the left of the sternum, he says: "Displacement to the right is due to adhesions and to hypertrophy and dilatation of the right ventricle, so that the apex beat is felt in the epigastrium or against the edge of the sternum." That, in employing the term apex beat, he refers to the point of maximum intensity is obvious, since in a case of hypertrophy and dilatation of the right ventricle the cardiac apex is really situated farther to the left than in a normal chest. So also Anders[4] says: "The precise apical pulsation may be localized by noting its point of maximum throb." However, he adds: "The extreme left border of the apical impulse in the fifth interspace extends nearly an inch beyond the actual maximum throb of the apex." He thus, emphasizes the manifestation of the site of the portion of the ventricular wall most prominent anteriorly rather than the extent of the ventricle toward the left, which is the important fact to be ascertained, since the situation of the apex beat is sought for in

[2] Lessons in Physical Diagnosis, 1899.
[3] Practical Diagnosis, 1907.
[4] Physical Diagnosis, 1907.

order to determine the extent of the heart toward the left and downward. His second statement, however, seems to show that he recognizes, although he fails to lay the chief emphasis upon, this important point. Da Costa,[5] Le Fevre,[6] and many others omit the term point of maximum intensity. Musser[7] says that a circumscribed area that rhythmically rises and falls is known as the apex beat, although it is a portion of the heart inside the true apex that strikes the chest wall. He makes no mention of the point of maximum intensity, and speaks of other areas of pulsation over the precordia as "new impulses." Butler[8] shows by his definition of the apex beat that he does not lay stress upon the maximum visible pulsation as Hare appears to do. After stating that the apex beat is somewhat internal to the apex formed by the ventricles, and that if there is much hypertrophy the enlarged and rounded heart strikes the chest wall, he adds: "The apex beat should be considered to lie at the point where the finger appreciates a distinct thrust from within." Another work which looks deeper than the visible pulsation is that of Gibson and Russell.[9] These writers say: "The normal apex beat is produced by the impulse of the apex of the left ventricle, but the term apex beat is very loosely applied, often used to denote the part of the precordia where pulsation happens to be most marked. It would seem better if, instead of stating that the apex beat is in such and such a space, it were stated that pulsation is visible in such and such spaces."

The best presentation of the subject in American works is that of R. C. Cabot,[10] who states clearly: "The maximum cardiac impulse is not due to the striking of the apex of the heart against the chest wall, but to the impact of a portion of the right ventricle. The importance of this fact is this: When we are trying to localize the apex of the heart, in order to determine how far the organ extends to the left and downward, it will not do to be guided by the position of the *maximum* impulse, for the apex of the heart is almost always to be found three-fourths of an inch or more farther to the left. This may be proved by percussion. The true position of the cardiac apex thus determined corresponds usually not with the *maximum* impulse, but with the point farthest out and farthest down at which *any rise and fall* synchronous with the heart beat *can be felt.*" It would seem more accurate to modify this statement to read: The point farthest downward and to the left at which a *distinct forward thrust of the heart* can be felt. This excludes the rise and fall of the chest wall, as a whole, which can often be seen and felt beyond the palpable thrust of the apex in cases with a heaving impulse which raises the chest wall. By careful palpation with a proper degree of pressure the limit, to the

[5] Principles and Practice of Physical Diagnosis, 1908.
[6] Physical Diagnosis, 1905.
[7] Medical Diagnosis, 1904.
[8] Diagnostics of Internal Medicine, 1901.
[9] Physical Diagnosis, 1893.
[10] Physical Diagnosis, 1905

left, of the clinical cardiac apex can often be distinguished as definitely as the edge of an enlarged spleen, provided it lies in an interspace and not behind a rib. We must acknowledge that the anatomical apex, formed by the tip of the left ventricle, being situated in a plane posterior to that of the right ventricle, comes into direct contact with the thoracic wall only when its apex is situated far enough to the left to strike the lateral wall of the chest.

Dietlen[11] says that the apex beat is almost always felt one interspace higher than the apex of the silhouette obtained by orthodiagraphy, or if the apex beat is under a rib, at least the width of one rib higher. He explains this by stating that the apex beat is felt in systole, while the silhouette shows the cardiac outline during diastole, and that while the heart changes only slightly in outline during the period of contraction and relaxation, such changes are greatest at the apex. It is the clinical apex, that portion of the heart wall situated normally about three-quarters of an inch internal to the anatomical apex, and the active cause of the palpable apex beat, that is of prime importance, since we must largely rely upon it for determining the size of the heart, orthodiagraphy being unavailable for routine examinations.

In marked contrast with the loose descriptions of some American text-books, apparently copied one from another, are the definitions almost invariably given in the works of German and British writers. Brugsch and Schittenhelm,[12] Ebstein,[13] Geigel,[14] Guttman and F. Klemperer,[15] and G. Klemperer[16] all define the apex beat as the portion of the palpable cardiac impulse situated farthest to the left and downward. As exceptions, we note Sahli,[17] who gives the Delphic definition of the apex beat as the visible and palpable lifting of the chest wall in the region of the apex; Edlefsen,[18] who appears to lay no special stress upon the extent of the heart to the left and downward; and Romberg,[19] who says that in cases with diffuse apex beat the apex is to be considered as situated at the point of strongest pulsation, or, if there is no point of maximum intensity, at the point farthest to the left.

Among the English and Scotch writers, Brown and Ritchie,[20] Sansom,[21] Sawyer,[22] and James MacKenzie[23] state definitely that the

[11] Deutsch. Arch. f. klin. Med., Band lxxxviii, S. 88.
[12] Lehrbuch der klinischer Untersuchungsmethoden, 1908.
[13] Leitfaden der ärztlichen Untersuchung., 1908.
[14] Lehrbuch der klinischen Untersuchungsmethoden, 1895.
[15] Ibid., 1904.
[16] Grundriss der klinischen Diagnostik, 1896.
[17] Lehrbuch der klinischen Untersuchungsmethoden, 1908.
[18] Lehrbuch der Diagnostik der inneren Krankheiten, 1899.
[19] Lehrbuch der Krankheiten des Herzens und der Blutgefässe, 1906.
[20] Medical Diagnosis, 1906.
[21] Diagnosis of Diseases of the Heart and Thoracic Aorta, 1892.
[22] Physical Signs of Diseases of the Thorax and Abdomen, 1908.
[23] Diseases of the Heart, 1908.

apex beat is the lowest and most external point of the cardiac impulse. Poynton[24] emphasizes the idea in these words: "In all cases a note should be made of *the extreme limit to the left* at which the impulse is palpable, and the maximum of the beat not alone relied upon."

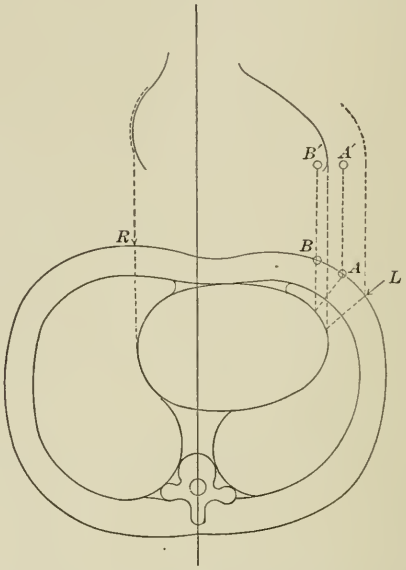
Barié[25] lays little stress upon the determination of the situation of the apex beat by palpation, on the ground that inspection is usually sufficient. He considers palpation as indispensable for finding the point of maximum intensity in cases of diffuse apex impulse.

Among the most important recent studies of the heart are those of Moritz,[26] and Dietlen,[27] who particularly oppose the determination of the size of the heart by the position of the apex beat and the left border of relative cardiac dulness, on the ground of their orthodiagraphic observations. They find that these physical signs, as determined by themselves, tend often to indicate a larger heart than really exists. Thus, Moritz[28] says: "By strong percussion one may very easily pass considerably beyond the left border of the heart," and, "the same is true if one uses as the limit of the left border of the heart the outer extremity of the apex beat, especially with vigorously pulsating hearts." (See figure.) Light percussion of the apical region and interpretation of the term apex beat, not as the point of maximum impulse or the most external portion of pulsation of the chest wall, but as the point farthest downward and to the left at which a distinct forward thrust of the heart can be felt, might obviate many of these discrepancies.

The importance of localizing the apex beat by palpation is that it is one of the two easily available methods of determining the extent of the heart to the left, the other, percussion, usually being of great value, but failing in cases with pericardial effusion, and in those in which the adjacent pulmonary resonance is obscured or lost through the presence of pleural thickening or effusion or pulmonary consolidation, through the presence of large breasts, or by the situation of the apex so far to the left, from any cause, that percussion elicits dulness of the lateral surface of the heart to the left of the apex. In such instances definite localization of the apex beat by palpation is of particular value, in the absence of deformities of the thorax, in determining the size of the heart or its displacement, the right border being located by percussion of the relative cardiac dulness. When an apex beat is visible it is usually even more definitely palpable, since firm pressure in an interspace may enable the observer to detect the impact of an apex situated so far from the chest wall as to strike it very lightly, especially during recumbency. In this connection it is important to emphasize the change in the relation of the heart to the chest wall, which occurs in moving from the recum-

[24] Heart Disease and Thoracic Aneurysm, 1907.
[25] Traité Pratique des Maladies du Cœur et de l'Aorte, 1900.
[26] Deutsch. Arch. f. klin. Med., lxxxi, 1; lxxxii, 1.
[27] Ibid., lxxxviii, 55.
[28] Münch. med. Woch., xlix, 1.

bent to the upright position. The cardiac apex is not only lowered,[29] but the apex, which is carried backward by gravity during recumbency, assumes a position nearer to the chest wall. For this reason palpation of the apex beat for the purpose of ascertaining the position of the apex is often easier when the patient is standing or sitting; occasionally the apex approaches the chest wall sufficiently to be palpable only when the patient bends forward. Percussion of the area of relative dulness must, of course, be performed while in the



Transverse section of the thorax (diagrammatic) seen from below, with the projection of the true outline of an enlarged heart (Moritz, Münch. med. Woch., xlix). Heavy percussion at *R* and *L* gives the area of dulness indicated by dotted lines in the projection and exaggerates the apparent size of the heart. The impulse felt at *A* and projected at *A'* is confused with the forward thrust of the apex felt at *B* and projected at *B'*, the clinical apex beat.

same position, in order that the outline of dulness, which also varies with change in position, may be considered in connection with the situation of the apex.

SUMMARY. The object of locating the clinical apex beat of the heart is to determine the size and position of that organ. The point of maximum impulse frequently does not define the site of the cardiac apex. The point of maximum impulse, either visible or palpable, may be produced by a portion of the heart wall at some distance from the apex. Except in cases in which the visible and palpable impulses coincide, as they usually do with a normal heart,

[29] Moritz, Dietlen, loc cit.

it is better to state simply that pulsation is visible in certain interspaces at a given distance from the median line. It is frequently impossible to determine the left border of the heart by percussion, on account of changes in the resonance of the adjoining area due to large breasts or intrathoracic conditions, or when the apex is carried so far toward the left by hypertrophy or by displacement of the heart, as a whole, that percussion, unless extremely light, tends to bring out dulness of the lateral surface of the heart. Auscultation, as a means of locating the apex, is at best a makeshift in the event of the failure of other methods.

The most valuable clinical method of locating the cardiac apex is the palpation of its impulse when this can be detected in an interspace. The apex beat should be considered as *the point farthest downward and to the left at which a distinct forward thrust of the heart can be felt.* The clinical apex so located, being close to the anatomical apex, is of real value in estimating the size or relative position of the heart. Because of the influence of posture upon the area of relative cardiac dulness, and the situation of the apex beat, it is advisable, for comparison with subsequent observations, to record whether the patient was examined in the erect or in the dorsal recumbent position.

ORTHODIAGRAPHY IN THE STUDY OF THE HEART AND GREAT VESSELS.

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The *x*-rays, as ordinarily used, have proved of distinct value in the study of the intrathoracic vascular system, especially in the detection of aneurysms, but only to a small degree have they aided investigations of the heart itself. The difficulty before the perfection of the orthodiagraph has been the fact that, because of the divergence of the rays, the shadow of the object observed was enlarged. This exaggeration of outline is increased in direct proportion to the nearness of the object to the tube and its distance from the screen upon which the shadow is thrown. Thus, in order to estimate the size of the object under observation, an elaborate system of measurements is necessary, and even then accuracy is extremely difficult.

While it is true that the distortion of the image may be greatly diminished by taking the picture at long range, as it were, yet we lose the very important activity of life. Thus, with the fluoroscope the pulsations of the heart and aorta and the movements of the diaphragm serve as points of identification. Mediastinal tumors, calcareous glands, and tuberculous deposits in the lungs are not infrequently seen in close proximity to the heart and great vessels, and but for the pulsating movements of the latter could not be distinguished from them. It is the difference between the study of the living and the dead.

Description of the Orthodiagraph. The Röntgen-ray pictures are shadows, and, as the source of the rays is a point on the anticathode of the Crookes tube, these shadows would be the bases of cones or pyramids, whose apex is this point on the anticathode. The object casting these shadows would completely fill these cones or pyramids at some point between the apex and the base. Thus, as ordinarily used, skiagrams or Röntgen-ray shadows of the internal organs are always enlarged and distorted to a greater or less extent. To overcome this distortion and to outline the internal organs in their exact size, parallel instead of divergent rays should be used, and these parallel rays should be perpendicular to the reading screen. It is impossible to obtain parallel Röntgen rays directly from our Crookes tube, so that in the effort to overcome this difficulty the orthodiagraph has been developed.

The orthodiagraph, first devised by Moritz,[1] and later much improved by Levy-Dorn and Groedel,[2] is an instrument for outlining, in its exact size without distortion, any object that can be seen by the Röntgen rays. The working principle may be outlined as follows: A pneumatic pen, the centre of the anticathode of the Crookes tube, or source of the Röntgen rays, and the centre of a small fluorescent screen are so mounted on arms that they are in a straight line. These arms are attached to a carriage, which, while allowing all motions in one fixed plane, will keep this straight line perpendicular to the plane. Thus, the pencil of rays coming from the tube, through a small circular aperture in a lead diaphragm, to the centre of the screen, while it may be moved over a considerable surface, will always occupy parallel positions.

In using the machine it is only necessary to move the carriage so that the lead spot in the centre of the screen touches the edge of the shadow to be outlined; a mark is made with the pneumatic pen on the sheet of paper, which is clamped upon a drawing-board fixed behind the tube. The lead spot is then moved to a new position along this edge and a second mark made. In this way the process is continued until the entire shadows are outlined. Then, upon removing the sheet from the machine, these marks, when connected

[1] Münch. med. Woch., 1900, No. 29. [2] Orthoröntgenographie, München, 1908

will give the exact size of the object casting the shadow. The fluorescent screen may be removed from the arm to which it is attached and a pointer substituted. The pointer is so adjusted that it takes the place of the lead centre of the screen, and is, therefore, in the straight line with the centre of the pencil of rays and the pneumatic marker. It is used to outline the body and any points that the operator may wish to record upon the diagram sheet before the current is turned on.

Danger of *X*-ray Burns. The danger of burning the subject is practically nil. The tube is covered by a ray-proof shield and only the small pencil of rays in use is allowed to pass through a circular opening about 2 cm. in diameter in a lead diaphragm. As the apparatus is kept in almost constant motion, no area is exposed more than a few seconds at a time. The pencil of rays is thrown upon a fluorescent screen which is covered with a plate of lead glass, which it is claimed protects the operator's face and eyes while observing the shadow.

Advantages of the Orthograph. The satisfactory use of this instrument, as is the case with many others, such as the microscope or stethoscope, necessitates practice and a thorough familiarity with the normal. The orthodiagram, when completed, however, is intelligible to any one who takes the trouble to inform himself upon the topographical anatomy of the interior of the thorax. The lines are clearly marked and the object is outlined in its exact position with regard to its surrounding landmarks—the ribs, the sternum, the liver, the diaphragm—and its size may be determined and compared with the normal.

On the other hand, the skiagram usually requires long experience to interpret correctly. A shadowy distorted outline is often all that can be made out. This may mean much to the expert, but practically nothing to the ordinary physician, who must accept the statement of another and is often but half convinced. The fluoroscope, as ordinarily used, allows of no photographic or diagrammatic record.

Another great advantage possessed is the rapidity with which the diagram can be made when the operator has acquired some experience. Within a few minutes the complete examination is made, and the diagram is ready for inspection. The time and expense required for the development of a plate are avoided. The only delay is that necessary to allow the eyes of the operator to become accustomed to the darkness. This time varies from a few minutes to fifteen or twenty, depending somewhat upon the brightness of the day. We urge most strongly the absolute necessity of this delay, as without it the examination will be unsatisfactory.

Method of Making a Tracing. The patient may be placed in either a standing, sitting, or reclining position. The importance of keeping absolutely still must be impressed upon him, as any move-

ment of the body during the procedure will necessarily vitiate the results. The landmarks are then taken by means of the pointer before the current is turned on. They can be taken with the fluoroscopic screen, but the tube may as well be spared this unnecessary work. The midsternal line is fixed by marking the episternal notch and the tip of the xyphoid; the neck, shoulders, axillæ, and sides of the chest are outlined and the nipples (in males) marked. As many of the ribs as are desired may also be indicated. The dorsoventral illumination is the most satisfactory, and the sitting or standing posture is preferable when the subject is able to assume either of these positions. The heart's silhouette when thus observed consists of a series of curves, two upon the right and four upon the left side (Fig. 1). Upon the right, rising from the dark shadow of the liver, may be seen the "right auricular curve," (1) which is formed entirely by the right auricle. Above this is the "curve of the great vessels," (2) formed

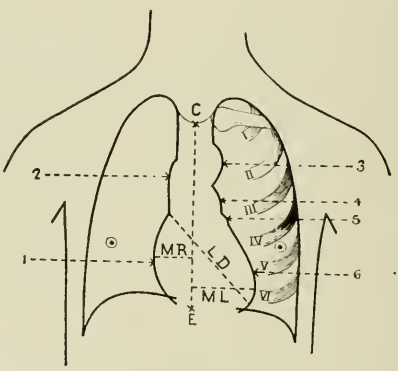

Fig. 1.—Orthodiagram of the normal heart.

by the descending cava or the aorta (in our experience this shadow is more often seen as a straight line). On the left, from above downward, is the "aortic curve," (3) formed by the arch of the aorta and its descending limb; below this is the "pulmonary curve," (4) formed by the pulmonary artery as it passes beneath the aortic arch. The next curve, the "left auricular," (5) is in our experience only exceptionally observed. It is produced in normal cases by the left auricular appendix, as only in abnormal conditions of the heart does the left auricle itself appear. The lowermost curve is the "left ventricular," (6) and is formed by the left ventricle.

It is convenient to trace the borders of the thoracic cavity (lung outlines), while the eyes of the operator are becoming accustomed to the darkness. Then the diaphragm is marked in its lowermost position during quiet respiration. The curves of the heart's silhouette are then outlined in the order indicated in Fig. 1. The right

border of the heart presents a well-marked dark shadow; the descending cava is lighter; the arch of the aorta, unless atheromatous, is often very faint, and could not be distinguished but for its pulsations; the pulmonary artery is faint, and the auricular appendix is not often distinguishable; the left border of the heart is clear and dark and in very active motion; the extreme apex is more difficult to see, because it is thin and also because its excursion of movement is large and rapid. By asking the subject to take a deep breath and hold it, the lower border of the apex can generally be outlined above the depressed position of the diaphragm. It must be borne in mind, however, that a deep inspiration alters the cardiac outline, that is, lowers the apex. The lower border of the heart is lost in the shadow of the liver, but its position can be easily estimated from the apical curve and that of the right auricle.

Observations. In order that we might properly appreciate diseased conditions, we undertook the study of a series of normal chests with the orthodiagraph. More than one hundred cases were studied, but from lack of sufficient data some of them have been omitted from our records. Our series of adults consists of 37 men, ranging in age from eighteen to fifty-five years, and 54 women ranging from nineteen to thirty-six years. Our measurements were made according to the accepted rules in this work. The method can best be understood from the diagram (Fig. 1). The total transverse diameter (T.D., the sum of M.R. and M.L.) may be considered as quite accurate, because the points from which these measurements are made are always clear and definite, but the longitudinal diameter (L.D.) is less accurate in some cases, because it is not always easy to tell where the right auricle ends and the descending cava begins. The extreme apex being thinner and in active motion renders the shadow at this point also less easy to trace.

Conditions Affecting the Heart's Size. It has been concluded from an examination of a large series of cases that the size of the normal heart depends upon a variety of factors. Thus, it depends upon the body height, the body weight, the age, the muscular development, the occupation, etc. Men, who have not attained full growth, even though of the same height and weight as adults, have smaller hearts. Further, the heart's size tends to increase in old age. Women of the same height and weight as men have proportionately smaller hearts. Finally, normal individuals presenting exactly like conditions may have different-sized hearts.

From our observations it appears that the size of the heart depends more upon the body weight than upon the body height. It was impossible in our series to work out a scale which seems to show any constant ratio between the heart's size and the body height. The body weight, on the other hand, compared very uniformly with the

heart's area. We have also constructed a similar chart from the measurements of Dietlen's[3] cases, which seems to agree with our findings.

TABLE I.—*Vertical Heart Orthodiagrams.* Male (37 Cases).

Weight pounds	Cases	M.R.	M.L.	T.D.	L.D.	
120–129	3	3.2	7.0	10.7	11.8	Min.
		3.7	7.2	10.9	12.6	Av.
		4.3	7.5	11.3	13.5	Max
130–139	5	3.5	7.5	11.0	12.0	Min.
		3.8	8.0	11.8	13.2	Av.
		4.2	8.5	12.5	14.0	Max
140–149	9	3.4	7.0	11.0	12.0	Min.
		4.0	7.7	11.9	13.4	Av.
		4.6	8.4	13.1	14.5	Max.
150–159	8	3.2	7.8	11.5	12.5	Min.
		3.9	8.4	12.3	13.5	Av.
		4.5	9.0	13.0	15.0	Max.
160–179	6	3.7	8.0	12.0	14.0	Min.
		4.0	8.2	12.4	14.6	Av.
		4.8	9.0	13.8	15.8	Max.
180–200	6	3.8	7.0	11.0	14.0	Min.
		4.2	8.7	12.9	14.7	Av.
		4.5	9.7	13.4	15.3	Max.

TABLE II.—*Vertical Heart Orthodiagrams.* Female (54 Cases).

Weight pounds	Cases	M.R.	M.L.	T.D.	L.D.	
100–109	2	3.2	6.7	9.9	12.0	Min.
		3.3	6.8	10.2	12.1	Av.
		3.5	7.0	10.5	12.3	Max.
110–119	3	3.0	7.0	10.0	11.5	Min.
		3.1	7.6	10.7	11.9	Av.
		3.2	8.0	11.1	12.4	Max.
120–129	14	2.3	6.4	10.2	10.5	Min.
		3.5	7.5	11.0	12.2	Av.
		4.2	8.6	12.2	13.8	Max.
130–139	19	3.0	6.4	9.6	11.2	Min.
		3.4	7.8	11.2	12.4	Av.
		4.0	8.8	12.6	13.3	Max.
140–149	5	2.6	7.0	10.0	12.2	Min.
		3.5	7.6	11.1	12.7	Av.
		4.1	8.3	11.8	13.2	Max.
150–159	7	3.1	7.6	10.9	12.3	Min.
		3.6	8.0	11.6	12.9	Av.
		4.8	9.3	12.8	14.2	Max.
160–175	4	3.5	6.5	10.6	11.8	Min.
		3.8	7.9	11.7	12.6	Av.
		3.8	8.5	12.3	13.0	Mean.
		4.1	9.0	12.8	13.2	Max.

[3] Deut. Arch. f. klin. Med., 1906–1907.

TABLE III.—*Vertical Heart Orthodiagrams.* Normal Cases. Males (42 Cases).

Age	Height in.	Weight Pounds	M.R.	M.L.	T.D.	L.D.	
18	62	125	3.0	7.0	10.7	11.8	Min.
32	68	150	4.0	8.1	12.1	13.7	Av.
55	74	200	4.8	11.0	14.0	15.8	Max.

TABLE IV.—*Vertical Heart Orthodiagrams.* Females (54 Cases).

Age	Height in.	Weight Pounds	M.R.	M.L.	T.D.	L.D.	
19	61	100	2.3	6.4	9.6	10.5	Min.
25	65	135	3.4	7.8	11.1	12.5	Av.
36	69	175	4.8	9.3	12.8	14.2	Max.

TABLE V.—*Dietlen's Horizontal Orthodiagrams.* Normal Cases.

	M.R.	M.L.	T.D.	L.D.
Average of 156 males	4.3	8.9	13.2	14.2
Average of 58 females	3.6	8.5	12.1	13.2

TABLE VI.—*Groedel's Vertical Orthodiagrams.* Normal Cases.

	M.R.	M.L.	T.D.	L.D.
Average of 60 males	4.6	8.4	13.0	14.0
Average of 54 females	3.9	8.0	11.9	12.9

TABLE VII.

	M.R.	M.L.	T.D.	L.D.	Area 70% of T.D. x L.D.
	M. F.	M. F.	M. F.	M. F.	M. F.
Dietlen's . .	4.3 (3.6)	8.9 (8.5)	13.2 (12.1)	14.2 (13.2)	131 (111)
Groedel's . .	4.6 (3.9)	8.4 (8.0)	13.0 (11.9)	14.0 (12.9)	127 (107)
Authors' . .	4.0 (3.4)	8.1 (7.8)	12.1 (11.1)	13.7 (12.5)	116 (97)

EXPLANATION OF TABLES AND CHARTS. Table I shows the heart measurements in our series of 37 men, whose average age was thirty-two years, average weight 150 pounds, and average height 68 inches, graded by weight. Table II shows the same arrangement of our series of 54 women, whose average age was twenty-five years, average weight 135 pounds, and average height 65 inches.

Fig. 2 shows graphically the increase of the heart measurements with the increase of the body weight in our series of 37 men. The upper line indicates the longitudinal diameter, the lower line the transverse diameter. These lines ascend by the scale of units given on the left of the chart, which expresses centimeters. The middle line indicates the product of L.D. by T.D., or approximately the square area of the heart's silhouette. (By a careful computation of the square area of the heart's silhouette in a number of diagrams from normal hearts, it was found to be about 70 per cent. of the product of L.D. by T.D.) This line ascends by the scale of units upon the right of the chart, which represents square centimeters. The reduction of the unit of measurement was necessary to keep this line within convenient limits.

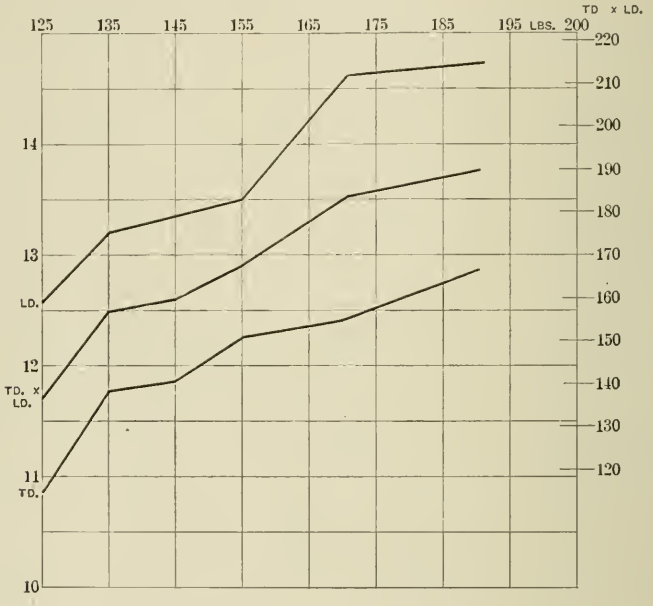

Fig. 2

It has been calculated from this chart that as the body weight of these men increased 60 per cent., the heart's area, as represented by the product of T.D. by L.D., increased 39 per cent. It is of interest to note that the size of the heart increased very rapidly up to 135 pounds of body weight, and that from that point it was more gradual.

Fig. 3 is the graphic arrangement of our 54 women. From this it would appear that as the body weight of these women increased 60 per cent., the heart's area, as represented by the product of T.D. by L.D., increased only 25.8 per cent., or far less rapidly than was the case with the men.

Fig. 4 is a graphic reproduction of Dietlen's table of horizontal

orthodiagrams of adult men grouped according to weight. The number of cases was 156, their average height was 169 cm.

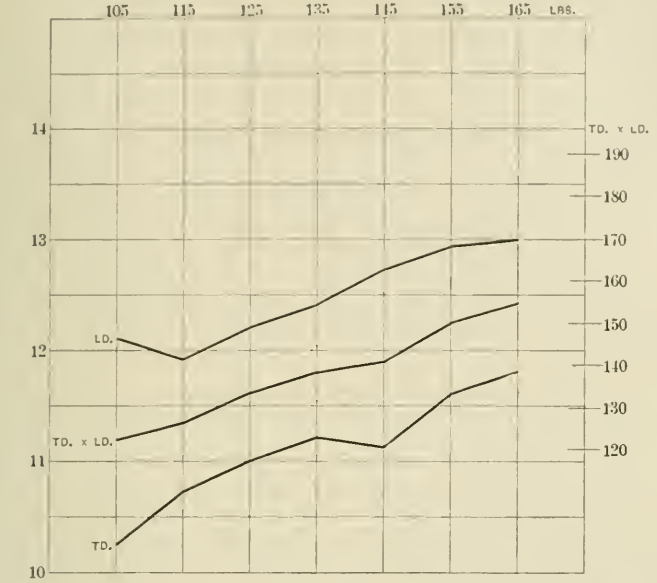

FIG. 3

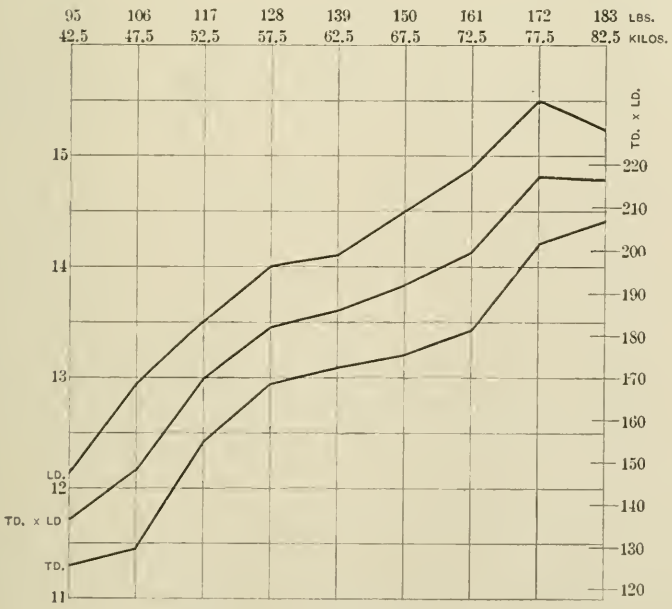

FIG. 4

(66.5 inches), and average age twenty-six years. This chart shows that as the body weight increased 60 per cent., the heart's area, as represented by the product of T.D. by L.D., increased 41 per cent., or practically at the same rate as was the case in our series of men. It is of interest to note in this chart the rapid rise in the heart's size from 95 pounds to 128 pounds, and the more gradual rise thereafter. The irregular appearance of the curve at the higher weights was probably due to the small number of men in these sections

Table III shows the maximum, minimum, and average ages, heights, and weights of our series of 37 men, given in Table I, with 5 others added later, bringing the number up to 42. Table IV gives the same arrangement of our series of women. Tables V and VI explain themselves. Table VII is compiled from Tables IV, V, and VI, and shows in compact form the results obtained by Dietlen, Groedel, and ourselves in measuring the hearts of normal adults.

It may be seen that Dietlen's and Groedel's measurements are, as a rule, greater than ours, but this may be explained, perhaps, by the class of cases observed. Our series of men was made up almost entirely of brain workers in good health, and, as it is generally acknowledged that the amount of muscular exercise has a direct bearing upon the size of the heart, these men are likely to show slightly smaller measurements. It must also be borne in mind that Dietlen's measurements were taken from horizontal diagrams, which are somewhat larger than the vertical.

Our women were, for the most part, young and healthy, leading active lives requiring more or less muscular exertion. Their measurements come nearer to those of Dietlen and Groedel, but are also somewhat smaller. It may be seen, however, that the three sets of figures vary only by tenths of a centimeter, differences, almost too small to be considered.

Percussion. One of the chief advantages derived from the use of the orthodiagraph is the aid which it gives to those who make use of percussion in heart examinations. Many clinicians depend very greatly upon the percussion boundaries of the heart in making a diagnosis, and still more in giving a prognosis in certain heart lesions. It is pretty generally conceded by those who have compared their percussion findings with those of the orthodiagraph that the former are in many instances quite accurate, and that, as a rule, percussion may be depended upon for clinical purposes. At the same time it should be borne in mind that the men who have made these tests have been especially interested in this line of work, and have, therefore, become especially proficient in percussion.

Various methods of percussion have been used by various observers, with about the same results. The method made use of in our work has been "the immediate palpatory," as it may be called. Without going into detail, we would say that the percussion boundary of the

left border of the heart was usually found to be correct, but the right border was far more difficult to locate accurately. This has not been the experience of some other observers, as they have found the right border easier to determine. In almost all instances in which we felt satisfied with our efforts at percussion, the boundaries were found to

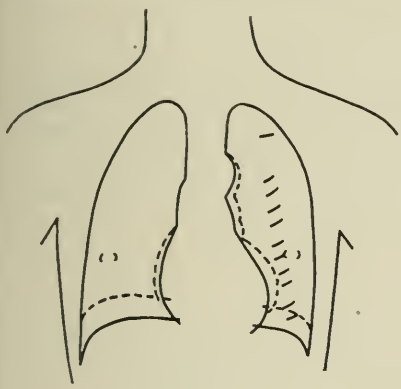

FIG. 5

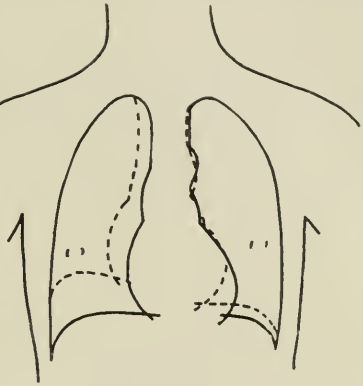

FIG. 6

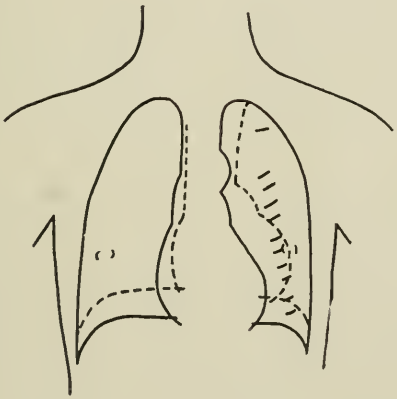

FIG. 7

FIG. 5.—The continuous line shows the position of the heart with the subject in the vertical position; the broken line shows it in the dorsal position.

FIG. 6.—The continuous line shows the position of the heart with the subject in the vertical position; the broken line shows it in the right lateral position.

FIG. 7.—The continuous line shows the position of t e heart with the subject in the vertical position; the broken line shows it in the left lateral position.

be correct, but in some subjects it was absolutely impossible to outline the heart by percussion, and this too in cases in which there was no excess of adipose tissue, or other demonstrable condition to interfere.

Figs. 5, 6, and 7 are orthodiagrams of a normal heart taken with the subject in the vertical, the dorsal, and the right and left lateral

horizontal postures. In each instance the continuous lines mark the position of the heart with the subject in the vertical position, while the broken lines show the effect of change of bodily position upon that of the heart.

The Form of the Heart's Silhouette in Different Heart Affections. It has long been known that the hypertrophy or dilatation resulting from various heart lesions produces more or less characteristic alterations in the shape of the organ. This has been demonstrated upon the living subject many times by percussion, but in the orthodiagraph we have a far more accurate means of determining the change in outline. Our studies along pathological lines have not been extensive enough to enable us to speak authoritatively, but we hope in a future publication to go into this feature more fully.

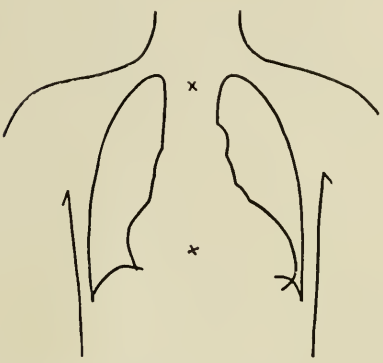

Fig. 8.—Orthodiagram of aortic insufficiency.

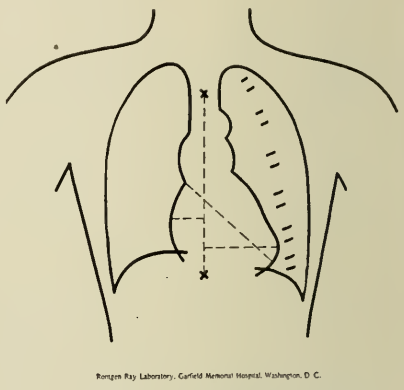

Fig. 9.—Orthodiagram of mitral stenosis.

For example, it may be seen from Fig. 8 that in aortic insufficiency the position of the heart is quite oblique, or rather foot-shaped; both diameters are greatly increased; the curve of the great vessels is more prominent, probably because of increased blood presure in the ascending limb of the arch of the aorta, or because of a dilatation of this section. The right auricular curve is prominent; the pulmonary curve is bulging; the left auricular curve is but faintly visible.

Of pure aortic stenosis we have no example, but the changes in contour are said to resemble very closely those of aortic insufficiency, only they are of less degree. The apex is more rounded, due to its increased breadth from the primary hypertrophy of the left ventricle.

Fig. 9 is from a case of pure mitral stenosis. The figure of the heart is more upright than in that of aortic disease; the curve of the great vessels is more pronounced than in the normal, and this we have observed in every case of mitral stenosis thus far examined by us. There is a decided bulging of the left auricular curve, due to the

dilatation and hypertrophy of this chamber of the heart. The left ventricular curve is relatively small, as might be expected.

Fig. 10 is from a case of pure mitral insufficiency. It is more upright than the aortic heart, but does not present quite the globular appearance which is said to be characteristic of this condition. The curve

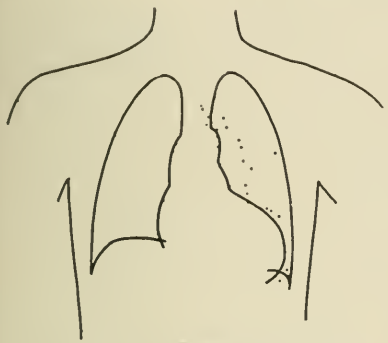

Fig. 10.—Orthodiagram of mitral insufficiency.

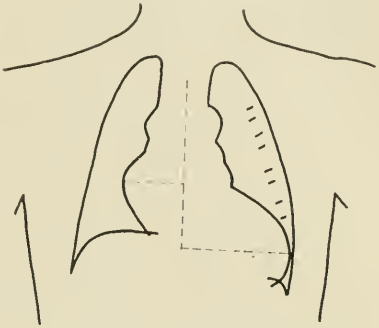

Fig. 11.—Orthodiagram of mitral and aortic insufficiency.

of the great vessels is prominent; the pulmonary curve is bulging, and the left ventricular curve has a tendency to rise higher toward the left axilla than in the normal.

Fig. 11 is a good example of combined mitral and aortic insufficiency. There is an enormous hypertrophy. The right auricular

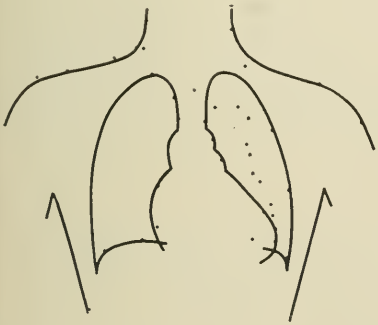

Fig. 12.—Orthodiagram of mitral insufficiency and stenosis.

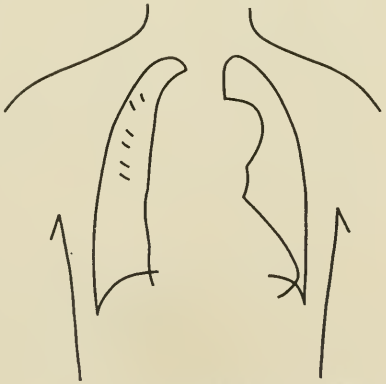

Fig. 13.—Orthodiagram of thoracic aneurysm.

curve is prominent, the curve of the great vessels is increased, while upon the left may be seen the step-like succession of bulging curves made by the aorta, the pulmonary artery, the left auricle, and, left ventricle. In other words, we have a combination of the distortions of outline produced in the conditions individually.

Fig. 12 is from a case of double mitral disease. The right auricular curve is marked, as is that of the great vessels; the pulmonary curve is bulging, and there is a decided prominence over the region of the left auricle, although it cannot be distinguished from the bulging curve of the left ventricle.

Fig. 13 shows a large aneurysm of the aorta.

Fig. 14 is an example of what has been called the "dropped" heart.

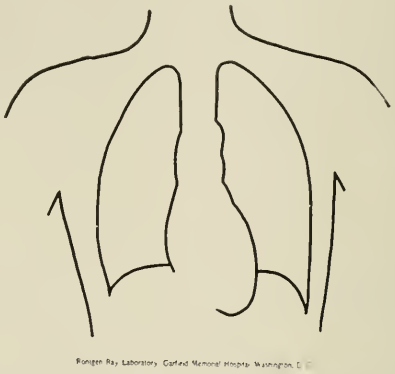

Fig. 14.—Orthodiagram of "dropped" heart.

Conclusions. While orthodiagraphy should not be looked upon in any way as a substitute for the other well-known methods of examination of the heart and great vessels, it is at the same time a valuable aid. It can be used to make fairly accurate outlines and measurements of the heart and great vessels, thus enabling us to make comparisons with the normal or with subsequent diagrams of the same case. The use of the orthodiagraph may also serve to prove whether or not the size of the heart is influenced to any appreciable degree by a single effort of exertion or by a single therapeutic or gymnastic treatment.

OBSERVATIONS ON ACUTE LEUKEMIA, WITH SPECIAL REFERENCE TO AUER'S BODIES.

By Reuben Ottenberg, M.D.,

George Blumenthal, jr., fellow in pathology, mount sinai hospital, new york.

Of the many supposed parasites that have been described in leukemia, none has been confirmed.[1] The bacterial infections which frequently occur in leukemia, and which are regarded by some as

[1] I have searched unsuccessfully for Löwit's bodies (Löwit, Central. f. Bakt., Orig., 1907, xlv, 600) in the blood, and for the spirochetes recently described by White and Proescher (New York Medical Journal, 1908, lxxxvii, 9) in the organs of one case of acute leukemia.

etiological, are undoubtedly intercurrent infections. Auer,[2] in 1903, observed certain peculiar structures in the large lymphocytes in a case of acute leukemia. The granules, crescents, and rods which he described were seen both in the fresh blood and in smears stained with various azure dyes or with Ehrlich's triacid mixture. These observations have been confirmed once by Pappenheim.[3]

Auer described three kinds of structures in the cytoplasm of the large lymphocytes—granules, crescents, and rods. Of these, the first two are indistinguishable from the azurophile granulations, now recognized as occurring regularly in lymphocytes.[4] The rods, on the other hand, have a distinct and characteristic appearance (Figs. 1, 2, and 3). They are found only in the cell body of the large lymphocytes. Generally they are single, sometimes two are seen (Fig. 4), and I have seen as many as four in one lymphocyte. In preparations stained by the Giemsa, Wright, or Hastings method they are red and remind one of the appearance of tubercle bacilli. Generally they are a little longer and more slender than the latter. Sometimes they seem nodular, generally the ends are tapering, sometimes abrupt.

Of the three cases reported here, I have seen only one myself. The other two were studied from smears in the blood collection of Dr. Libman. One of these two cases proved at postmortem to be a case of chloroma. The one case which I had an opportunity to follow was a typical case of acute lymphatic leukemia of the rapidly fatal type, with suppuration of the jaw, general glandular enlargement, large spleen, high continued fever, and multiple hemorrhages from the gums, intestines, and in the skin. The leukocytes varied between 170,000 and 225,000 in number, and the percentage of large lymphocytes was between 75 and 85.[5] Two blood cultures were negative. Cultures from the jaw during life and from the spleen postmortem showed Staphylococcus aureus. At autopsy the spleen was much enlarged (weight, 2 pounds, 7 ounces) and had numerous infarcts. There were many lymphoid infiltrations in the lungs. All the lymph nodes were enlarged and were greenish-gray in color. The bone marrow (femur) was light grayish brown in color and mushy in consistency.

The blood was examined for the rods from day to day. They were never present in anything like the large numbers in which they occurred in Auer's case (often four or five in a single microscopic field). It was generally necessary to examine one hundred to six hundred cells in order to a find a rod. No especial connection could be made out, such as there was in Auer's case, between the number

[2] Amer. Jour. Med. Sci., 1906, cxxxi, 1002. [3] Folia Hæmat., 1908, v, 347.

[4] Naegeli's statement that azure granules which are present in about one-third of normal lymphocytes are entirely absent in acute leukemia, is certainly incorrect.

[5] One interesting feature of the blood picture was the number of large lymphocytes whose nuclei looked as if dividing. One undoubted mitosis in the circulating blood was seen (Fig. 5).

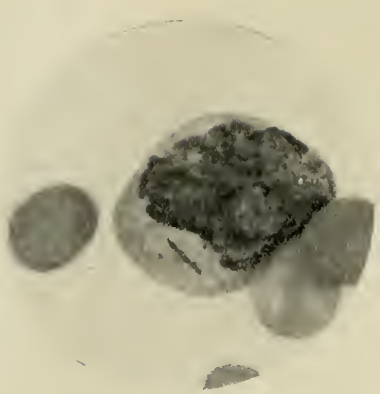

FIG. 1

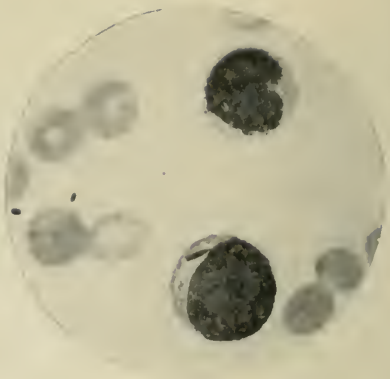

FIG. 2

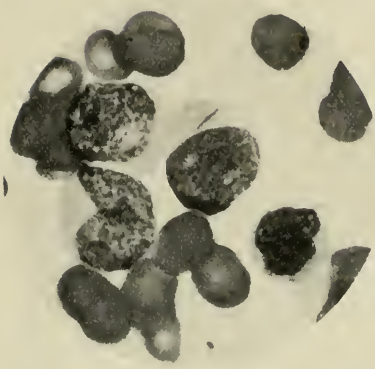

FIG. 3

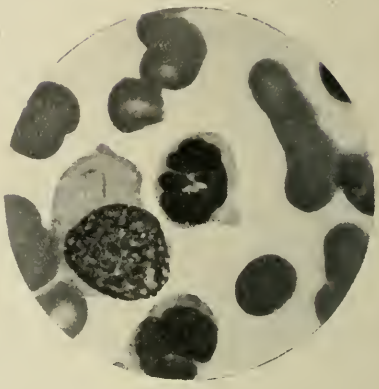

FIG. 4

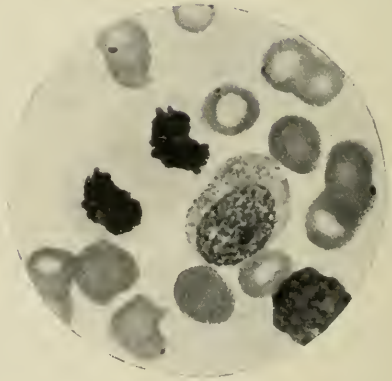

FIG. 5

FIG. 1.—Auer body in a large lymphocyte. (× 1500) The granular appearance of the rod is well marked in the original print, but not well shown in the reproduction.

FIG. 2.—A rod somewhat shorter and thicker than those usually found. (× 1000)

FIG. 3.—A slender rod tapering at both ends, distinctly nodular in character. (× 1000)

FIG. 4.—Two rods in the same lymphocyte. (× 1000)

FIG. 5.—Mitosis in a large lymphocyte. (× 1000)

of rods and the hemorrhages. On three days no rods at all were found (as also happened in Auer's case).

In the other two cases, likewise, the rods were present in small numbers only. Of these two cases, one was remarkably similar in clinical characteristics to the case described above. It was also an acute case of three weeks' duration, commencing with stomatitis and suppuration of the lower jaw, continuing with high fever and general glandular enlargement. The number of leukocytes varied between 116,000 and 130,000, the large lymphocytes between 75 and 78 per cent.

The other case proved postmortem to be a case of chloroleukemia. It presented so many interesting features as to be worth describing in a little more detail. The total duration of the illness was three months. The symptoms were those of tonsillitis, with bone pains, progressive anemia, glandular enlargements, pleural effusion, and irregular fever. The white blood cells ranged between 25,000 and 30,000, the lymphocytes composing 90 to 97 per cent. of them. The large lymphocytes were 80 to 83 per cent. At autopsy the spleen was enlarged, there were enlarged mesenteric lymph nodes (one the size of an apple), of a grass-green color; the kidneys were enlarged and showed very numerous grass-green projecting tumors, varying in size and shape from a pin-point to 1 cm. in diameter. The marrow of the tibia, the only bone examined, was pale pink. On section of the green tumors in the kidneys and in the lymph nodes, they were seen to contain not only the usual large ungranulated mononuclear cells, but also large numbers of mononuclear cells with eosinophile granulations.[6] The contrast between the blood, which contained very few eosinophilic cells, and the tumors, which were full of them, was very striking. It seems probable that the green color of these tumors (which has never been adequately explained) may be due to the eosinophile granules, which in fresh preparations have a pale greenish tinge.

In studies of this kind the control observations are of great importance. Do these rods occur in the lymphocytes in any other disease, or in health? I have examined the blood of many normal subjects, both infants and adults, as well as of many other types of disease, including myelogenous and chronic lymphatic leukemia. No rods were found. It should be remembered, however, that in the three cases of leukemia in which the rods were found it was necessary generally to search through several hundred large lymphocytes in order to find a rod. In acute lymphatic leukemia this is easy. But in normal blood or the blood of other diseases the large lymphocytes form so small a proportion of all the cells that it takes hours to see as many cells of this variety as can be seen in a few minutes in a case

[6] The occurrence of eosinophile cells in chloroma has been described by Dock and Warthin, Medical News, November, 1904, and by F. F. Leighton, Jour. Path. and Bact., October, 1907.

of acute leukemia. I have, however, examined many hundreds of large lymphocytes from a rather remarkable case of chronic lymphatic leukemia which is at present under observation. In this case the number of large lymphocytes is high, at times as high as 86 per cent., and an examination of these cells made by Dr. Weil according to the new oxidase method[7] indicates that they are of myelogenous origin. In this case the rods have not been found.

I have also recently examined the blood of a case of acute leukemia of the aplastic type in a child. Though the clinical symptoms in this case were those of a typical and very acute lymphatic leukemia, during the days which the child spent in the hospital before its death the white blood cells were never above 12,000, and the large lymphocytes 16 per cent. (though the small lymphocytes were as high as 86 per cent.) Streptococci were isolated from the blood. No rods were found.[8]

The interpretation of these rods is difficult. They might be regarded as: (*a*) Artefacts; (*b*) cell secretions; (*c*) parasites. That they are artefacts can be excluded. They have been seen in the fresh blood (Auer and Pappenheim). They occur only in one type of cell and only in one place in that cell; and they can be stained by a number of different methods. They have a definite and regular size and shape. Whatever they are, they are real. The question of these rods being products of cell activity cannot be answered definitely at present. They may possibly belong to the same order of things as the azurophile granules seen in many lymphocytes. They may be crystals. (They, of course, have nothing to do with Charcot crystals, which[9] occasionally occur not only in the tissues, but in the blood of leukemia.) The fact of their occasionally being found in vacuoles of the cell body might be regarded as being in favor of their exeretory nature. They are not likely to be products of cell disintegration, as they are usually found in well-preserved cells. There is no proof that they are parasites. Attempts made to cultivate them, from the blood of the patient, on agar, bouillon, ascitic fluid, and sodium citrate, were all negative. As the leukocytes in these nutritive media degenerated and lost their staining power the rods also disappeared.

It was hoped that some evidence on this question might be obtained from the organs postmortem, but examination of smears from the spleen, bone marrow, lymph nodes, and other organs failed to show

[7] Schultz, Ziegler's Beiträge, 1909.

[8] This case probably belonged to the very interesting group of "aplastic leukemias," of which cases are described by Emerson. (Johns Hopkins Hosp. Bull., 1907, p. 82), Hand (Arch. Ped., December, 1905), Wolff (Berl. klin. Woch., 1905, xlii, 35), and Jeanselme and Weil (Bull. et mém de la soc. méd. des hôp. de Paris, 1904, xxi, 185). It may be, however, that cases of this kind are really acute bacterial infections, and that the blood picture is secondary. "It is certain that true septicemias can produce a blood picture quite like acute leukemia" (Emerson, loc. cit., p. 73).

[9] Wells, Chemical Pathology, p. 259.

the rods or anything like them. This negative finding, however, should not count for too much, as the cells in the smears postmortem were all degenerated and stained poorly, whereas in the blood rods were seen only in perfectly preserved lymphocytes. (Auer did not obtain an autopsy. Pappenheim does not state in his description whether the bodies were found in the organs postmortem or not.)

An effort was made in one other way to throw some light on the question—namely, by inoculation. There have been many unsuccessful attempts at inoculation of leukemia, not only from man into the lower animals,[10] but from man to man (Schupfer)[11] and from dog to dog (Weil and Clerk[12]). Recently Ellerman and Bang[13] have reported the successful inoculation of the true leukemia of the fowl and its transmission through several generations of fowls.

In the present experiments macacus monkeys were chosen. One monkey received intravenously 15 c.c. of blood drawn directly from the patient. The other received in the same way 10 c.c. of spleen pulp from the same patient postmortem. Both monkeys have been kept under observation for six months and have apparently remained healthy. The blood of both monkeys has been examined frequently and has at times shown a marked relative lymphocytosis, small lymphocytes predominating. The experiments are to be regarded as negative, and it seems probable that leukemia, like malignant tumors, cannot be inoculated from one species of animal into another.

Conclusions. 1. The rods described by Auer can be found in many cases of acute leukemia.

2. They have never been found in any other disease.

3. There is no evidence that they are parasites.

4. The attempted inoculation of human leukemia into monkeys was unsuccessful.

5. Leukemia (like tumors) can probably only be successfully inoculated, if at all, into animals of the same species.

I wish to thank Dr. F. S. Mandlebaum for his kindness in making the accompanying photographs.

[10] Teichmüller, Deut. Arch. f. klin. Med., 1907, p. 62.
[11] Review in Jour. Amer. Med. Assoc., 1905, p. 882.
[12] Arch. de méd. exper., 1904, xvi, 462.
[13] Centralbl. f. Bakt., 1908, xlvi, 595.

VOLKMANN'S CONTRACTURE.

By Nate Ginsburg, M.D.,

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Richard Volkmann,[1] in 1869 and later in 1875, first described a deformity of the hand and wrist resulting from some interference with the blood supply of the flexor group of muscles of the forearm, usually following the application of splints for fracture occurring in the region of the elbow-joint. The involvement of nerve trunks was mentioned, but to these structures the principal changes were not attributed. The condition is usually styled Volkmann's ischemic paralysis, in order to attribute the profound anatomical changes producing this deformity to the existence of a primary myositis of ischemic origin, due to various factors surrounding the individual case. Writers earlier than Volkmann are said to have called attention to this condition, and to Leser[2] undoubtedly belongs the credit of having emphasized the importance of this type of deformity prior to Volkmann's more definite description.

From a review of the literature on the subject which has appeared since Volkmann's first publication, one is led to believe that this deformity is of rare occurrence. Dudgeon,[3] in 1902, searched the literature and reported only fifteen cases. He apparently failed to collect cases occurring outside of England. Powers[4], in 1907, collected 52 cases, and Taylor[5], in 1908, reported 59 cases, but failed to include two cases reported by Ward. Following the completion of this paper comes the last report by Thomas[6], of 107 cases, to which I can add the case operated upon by Taylor and the one to be reported in this paper. This brings the total number of cases of which we have authentic record up to 109. While it appears that this condition is one of rare occurrence, we are led to believe, however, that many more cases have been observed than have found their way into printed reports. Surgeons to many hospitals with large surgical clinics have had the misfortune to observe this deformity, resulting from either the injury itself or the treatment instituted to immobilize the parts. The printed reports of cases come principally from England, Germany, and this country; so that it is apparent that many cases may have occurred in other countries which have never been reported. Following Volkmann's original publication, attention was directed to this subject by the English

[1] Billroth's Path. u. Chirurgie, 1869, ii, 846; Centralbl. f. Chirurgie, 1881, viii, 801.
[2] Volk. Sammlung. klin. Vorträge, 1884, No. 249.
[3] Lancet, 1902, p. 78.
[4] Jour. Amer. Med. Assoc., 1907, xlviii, 759.
[5] Annals of Surgery, September, 1908, 394.
[6] Ibid., March, 1909, 330.

physicians, notably Littlewood,[7] Page,[8] Barnard,[9] and Dudgeon, the latter having written quite at length on the deformity from a neurological standpoint.

There is by no means a unanimity of opinion as to the causative factors producing this deformity, which is not always a true paralysis. Some observers have believed various accidents attendant upon the injury to be the cause of the resulting contracture, while others believe that the malformation is the result of improper treatment, that is, too much splint pressure.

If we term this deformity and physical disuse of the hand, which, as a rule, is out of all proportion to the degree of injury sustained, Volkmann's contracture, we are not bound to confine the primary anatomical changes solely to the muscle tissue. Whether the condition is the result of the profound ischemia of the flexor and pronator muscles of the forearm, resulting in a myositic degeneration with subsequent scar tissue formation due to a mechanical obstruction of the arterial blood supply to this group of muscles, or whether it be due to a disturbance in the integrity of the vascular structures owing to injury sustained by the trauma producing the fracture (Bloodgood), the type of deformity is usually the same. That this condition of marked contracture and loss of flexor motion of the fingers in the affected limb is not always due to a primary myositis, to the exclusion of other causes, I shall show from the case here reported, in which muscle changes were of secondary importance compared with the disturbance in the continuity of the nerve structures.

Definition and Cause. Volkmann's contracture is essentially a deformity of the fingers of the hand, in which the wrist-joint takes part, and comes on rapidly with loss of muscular power of the forearm. Two cases out of 109 have been observed in which the contracture affected the foot, due to injury and involvement of the flexor muscles of the leg. The condition is almost invariably associated with a fracture of the forearm or humerus in the region of the elbow-joint. It is usually observed in young children under the age of fifteen years, although Dudgeon treated a case occurring in a man of twenty; and Wallis[10] and Ward[11] each observed a similar deformity in women of this same age. Slöffer[12] reported a remarkable case in a boy, aged eighteen years.

The fundamental factors producing the condition are interference with the circulation of the muscles of the part, and injury to the nerve structures in close relationship to the involved muscles. That the condition of contracture as seen in the well-developed type is not always primarily of myositic origin is shown by cases following the prolonged application of an Esmarch bandage above the elbow-joint, and by Slöffer's case, in which thrombosis of the axil-

[7] Lancet, 1900, 290. [8] Ibid., 1900, 83. [9] Ibid., 1901, 1138.
[10] Practitioner, 1901, lxvii, 129. [11] Lancet, 1902, 372.
[12] Wien. klin. Woch., January 3, 1901, 24.

lary artery followed a gunshot wound of the thorax. The ball lodged in the axilla, and injured the artery.

Dudgeon reported pressure sores in seven out of fifteen cases, due to splints too tightly applied. Taylor states that at least 80 per cent. of reported cases have resulted from splint pressure for fractures of the forearm or humerus. Davies-Colley[13] observed pressure sores occurring in his cases, and ascribed this as the specific cause of the contracture. Page believed the deformity to be the result of a combination of pressure, fixation, and ischemia. Although Volkman stated that he had never seen a case result from the use of an Esmarch bandage, but knew it to be a cause, the case reported by Wallis, in which an Esmarch bandage was applied for over an hour, is proof of the influence of this factor in the production of this condition. Anderson believed the prolonged fixation of fractures of the forearm by any form of apparatus that interrupted the free circulation of blood through the muscles and nerves of the part acted as the causative influence producing this type of contracture. Bloodgood[14] doubts the myositis theory resulting from anemia of the muscles, believing that, although such cases undoubtedly do occur, the probabilities are that more cases are due to injury of the bloodvessels at the time of the accident.

It is curious that with the tight application of splints, usually an anterior and a posterior, to the extent of producing a pressure sore, the muscles involved should always be the flexor group, to the total exclusion of the extensors. It is due to this fact that the typical contracture is produced. That the extensors are not, as a rule, involved may be explained on the following anatomical grounds. On the anterior surface of the forearm the soft structures overlie the radius and ulna, forming at the internal aspect of the upper forearm quite a pad of soft tissue. In addition to this arrangement, the body of the flexor group of muscles is chiefly anterior, coming in direct contact with the splint when applied. The relation of a splint on the dorsal surface of the forearm is such that the ulna is interposed between it and the soft tissues, chiefly the extensor group of muscles. It is to the interposition of this bony structure, which undoubtedly receives the splint pressure, that the extensor muscles owe their escape when splints are so tightly applied as to cause a tissue necrosis with the production of a sore. While this reasoning seems adequate to explain the deformity as a direct result of faulty splint adjustment, it does not definitely solve the factors producing a contracture after long application of an Esmarch bandage, or a direct injury to the vessels some distance from the site of the muscle changes. It is reasonable to expect as much damage to the extensors from these causes as the flexor group suf-

[13] Guy's Hospital Gazette, 1898, n. s., xii, 460.
[14] Progressive Medicine, December, 1908.

fers, and yet such does not appear to be the case. It is therefore apparent that some other factor must obviously enter into the causative influences producing this deformity. Since no other structures are more exposed to injury than the nerve trunks with their close proximity to the arteries, a constriction sufficient to close the large vessels of the brachial region lasting over a long time must necessarily exert marked pressure on the median and ulnar nerves, and induce a neuritis with subsequent nerve degeneration. That the extensor muscles do not suffer is due to the fact that the musculospiral nerve, which ultimately distributes branches to this group of muscles, is deeply placed in the upper arm between the triceps muscle and the humerus. In the region of the elbow-joint the nerve is protected by its position between the long extensors of the forearm. It is, therefore, not subjected to as much trauma as the other cords because of their superficial position in the upper arm.

Embraced as it is by the pronator radii teres muscle, the median nerve is practically subjected to the same forces inducing injury as is this structure. It would seem, therefore, that if pressure over this muscle and its associated group is sufficiently great to cause a profound ischemia, the nerve must primarily share the fate of the muscle which encloses it, and suffer degenerative changes to some extent. Sensation of the fingers does prove that the nerve is involved in very many instances, and the occurrence of flexor muscle paralysis with no marked myositic changes strengthens this view. If we accept a primary myositic and neurogenic origin for this type of contracture, we are able to bridge over deficiencies in the causative factors occurring in cases in which the integrity of the muscle structures is less disturbed than that of the nerves. In this way can be explained those cases of Volkmann's contracture in which no fracture existed, or in which no profound changes took place in the muscles.

Symptoms. The symptoms are usually of rapid occurrence, "coming on in half a day or less in very severe cases" (Volkman); but they may be delayed over a period of many days. The promptness with which the hand assumes the condition of contracture, with the characteristic deformity of the fingers, is surprising. If splints have been applied there is usually considerable pain in the forearm and fingers, due to the great pressure, which is augmented by the traumatic swelling of the parts. Some difference of opinion seems to exist in regard to the presence of pain in these cases before the immobilization of the limb is relieved. Dudgeon believes pain is conspicuously absent in most cases, and attributes the progression of the deformity to the lack of interference due to no complaint on the part of the patient. Tightly applied splints for any injury usually cause discomfort, and it is reasonable to suppose that when splints are so firmly adjusted as to produce a pressure sore, the patient will feel decided discomfort and much pain until

their removal. This appears to be the experience of most observers of this condition.

It is important to remember that the condition varies in severity in direct proportion to the length of time the splints are allowed to remain applied, and the degree of pressure and injury sustained. It is due to this fact that much difference of opinion exists regarding the prognosis and treatment, because one case may be irreparably damaged before splint removal, while another with prompt treatment may be of a mild type with apparent functional recovery.

The characteristic deformity is usually that of a pronated forearm slightly flexed at the elbow. There is a decided flexion of the hand on the wrist-joint, and an extension of the metacarpal-phalangeal joints. The interphalangeal joints of the fingers and the terminal joint of the thumb are flexed, and the fingers are often buried in the palm of the hand. Extension of the fingers is impossible with the hand extended at the wrist by any justifiable force, and when the wrist is sharply flexed, the extension of the fingers can usually be procured, although in very severe types even complete extension cannot be made in this position. When pronounced myositis has occurred, later there is usually loss of the soft resiliency of the flexor group of muscles, and a greater degree of resistance is present with a board-like sensation. Add to this condition a cicatrized sore at this site, and the thickening of the tissues may be very marked.

Pressure sores may occur on the flexor surface of the upper forearm, and on either aspect of the wrist. In the case here reported, the photographs show the large shell-like scars on both surfaces of the wrist region. The anterior sore involved the superficial flexor tendons and the median nerve, causing a dissolution of their continuity. Schram reported 60 per cent. of pressure necrosis of the skin in his series. If a pressure sore is present, it usually occurs over the flexor surface of the forearm. Splint pressure may also rarely produce a sore on the posterior surface of the forearm near the elbow. These wounds may not always do so, but depending upon their severity, the necrosis may involve nerve and tendon structures and lead to a pitiful case. From his observation of cases of this severe type, it is possible that Davies-Colley ascribed the contracture to the result of a pressure sore.

Before the splints are removed, marked discoloration and swelling of the hand may be observed with bleb formation on the fingers. In old cases the loss of the use of the hand and nerve involvement will produce a shining, glossy skin, which is cold to the touch; if sensation is impaired, accidental burns may result, as occurred in the case presently to be reported. Trophic changes are absent in this condition, and ankylosis of the joints, although sometimes apparent, has never been observed in a true Volkmann's contracture. Shortening of the limb, which is not of infrequent occurrence in cases of long standing, is suggested by Turney as being due to

the lack of growth of the bones owing to the diminished support they are required to afford the affected muscles.

Nerve Reactions. The normal electrical reactions of both muscles and nerves is thought by Oppenheim[15] and Turney[16] to be an important diagnostic factor. Dudgeon and Hedley[17] believe with Turney concerning this feature of the disease. Depending upon the time of observation and the duration of the condition when examined, the results of electrical examinations of the muscles and nerves will greatly vary and therefore account for the different expressions of opinion. Involvement of nerve trunks was found to exist in sixty-two of the one hundred and seven cases contained in the table of cases collected by Thomas. He believes this lesion was probably present in some of the other cases, as in many papers the details of the examinations were not given.

Pathology. The histological changes causing this deformity are still a matter of some speculation. Volkmann believed that the deprivation of oxygen to the muscles owing to obstruction of the blood current caused a condition of muscle asphyxia with the subsequent production of a condition simulating rigor mortis. The venous stasis which occurs simultaneously with the occlusion of the arterial current hastens the onset and adds to the severity of the deformity. Raymond Johnson[18] and Riedinger[19] believed the splint-pressure inducing an anemia of the part produced an inflammation and subsequent fibrous degeneration of the muscles. Davies-Colley thought the condition could be largely accounted for by attributing the pathological changes to the pressure sore invading the soft parts of the flexor region of the upper arm. Wallis and Ziegler[20] account for the structural changes by extravasations of blood serum and round cells into the tissues affected, with subsequent fibrous tissue proliferation, causing contracture of the muscle substance. Kraske[21] thought extreme cold could produce this deformity; and Hildebrand[22] and Bloodgood believe that some direct injury or interference with the blood current at the time of the accident, rather than splint pressure, is the underlying cause in most of these cases. Little mention seems to be made of nerve injury, and yet an analysis of the cases with the symptoms resulting from the injury shows early and often severe nerve involvement. Hildebrand suggested that nerve changes might be primary in their origin, but his view was not shared by Bardenheuer,[23] who thought that nerve degeneration followed secondarily. No doubt many cases will show nerve destruction at the time of the accident, in addition to the profound histological changes in the muscles.

[15] Diseases of the Nervous System, 1904.
[16] Quoted by Dudgeon, Lancet, 1902, p. 78.
[17] Ibid., p. 82.
[18] Lancet, 1898, i, 722.
[19] Sitzungs. Berichte, der phys.-med. Gesellsch. zu Würzburg, 1902, No. 3, 33.
[20] Lehrbuch der Allg. Path., etc., p, 306.
[21] Centralb. f. Chir., 1879, No. 12.
[22] Deut. Ztschr. f. Chir., 1890, xxx, 98.
[23] Centralb. f. Chir., 1906, xxxiii, 505.

The nerves at operation are often found swollen and thickened or so degenerated as to present only a fibrous strand of tissue. Some cases at operation have shown a wasting of the muscles with a pale and dry appearance.

In the case reported herewith the muscle substance which was exposed at operation showed no macroscopic changes which would indicate injury or degeneration. In this case the deep pressure sore on the anterior surface of the wrist had invaded all the superficial flexor tendons and the median nerve. The nerve had been destroyed at the middle of the forearm, and the distal end could not be found at the wrist. Most of the superficial tendons had also been lost through infection.

Diagnosis. A fracture of one or both bones of the forearm or of the lower extremity of the humerus occurring in a child under fifteen years of age accompanied by much swelling, with the immediate application of splints, usually of the anterior and posterior variety, resulting in discoloration of the hand with the presence of much pain, and a tendency for the fingers to contract, should at once suggest the possibility of the presence of a Volkmann's contracture. Removal of the splints will at once present the characteristic features of this deformity, especially if added thereto a pressure wound is seen. Volkmann stated that the paralysis and contracture arise simultaneously, not as in paralysis of nervous origin, in which contraction is a later phenomenon and of gradual onset. While the contracture, as a rule, is of rapid occurrence, the hand assuming the position shown in the photographs, the deformity may be somewhat delayed by the continued use of an anterior splint after the fingers have begun to contract.

At times some difficulty may arise in differentiating a Volkmann's contracture from other conditions in which pure nerve lesions are the underlying causes. In ulnar and median nerve paralysis the prompt loss of sensation in structures to which these nerves are distributed will result. Muscle paralysis will follow at once, depending upon the group of muscles to which the nerve distributes branches. In ulnar nerve paralysis the reactions of degeneration in the wasted muscles are present, but there is no reaction to stimulation with the faradic current. The resulting atrophy of the muscles of the hand, and the development of the so-called "claw-hand" in paralysis of this nerve, will clearly indicate the nature of the lesion.

Trophic lesions may be present, and there is usually no flexion of the wrist in nerve palsies. In musculospiral nerve paralysis there is usually a bilateral wrist-drop, unless the nerve on one side is injured from the surface. The muscles involved in this type of deformity are the extensors of the forearm, and the lesion is easy of recognition.

Prognosis. "There is no more pitiful condition than this after an injury, usually a fracture, in healthy strong children"

(Bloodgood).[24] The prognosis in cases in which the hand and fingers are affected with much deformity is bad, and if nerve involvement is present, as appears to be the case in many instances, the outlook for a good functional result is practically hopeless. Anderson[25] opposed Volkmann's gloomy prognosis and believed that function of the hand and fingers could in a large measure be restored, but he did not cite any cases to substantiate his claim.

The extent of the damage done to the muscles and nerves will in a very large measure determine the outcome of the case, if prompt treatment is instituted. It is due to this fact that early cases with little time for extensive changes to occur have yielded to measures tending to restore the normal tone of the structures involved. Some cases are undoubtedly hopeless from the beginning, and no form of treatment will preserve the use of the affected hand.

Treatment. There is apparently wide difference of opinion in regard to the course of treatment to be pursued for the purpose of restoring the hand to its normal appearance. So great has been the difference of opinion as to the specific underlying cause of this type of contracture, and the proper treatment to be undertaken, that we are given Volkmann's almost hopeless prognosis on the one hand, and later the hope expressed by Anderson that it was the exceptional case which did not recover. This difference of view is obviously due to the varying degrees of injury which may be sustained, resulting either in a deformity of almost transient type or one of hopeless contracture and physical disability. When little splint pressure has existed, with only a slight or no break in the continuity of the skin, and prompt recognition of the condition (with measures for relief), the resulting deformity will not be of the type generally seen after the long application of splints with deep pressure wounds involving the muscles, tendons, and nerves. In the latter class of cases any form of treatment will often be of little use, and hence the choice of the many methods of correcting the deformity will vary with the individual case and the skill of the operator. A careful review of the cases tabulated by Dudgeon, Ward and Powers, and later by Thomas, will show some of the best results following an expectant line of treatment supplemented with massage, passive motion, and electricity.

It matters little which factor is foremost in producing this deformity, or whether it can only occur from splint-pressure, and not result from too long application of an Esmarch bandage—the fact remains that the disuse and appearance of the hand resulting from either cause conforms with the contracture first noted by Volkmann, and the correction of the condition confronts the surgeon, who is left to select the operation which best suits the individual case.

[24] Progressive Medicine, December, 1908.
[25] The Deformities of the Fingers and Toes, p. 66.

In spite of the greatest care, splints properly applied for a fracture of the forearm or humerus near the elbow will often, in a few hours, become tight and uncomfortable from the swelling and œdema of the part. It is, therefore, a wise precaution to see all fracture cases within a few hours after the reduction of the fracture and the immobilization by splint application. If there is discomfort, prompt removal of the dressing and its reapplication, allowing for the swelling of the part, will be the best prophylactic means of obviating the development of this deformity.

The evidence adduced by the experimental work of Lapinsky[26] and Leser[27] tends to prove that the continued immobilization, following tight splint pressure with resulting ischemia of the muscles of the part, is a very important factor in the production of this deformity. Therefore, with the symptoms of this condition, the prompt removal of the splints or plaster bandage is of the first importance. Every effort should be made toward restoring the hand to its normal anatomical and physical appearance. The splints may be left off if necessary, allowing the fracture to care for itself. Passive motion, massage, electricity, and even active motion must be resorted to at intervals of a few hours to correct the condition. A deformity due to the improper reduction of the fracture is of much less significance compared with the severe result from the reapplication of splints if the hand and wrist are already contracted.

Once present, the condition must be carefully studied with a view to determining the extent of muscle, nerve, and tendon involvement. If large and deep pressure sores are present, little can be done until they are healed. When tendon and nerve structures are much involved, and subsequent destruction results, due to infection of the wound, the outlook is almost hopeless from any standpoint, and the loss of time awaiting the healing of the wound is not a great factor in the treatment.

If pressure sores are slight and heal quickly, treatment should be resorted to promptly.

Non-operative Treatment. There are some cases which will probably recover without operative treatment. This class includes cases with slight injury to the muscles and nerves with little resulting deformity, and in which there has been prompt recognition and treatment as suggested. The treatment is often tedious, discouraging at first, and must be carried out over a long period of time, often many months, and sometimes years. In this class of cases the principle measures to be employed are massage, passive and active motion, and electricity, with permanent removal of the

[26] Deut. Ztsch. f. Nervenh., 1900, xvii, 322.
[27] Samml. klin. Vorträge, Leipsic, 1884, p. 249.

splints if deemed necessary. Such cases have been reported by Leser, Niessen,[28] Dudgeon, and others.

The extension of the fingers and hand under an anesthetic as the sole measure of treatment, employed by Volkmann, has proved valueless, and often difficult to accomplish. Mechanical extension of the fingers and hand over a long period of time (Sayre[29]), and continuous elastic traction (Martin[30]), have been employed with good results. Robert Jones,[31] of Liverpool, obtained a good result by separate splinting of the fingers, with flexion at the wrist. By increasing the length of the splints he was enabled eventually to secure extension of the fingers. He believes this gives better results than operative treatment The various means of treatment with the success attained by each tends to prove the assertion that all grades of injury and deformity may occur, to which special types of treatment are adapted.

Operative Treatment. Garré,[32] in 1895, and later Henle[33] and Raymond Johnson, first advised resection of portions of the radius and ulna in order to restore the hand to its normal position. By shortening the forearm the flexion of the fingers and hand at the wrist are overcome and motion is permitted, provided the muscles have not undergone complete fibrous transformation.

In shortening the bones care must be taken to perform the resection at different levels to avoid fixation of the bones from the union of the callous in the interosseous space. This operation is short, and simple in its performance, and has given very good operative relief in many cases in which it has been tried. The danger of non-union (Raymond Johnson) is slight, but shortening of the arm is produced, and if there is much growth of the bones later, some contracture may recur (Erdman[34]).

Tendoplasty. This operation has been successfully employed to overcome the marked flexion present. The English surgeons, particularly Page, Littlewood, Barnard, Wallis, and Ward, have done this operation with marked success, in some cases almost completely restoring the use of the hand. This operation has many warm advocates, and is preferable to resection of the bones in some cases, particularly when the tendons and nerves are involved in the newly formed fibrous tissue affecting the muscles, or in the scar resulting from a pressure sore.

Lengthening of the tendons is a tedious and often difficult operation to perform, but allows of an exploration of the injured struc-

[28] Deut. med. Woch., 1890, xvi, 796.

[29] Mentioned by A. S. Taylor as presented before the Pediatric Section of the New York Academy of Medicine, March 12, 1901.

[30] Congress Français de Chirurgie, Paris, 1903, xvi, 934.

[31] Amer. Jour. Orthop. Surg., 1908, v, 377.

[32] Deut. med. Woch., 1895, xxxi, 484.

[33] Henle. Quoted by Dudgeon.

[34] New York Surgical Society, April 8, 1908, quoted by A. S. Taylor.

tures and a transposition of the ulnar and median nerve if either is found too tightly in the grasp of the scar tissue.

The danger of mistaking tendons and accidentally injuring the nerves in the forearm are somewhat exaggerated, and the chief difficulty in the performance of this operation is the length of time required. If the scar resulting from the pressure sore is large, it should be excised and transplantation of a skin flap done, the upper abdomen being a convenient region from which to utilize the necessary tissue. The latter method was pursued by Dr. Stewart in the case here reported.

Tenotomy. Tenotomy has been practised in a few instances to overcome the deformity present. This measure eliminates all future possibility of obtaining any use of the hand, and while it improves the appearance of the limb, it is doubtful whether this procedure should be followed even after every possible means has been tried to restore the function of the fingers.

These are the principle operative procedures that have been tried for relieving Volkmann's contracture. Prompt relief to nerves which appear to be involved in the scar tissue should be afforded and superficial transplantation done, as recommended by Kleinschmidt[35] and Freeman,[36] who believe in very early nerve involvement in these cases. Bardenheuer believes the nerve changes in these cases are secondary to muscle involvement, and advocates an operation for the purpose of separating the muscles.

Whatever operation may be done to cure the condition, every assistance should be given the patient, following recovery from operation, to restore the use of the hand. For this purpose massage, electricity, and active and passive motion should be tried, and carried out over a long period of time. It is surprising the improvement that will follow after a long course of the above treatment, covering many months and sometimes years. In addition to these means, congestive hyperemia preceding the bath and massage is recommended by Taylor.

The following case was observed and studied by me: J. S., a male, aged twelve years, a school-boy, was first seen on August 30, 1908, in the out-patient surgical clinic of the Philadelphia Polyclinic, in the service of Dr. Francis T. Stewart, to whom I am indebted for the privilege of studying and reporting the case. There are no incidents in the medical history of the case previous to the accident which have any bearing upon the present condition. On August 20 the boy fell out of a second story window and sustained a fracture of the right radius about 3 cm. above the wrist-joint (Colles' fracture). He was treated in the hospital surgical clinic about forty-eight hours after the injury was sustained, when the splints (both an anterior

[35] Centralb. f. Chirurg., xxxiv, 81.

[36] Surg., Gynec., and Obs., July, 1907.

and posterior) which were applied within an hour after the fracture were removed because of great pain, swelling, and discoloration of the fingers of the hand. There were a number of blebs on the palmar surface of the thumb. He had suffered continual pain following the reduction of the fracture and the application of the splints, which increased in severity and then gradually disappeared, to be followed by numbness of the part. Upon removal of the splints, the right hand was observed to be swollen, discolored, and somewhat colder than its fellow. There was a little flexion of the hand on the wrist, and some contraction of the interphalangeal joints of the fingers. The thumb was adducted, and three or four blebs containing bloody serum were present. The deformity was not very marked, because the splints had been very tightly applied by means of strips of adhesive plaster and a bandage of muslin. Over the flexor surface of the wrist there was a large oval pressure sore extending into the subcutaneous structures. A similar sore was present on the dorsal aspect of the wrist, but did not involve the extensor tendons. A slight break in the continuity of the skin just below the elbow-joint, where the upper edge of the posterior splint had pressed, was present, with two or three blebs. There was no pressure wound over the body of the flexor muscles of the forearm.

Some flexion of the forearm at the elbow-joint was present and persisted for several weeks. Motion of the fingers and the hand was very slight, and painful. Skiagraphic examination revealed a fracture of the radius about 3 cm. above the wrist joint, with some anterior projection of the lower fragment.

The posterior splint was removed permanently, and every effort was made to heal the large pressure sore on the flexor surface of the wrist. Any motion of the fingers or hand was attended with such excruciating pain that for the first week the wounds on both surfaces of the wrist were dressed daily and lightly bandaged to an anterior splint extending to the tips of the fingers. The progression of the deformity was rapid, and by the end of the first week the characteristic pronation and flexion of the hand, with contracture of the fingers described, was present. The splint was removed and kept off, and since nothing could be done until the deep wounds healed, the limb was dressed daily and carried in a sling. Infection of the sore on the flexor surface of the wrist followed, and the superficial tendons sloughed away, leaving a thick contracted scar, as shown in the accompanying photographs (Figs. 1, 2, and 3). The healing of this wound occupied many weeks.

At the time the electrical examinations were made by Dr. Spiller, November 15, 1908, the deformity was very marked, and corresponded to the classical description of Volkmann's contracture. The following is Dr. Spiller's report: "J. S. has prompt reaction to faradic current in the right forearm, in the muscles of both flexor and extensor surfaces. There is slight contraction of the flexor

and extensor muscles below the wrist. Sensation for touch and pain are greatly impaired below the wrist and apparently normal above the wrist."

FIG. 1.—A large scar on the dorsal surface of the hand; flexion of the hand at the wrist, and of the fingers at the interphalangeal joints.

FIG. 2.—Marked pronation of the hand; flexion of the forearm on the elbow, and extension of the fingers at the metacarpal-phalangeal joints.

FIG. 3.—A large scar on the anterior surface of the wrist, resulting from the pressure sore; the fingers and thumb present the characteristic contracture observed in this type of deformity.

So greatly was sensation impaired in the fingers of the hand that an accidental burn occurred over the first interphalangeal joints without the knowledge of the patient.

When the pressure sores were finally healed, the hand presented practically a useless appendage, and the tips of the fingers were almost buried in the palm. With complete flexion of the hand on the wrist, the fingers could not be fully extended, and pronation of the hand was marked. Passive and active motion had only succeeded in overcoming the flexion at the elbow.

It was apparent that this case presented a combination of many factors, producing the deformity: involvement of nerve, tendon, and probably muscle structures.

Operation, November 23, 1908, by Dr. Francis T. Stewart. The operation was performed under ether, and required over two hours. Excision of the scar on the anterior surface of the wrist was performed, leaving a large denuded area. The longitudinal incision was carried up to the middle of the forearm, and a careful dissection of the adherent and fibrous structures was made. The tendons of the flexor sublimis digitorum and palmaris longus muscles were absent, and all that remained was their proximal ends. The ulnar nerve was intact and uninjured, and the pulsation of the ulnar artery was feeble. The pronator quadratus was so changed as to present merely a white quadrilateral fibrous structure with no macroscopic remains of muscle tissue. The median nerve was destroyed from the middle of the forearm to below the annular ligament at the wrist, and only the proximal end could be found where it emerged from the mass of the pronator radii teres muscle. The nerve seemed to be continuous with a thin fibrous strand of tissue. The radial pulse was also feeble.

The anterior projection of the lower fragment of the radius formed a sharp ledge, over the border of which the structures were pressed by the tightly applied splints. It is quite likely that this feature of the accident greatly assisted in the subsequent destruction of the tendons and median nerve which followed the primary injury. Incision carried to the flexor muscles showed no macroscopic alteration in their appearance suggestive of a very pronounced myositis.

Tendoplasty was performed, utilizing the deep flexor tendons and the remains of the superficial group of tendons. The fingers and the hand were extended, and the forearm placed midway between pronation and supination. Transplantation of a flap from the epigastric region, leaving one end attached, was done to fill in the large gap left by the removal of the scar on the flexor surface of the wrist. The hand was bandaged across the upper abdomen for ten days, at the end of which time the attached end of the flap was cut and the hand freed..

Postoperative Course. The graft, consisting of the skin and subcutaneous fatty layer, healed promptly and was not followed by

a contracted scar. At the end of two weeks active and passive motion were begun, and supplemented with massage, electricity, and baths, which are being continued at the present time. Contracture of the fingers has recurred, but the deformity is not as severe as it was previous to the operation. The thumb is still adducted, but allows of slight motion, and there is some improvement in the extensor and flexor movements of the fingers. Sensation in the hand has distinctly improved, but the limb is very far from being a useful extremity. At this writing, steady improvement has been noted in the recovery of the motion of the fingers. It is to be hoped as time goes on that enough motion of the fingers and hand may be secured to enable the patient to perform the more necessary acts.

In this case we have the contrast of a careful painstaking operation, the purpose of which was to free any adherent structures in the forearm, and to lengthen the tendons, thereby correcting the deformity of the hand, with the operation of resection of portions of the radius and ulna recently performed by Taylor. The operation done in this case was tedious and time consuming, but it seemed preferable to any other procedure. In both cases the ultimate result was poor, owing to the incurable state of the damaged structures. Although improvement is slowly going on, it is extremely doubtful whether in either case complete recovery will ever take place.

Conclusions. 1. The specific underlying factor producing a Volkmann's contracture is still a matter of dispute; but the evidence seems to warrant the assumption that it is a deformity in which muscle, nerve, and tendon structures share. Early, and probably primarily, the nerve structures share the same injury which induces muscle anemia with subsequent myositic degeneration. The contracture is the result of a myositis, with accompanying nerve lesion, and may vary from a transient type to that of hopeless deformity.

2. This deformity follows, in most cases, too tightly applied dressings for fractures of the forearm or humerus in the region of the elbow-joint, in children under the age of fifteen years. Usually the pressure has been of sufficient intensity to produce a pressure sore, which is present in 80 per cent. of the cases. It may arise within less than twenty-four hours, or be delayed for many days.

3. Severe pain, followed by numbness, discoloration of the hand with bleb formation, and contraction of the fingers are first noticed. Later, the characteristic flexion of the fingers at the interphalangeal joints, with flexion of the hand at the wrist, and marked pronation occur. Some flexion at the elbow is usually present.

4. Careful application of dressings, with frequent inspection, is the best prophylactic measure. Prompt recognition of the symptoms, with measures to restore the hand to its normal appearance,

constitute the safest means of obviating the subsequent development of what may prove to be an irremediable condition. Active and passive motion, massage, and electricity, with permanent removal of the splint, should be advised if deemed necessary.

5. Operative treatment depends upon the individual case. No specific procedure can be suggested, but tendoplasty and resection of both bones of the forearm are the two methods of operative treatment offering the best chance of recovery of use of the hand. The nerves should always be examined to determine the extent of involvement, and should be freed if necessary.

6. The prognosis is unfavorable depending upon the duration of the case and degree of involvement of the damaged structures. Partial recovery is not unusual in cases seemingly hopeless in the beginning.

A CASE OF APPENDICITIS IN WHICH OXYURIS VERMICULARIS WAS FOUND IN THE APPENDIX.[1]

By Astley Paston Cooper Ashhurst, M.D.,
SURGEON TO THE OUT-PATIENT DEPARTMENT OF THE EPISCOPAL HOSPITAL, PHILADELPHIA.

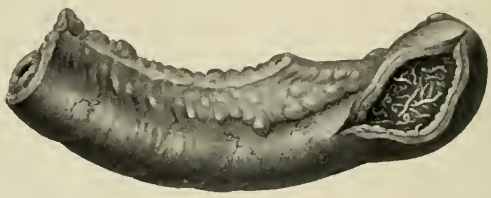
Laura P., a Polish girl, aged fourteen years, was admitted on March 14, 1909, to the Episcopal Hospital, in the service of Dr. G. G. Davis, to whom I am indebted for the privilege of operating and of recording the case. Owing to the patient's ignorance of the English language, it was difficult to obtain an accurate history, but it was learned that she had been ailing for a week or ten days, and that twenty-four hours before admission she had been taken with severe abdominal pain, followed shortly by vomiting. The pain was at first general, but later settled to the region of the appendix. Examination at 9.30 P.M., soon after admission, showed great tenderness with some rigidity over the right iliac region. No mass was present. The temperature was 100.6° F., the pulse 88, and the respirations 34 per minute. The white blood cells numbered 25,000 per c.mm. The diagnosis was acute appendicitis.

Operation at 10 *p.m.* Through the transverse incision of G. G. Davis,[2] an appendix was removed which was congested at the tip, but presented no other gross evidences of disease. The abdominal wound was closed without drainage. When the appendix was slit open at its tip, the lumen of the organ was found to contain a number of minute worms, resembling "seat worms." Subsequent examination under the microscope by Dr. C. Y. White, director of

[1] Read at a meeting of the College of Physicians of Philadelphia, April 7, 1909.
[2] Annals of Surgery, 1906, xliii, 106.

the laboratories of the hospital, confirmed the diagnosis of Oxyuris vermicularis. When warmed up, the worms squirmed around actively. Under the microscope the appendix presented the ordinary picture of acute suppurative appendicitis in the early stage, its walls being studded with miliary abscesses. The patient's convalescence was uneventful, and examination of her feces revealed the presence of many more similar parasites.

My object in reporting this case is to call attention to the fact that the presence of intestinal parasites in the appendix is by no means rare, but that they are seldom, if ever, the direct cause of appendicitis. As I have pointed out in the chapter on the History of Appendicitis, prepared for the third edition (1905) of Dr. John B. Deaver's monograph, *Fabricius ab Aquapendente*, even as early



Vermiform appendix containing Oxyuris vermicularis. (Natural size.)

as 1634, mentions having found at times a worm in the appendix at autopsy; Santorini (1724) made the same observation, and thought that the chief function of the appendix was to serve as a nest for round worms, where they might be cherished and be prevented from escaping into the general intestinal tract.

Many varieties of parasites have been found in the appendix, especially Ascaris lumbricoides and Oxyuris vermicularis. F. D. Patterson[3] has collected ten cases in which lumbricoid worms and eight in which oxyurides were found in connection with diseased appendices. Tapeworm, trichocephalus, echinococcus, and bilharzia disease were also noted, in one instance each. To his list of oxyuris should be added two cases, reported by Beyea[4] and by Rammstadt.[5]

Still,[6] in 200 consecutive autopsies on children less than twelve years of age, found oxyurides in the intestinal tract in thirty-eight cases (19 per cent.), and of these cases no less than twenty-five, or two-thirds, had the parasites inside the appendix. Oppe, according to Sprengel[7], found oxyurides six times among sixty appendices removed at operation. Erdman[8] found, among 29 operations for

[3] Amer. Jour. Med. Sci., 1906, cxxxi, 859.
[4] Univ. Med. Mag., 1900, xiii, 67.
[5] Deut. med. Woch., 1902, xxviii, 919.
[6] Brit. Med. Jour., 1899, i, 898.
[7] Appendicitis, Deutsche Chir., Stuttgart, 1906, S. 182.
[8] New York Med. Jour., 1904, i, 537.

appendicitis in children under ten years of age, 4 cases with oxyurides in the appendix. Rostewsew, cited by Sprengel, found parasites three times in the appendix among 278 autopsies on persons who had not died of appendicitis; and also found them present the same number of times among 163 cases of appendicitis: from which he concluded that their influence in causing appendicitis was trifling.

It is well known that L. Metchnikoff holds parasites responsible for the majority of attacks of appendicitis; and he insists that in every case, before operation is undertaken, a microscopic examination of the feces should be made, and that in cases in which it is possible the effect of vermifuge remedies should be tried, even if this examination is negative. In 1901,[9] when he appears first to have called attention to this matter, he referred to four cases in which recurrent attacks of appendicitis ceased after lumbricoid worms had been expelled by the action of vermifuges. He acknowledges that it is not at all probable that the lumbricoids could perforate the appendix or other portion of the intestinal canal, but explains their action on the theory that they first produce erosions of the mucosa, and then deposit in these erosions the microörganisms with which their bodies are covered. In this way, Metchnikoff contends, parasites can be the primary cause of ulceration and perforation of the intestinal wall.

Yet I think the case reported herewith goes to show that even if the worms can be held accountable for the origin of the disease (which is a mere assumption), the lesions in the appendix, even at an early stage, are such as to make prompt operation the safest form of treatment. In this case the walls of the appendix were found on microscopic examination to be studded with miliary abscesses, and it was the opinion of the pathologist that the presence of the worms was a mere coincidence; delay in operation in this, as in most other cases, would have resulted in a localized peritonitis within a comparatively short space of time. From the fact that intestinal parasites are found within the appendix in 12.5 per cent. of children under twelve years of age (Still), it is not logical to conclude that they are on that account frequent causes of appendicitis. It is interesting to note their frequency, and it is important to take measures for their extermination; but the surgeon should not waste valuable time in attempting to cure symptoms of appendicitis by vermifuges, when it is not proved that the worms, even if present, are responsible for the symptoms of disease.

[9] Journal des Praticiens, 1901, xv, 185.

REVIEWS.

Modern Medicine. Its Theory and Practice. In Original Contributions by American and Foreign Authors. Edited by William Osler, M.D., Regius Professor of Medicine in Oxford University, England, assisted by Thomas McCrae, M.D., Associate Professor of Medicine and Clinical Therapeutics in Johns Hopkins University, Baltimore. In seven volumes. Vol. VI, Diseases of the Urinary System, of the Ductless Glands, of the Muscles, Diseases of Obscure Causation, Vasomotor and Trophic Disorders, Medical Aspects of Life Insurance. Pp. 799. Philadelphia and New York: Lea and Febiger, 1909.

Of the many and important subjects discussed in Volume VI of Osler's *Modern Medicine* major interest attaches to diseases of the urinary system and of the ductless glands—which together comprise about one-half of the volume.

The book opens with an introductory chapter by John McCrae on the normal and diseased physiology of the kidney, which summarizes well what is known of the subject, and is followed by an instructive chapter on malformations and circulatory disturbances of the kidney. A. E. Garrod contributes a chapter on anomalies of urinary excretion, in which, among other matters, he makes brief reference to tests of the functional competency of the kidneys, and discusses well such conditions as albuminuria, hematuria, pyuria, etc. In another chapter he tells us what is really known of uremia, and if he has been unable definitely to state the immediate cause of uremia, he has mentioned with sufficient critical comment the existing theories, except, perhaps, that which predicates major significance to the liver, and he describes accurately the clinical manifestations and the treatment of the condition. The important subject of nephritis, or Bright's disease, is discussed in a chapter of over one hundred pages by James B. Herrick; and to this he adds a short discussion of amyloid kidney. His discussion of the, or a, classification of the nephritides is rather illuminating; his opinion regarding the chronic cases is, perhaps, well reflected in his statement that "much energy is wasted and much needless disappointment experienced in the attempt to make a given case fit into a classification that is largely artificial and necessarily imperfect." He finally distinguishes a chronic parenchymatous, a chronic interstitial, and a mixed type, and of the

chronic interstitial, a primary, a secondary, and an arteriosclerotic form. In discussing the treatment of acute nephritis he pertinently points out the irrationality of giving large amounts of water with the idea of flushing the kidneys, emphasizing the fact that since the damaged and inflamed kidneys need rest, they refuse to excrete the excess of water which, retained, usually serves only to augment the œdema. His own practice is to be guided by the thirst in giving water, and this, perhaps, is as good a practice as any. The undoubted clinical value of a sodium-chloride-poor diet in œdema is also pointed out, without commitment to the theory that chloride retention is the cause of the œdema or that which looks upon the chloride retention and the œdema as phenomena of nephritis not related as cause and effect. The old-fashioned Basham's mixture is the author's favorite preparation of iron—when iron is needed. The pathogenesis of the cardiovascular changes seems still to be in an unsatisfactory state. Thomas R. Brown contributes interesting chapters on the bacteriology of the infections of the urinary tract and the urinary findings in these infections, on pyogenic infections of the kidney, ureter, and perirenal tissues, and on tuberculosis of the kidney. The general excellence of the chapters precludes any adverse comment, although perhaps it would have been well to accentuate somewhat the unilateral hematogenous infections of the kidney that recently have been submitted to clinical study, and are sometimes recognized and cured by operation. Tumors of the kidney, urinary lithiasis, and diseases of the prostate are discussed by Hugh H. Young—than whom it is doubtful if any one is better qualified. His observations on genito-urinary diagnosis may be read with profit by all practitioners.

The section on diseases of the ductless glands is contributed by George Dock. The discussion of Addison's disease, Graves' disease, cretinism, myxœdema, tetany, akromegaly, etc., is all that the most critical reader may expect. Considerable attention is devoted to disordered function of these ductless glands and to aberrant or ill-developed diseased manifestations, so-called *formes frustes*—which are all too commonly overlooked. The discussion of the treatment of exophthalmic goitre will afford some satisfaction to the conservative clinician who hesitates to hand over to the surgeon all his cases without a thorough trial of efficient, medical, remedial measures; these are well discussed and offer considerable hope of a successful issue in many cases. The indications as well as the dangers of operative treatment are also pointed out. The section is altogether worthy of its author—who contributes also a short article on osteomalacia.

The editors themselves contribute to this volume: Dr. Osler, articles on Raynaud's disease, angioneurotic œdema, and diffuse scleroma, and Dr. McCrae, an article on arthritis deformans.

Dr. Osler's name has long been identified with at least two of the diseases that he describes; the present articles may be viewed as authoritative pronouncements on the subjects. Dr. McCrae has handled very well the intricate subject of diseases of the joints commonly described under the designation arthritis deformans, but also passing current under a diversity of other names. Our real ignorance of the subject, especially of the etiological factors, is answerable for the existing complexity of the discussions and unsatisfactory classifications, and if Dr. McCrea has not brought order out of chaos, he has told us about all that is known on the subject, and what he says is based upon a large personal experience.

There are other articles of value: Warfield T. Longcope describes Hodgkin's disease, with painstaking investigations of which his name has become identified; Daniel J. McCarthy discusses astasia-abasia, adiposis dolorosa, myasthenia gravis, paramyoclonus multiplex, and periodic paralysis—intricate subjects well described; Walter R. Steiner writes on myositis, Thomsen's disease, and myotonia congenita; and Charles P. Emerson describes achondroplasia, hypertrophic pulmonary osteoarthropathy, Paget's disease, osteogenesis imperfecta, osteopsathyrosis, leontiasis ossea, microcephalus, and facial hemiatrophy. The volume is concluded with a really excellent discussion of the medical aspects of life insurance by Charles Lyman Greene.

As a whole, the volume contains a relatively large number of articles of diversified interest; a high level of general excellence is maintained throughout in the discussions—which for a long time must remain authoritative. A. K.

Appendicitis and other Diseases of the Vermiform Appendix. By Howard A. Kelly, M.D. Pp. 489; 215 illustrations, 3 lithographic plates. Philadelphia: J. B. Lippincott Company, 1909.

Dr. Kelly explains succinctly the purpose he had in view in the preparation of this new edition of his monumental work on the vermiform appendix, by saying that after the publication of the former it soon became evident that a compact resume dwelling with especial care on the practical side of the subject would better meet the daily needs of the great army of general surgeons throughout the country. His preface to the first edition is republished, although some of it is quite inapplicable to this book. The statement that he has "to thank Dr. John B. Deaver, of Philadelphia, for the use of his large stock of material, as well as Dr. A. O. J. Kelly," seems to require explanation. Why should he thank Dr. Deaver for the use of Dr. Kelly? It sounds like a form of peonage. And why should the "inspired declaration of the great apostle"—"Others have labored,

ye have entered into their labors"—be regarded as appropriate for a work on the appendix? If it had been a text-book of obstetrics its applicability would be obvious.

Pleasantries apart, there is little to say of the book except in praise. The former and larger work is so well known that an extended review of this resume does not seem indicated. The history of the diseases and surgery of the appendix is accurate and interesting. The anatomy is well described and well illustrated. The chapter on the physiology of the appendix is almost as brief—and for much the same reason—as the celebrated chapter on snakes in Ireland. The author's attitude about it is, however, highly to be commended as displaying the true scientific spirit. Most of us—including the reviewer—have written of the appendix as "merely vestigial" and as "functionless," but it is apparent on reflection that such statements may or may not be true, and should not be unqualified. Dr. Kelly, beginning with the question: "Has the vermiform appendix any function?" answers it as follows: The argument from analogy for a special function may be briefly stated thus: In the first place, the functions of a number of organs heretofore considered functionless, as the thyroid, thymus, and suprarenal glands, have proved of such inestimable value—nay, so necessary to normal life—that the scientific physiologist will do well to reserve his judgment in regard to the appendix, and occupy an attitude of expectant observation. Again, as in the upper part of the digestive tract special secretions are poured in from such important organs as the liver and pancreas, so in the lower may we not expect, after the sudden transition from ileum to colon, to find some analogous organ or organs profoundly modifying the food under its altered conditions? He adds that we await the final test of demonstration. The bacteriology and pathology of the appendix are admirably summarized, and in this latter section—as, indeed, throughout—the illustrations are to be especially commended.

The chapter on the clinical history of appendicitis includes a consideration of the most common of the complications and sequels, and could scarcely be improved upon. In the chapter on diagnosis it is stated that "in some obscure cases a valuable aid in examining the patient, described by B. McMonagle (personal communication), is the pain elicited by holding the fingers firmly over the abnormal site of the appendix and requiring the patient to contract the right psoas muscle by flexing the lower limb, held rigid at the knee, on the body." This seems itself obscure. How is the "abnormal site" determined in "obscure cases" before this procedure is adopted?

The differential diagnosis is comprehensive and instructive. In the chapter on appendicitis and typhoid fever the author quotes with approval the opinion of J. B. Murphy, who says: "It is my opinion that typhoid appendicitis should not be operated upon, unless there is a perforation. All my cases recover, those operated and not operated. At the same time, I feel that operation should not

be performed except in special cases." Later (page 256) the author says: "Typhoid appendicitis rarely calls for operation, and it is always a lamentable misfortune to subject such a patient to an unnecessary surgical operation with a long, wearing illness before him." If this were the accepted rule of the profession—as it is coming to be—many lives would doubtless be saved. Appendicitis in childhood receives full consideration, and the excellent paper of Meriel on appendicitis occurring after the fiftieth year is fully summarized. Typhlitis—the despised and rejected—is once more recognized as a distinct pathological entity, though it is rare and far from being equally distinct clinically. The chapters on treatment are full of valuable advice. There are few surgeons who will not subscribe to the author's two concluding "aphorisms:" "It is better to operate occasionally in error in a doubtful case than to wait too long for more positive signs and to lose a life now and then. No patient is ever killed by a skilful early operation. Many die for want of one." "However, this facility with which an operation can be done must not be used as an excuse for slipshod and unnecessary operations."

The advice to the general practitioner who does not often do surgical operations that he would do better not to operate upon an appendicitis case when a satisfactory surgeon cannot be found, unless there is a well-defined mass in the right iliac fossa, with heat and tenderness—evidence of abscess formation—is certainly sound, as are the subsequent easily understood and excellently simplified directions for this procedure in case operation is unavoidable.

The articles on the operative treatment and on the postoperative sequels embody Dr. Kelly's well-known views, which are substantially unchanged. The chapters on appendicitis in gynecology and in obstetrics, on newgrowths of the appendix, on its hernias, and on the medicolegal aspects of appendicitis, are up to the high standard of the rest of the book, which contains more valuable information on the subjects treated of than is to be found in the same number of pages elsewhere. A little looseness in writing—or in the condensation of the material of the larger book—is occasionally noticeable, *e. g.*, "There are no symptoms nor combination of symptoms" (p. 154), but, as a rule, the information given is presented in an attractive and interesting manner. J. W. W.

The Pancreas. Its Surgery and Pathology. By A. W. Mayo Robson, D.Sc. (Leeds), F.R.C.S. (Eng.), London, and P. J. Cammidge, M.B. (Lond.), D.P.H. (Camb.), London. Pp. 546; 175 illustrations. Philadelphia and London: W. B. Saunders Company, 1908.

The various diseases of the pancreas, inflammatory, cystic, and neoplastic, have been well understood for many years, and but little has been added to the work of such pioneers as Fitz and Senn, for

instance. The etiology of acute pancreatitis, the relation of the islands of Langerhans to diabetes, and the development of chemical tests whereby disturbance of the function of the pancreas may be detected, are instances, however, of details in which advances have been made. The treatment of pancreatic disease by surgical measures is of more recent date, and to the senior author of this monograph is due great credit for his persistent energy and pioneer work, especially in the treatment of chronic pancreatitis.

The first one-half of this large volume (Chapters I to X) is devoted to the anatomy, physiology, and pathology of the pancreas, and includes a chapter upon fat necrosis. These subjects are well handled and complete, most of the matter relating to the so-called chemical pathology of the gland being devoted to a consideration of the changes in the urine and feces, with especial reference to the well-known test of Cammidge, the junior author of this monograph. The authors state that they "never rely upon the 'pancreatic reaction' alone in making a diagnosis of pancreatitis or malignant disease of the pancreas, but always take into account the results of a complete analysis of the urine and a chemical examination of the feces, as well as the clinical symptoms." The improved or *C* reaction is intended to supersede the methods published previously by Cammidge; its technique is long and complicated, and in its present form it would seem to be available only to those who are versed in chemical methods.

Chapter XI is devoted to the subject of pancreatic diabetes, and although it is suggested that such may be prevented by operating early upon cases of pancreatitis and gallstones, it hardly seems worth while to devote over forty pages to it in a book of this character. Chapter XII, while mostly a repetition of the subsequent text, groups all of the symptoms and tests which may be of use in diagnosis, the chemical tests occupying much more space than the symptoms. The injuries and inflammatory affections are considered next (Chapter XIII to XVI), and, with the exception of some padding with case reports, present an excellent and complete description of the symptoms and treatment of these affections. The authors urge early operation for the acute cases, and advise rapidity in the execution of the operation. In chronic interstitial pancreatitis cholecystenterostomy is preferred, and the authors believe that "only in case of absence or contraction of the gall-bladder, or in case of unusual difficulties from adhesions or from the serious condition of the patient should cholecystotomy be done." The remainder of the text is devoted to pancreatic calculi, cysts, and tumors, which are discussed in a brief but satisfactory manner.

The illustrations generally are good, although some, Figs. 120, 150, and 174, for instance, could not exist without their legends. An index of authors is appended, and a bibliography at the end of each chapter adds materially to the value of the volume. G. P. M.

Clinical Diagnosis. A Text-book of Clinical Microscopy and Clinical Chemistry. By Charles P. Emerson, M.D., late Resident Physician, Johns Hopkins Hospital, Baltimore. Second edition; pp. 686; 126 illustrations. Philadelphia and London: J. B. Lippincott Company, 1909.

Essentials of Medicine. By Charles P. Emerson, M.D. Pp. 383; 117 illustrations. Philadelphia and London: J. B. Lippincott Company, 1909.

The first edition of Emerson's *Clinical Diagnosis* was noted in these columns shortly after its publication, and its commendable features were pointed out. In its second edition, the book has been much revised and considerably enlarged; the author states that he has re-written fully one-half of it, and that he has incorporated much new matter, notably the bacteriology of the sputum and the urine, and a number of new illustrations. It is not necessary to speak in detail of a book now so well known and widely used; it suffices to chronicle the publication of a new edition and to say that the book fully meets the requirements of clinical laboratory diagnosis.

In the mean time, Dr. Emerson has prepared another and smaller book, which comprehensively is designed as a text-book for students beginning a medical course, for nurses, and for all others interested in the care of the sick. The avowed purpose of the book attracts some attention, since it is no mean task to encompass in a small volume the special requirements of the three classes of persons mentioned. Personal inclinations commonly influence judgments regarding what properly should be included within essentials, so that opinions concerning the value of the book necessarily must vary; but bearing in mind the task imposed upon himself by the author, one may say that the book contains many facts clearly and succinctly stated—it is true, in a more or less popular phraseology, but well adapted to ready assimilation by those that know little of the subject. In addition, however, the book may be read with interest and profit by those who have passed beyond the initial stage of learning. A. K.

A Text-book of Operative Surgery. By Warren Stone Bickham, M.D., Phar.M., Junior Surgeon to the Touro Hospital, New Orleans; late Assistant Instructor in Operative Surgery in the College of Physicians and Surgeons, Columbia University, New York. Third edition; pp. 1206; 854 illustrations. Philadelphia and London: W. B. Saunders Co., 1908.

The first edition of this text-book of operative surgery appeared in 1903, and was followed by the second edition in 1904, now nearly five years ago. As might be expected, it has been necessary to add

much new matter, owing to the development of new fields of operative surgery during this period. The volume is larger by nearly 200 pages, and contains about 300 more illustrations than the second edition. The plan of the volume remains unchanged, and the same criticism can be made now that was made by the reviewer of the first edition, namely, that the constant repetitions required in the effort to make the description of each operation complete in itself add unnecessarily to the size of the book, and detract from its value as a text-book, though perhaps rendering it more useful as a book of reference. The failure to discuss the indications and contra-indications for operation, or the dangers, results, and complications of the operations described, also makes it less useful as a text-book than is a volume which includes a certain amount of surgical therapeutics. The amount of surgical anatomy included is minimal. But as the author makes no pretence to give more than the technique of the operations, these shortcomings cannot be considered as justifying adverse criticism; and to urge the inclusion of more matter would be injudicious, the volume being excessively bulky as it is.

Plastic surgery is still excluded, though it surely forms more a part of general surgery than such procedures as osteoplastic resection of the spine, an operation never very widely employed, and now, very properly, we believe, in disrepute: yet to this one subject nearly thirteen pages are devoted. The section on amputations is especially complete. Orthopedic surgery is slighted, as are gynecological operations. While the operations on bones in general are sufficiently described, bone wax is not even mentioned. The accounts of prostatectomy are antiquated, the author apparently having failed to grasp the essential points of the technique of suprapubic prostatectomy as practised by Freyer, and of the perineal operations developed by Albarran, Hartmann, and Proust. Young's method of "conservative perineal prostatectomy" is not mentioned. Dr. Bickham states that "other" operations on the prostate than those described have been omitted as belonging more especially to genito-urinary surgery; but to relegate all the best operations to the realm of specialism, and to permit the general surgeon to attempt only those which are truly blind, bloody, and barbarous, is to enunciate a principle in surgery which we believe to be thoroughly erroneous.

The operations on the gall-bladder are incompletely and inadequately described, and the operations of gastrostomy known by the names of Marwedel and Ssbanajew-Frank (not Franck), might well have been omitted. The account of perineal cystotomy (lateral lithotomy) is hazy, and the nomenclature of perineal operations on the male is in general confused, the old operation of "perineal section" (the "London" operation) being described as Wheelhouse's, merely because the use of Wheelhouse's staff is advised; and Cock's operation is called perineal section. The illustrations are very clear, even semidiagrammatic; but they sometimes will give a decidedly

false impression to the student, and not seldom are lacking in those minor details which count for so much in bringing an operation to a satisfactory conclusion. If another edition shall be called for, we will hope to see much condensation of essential matter, and to find many procedures seldom seen but in print totally excluded from the volume. A. P. C. A.

A Text-book of Special Pathology. By J. Martin Beattie, M.D., Professor of Pathology and Bacteriology in the University of Sheffield, and W. E. Carnegie Dickson, M.D., Lecturer on Pathological Bacteriology in the University of Edinburgh. Pp. 599; 191 illustrations. Philadelphia: P. Blakiston's Son & Co., 1909.

As in their *Text-book of General Pathology*, noticed in these columns several months ago, Drs. Beattie and Dickson, in their *Text-book of Special Pathology*, state that if any excuse were necessary for adding another to the many text-books of pathology, it would be found in the fact that the present volume is based on the teaching of the Edinburgh school, and that some fundamental points, which have been taught at Edinburgh for years, have not elsewhere received sufficient attention. Such, perhaps excusable, insularity is not the best incentive to the preparation of a scientific treatise and must be ignored in estimating the value of the book—which should be judged solely by its intrinsic merits. Thus judged, the book is found to contain the main facts of special pathology, clearly and concisely, in some places somewhat dogmatically stated. Special emphasis has been laid upon gross morbid anatomy—in which the book is relatively strong; but microscopic anatomy is rather neglected and there is little or no discussion of pathogenesis. There is some reason for excluding diseases of the eye and the ear; but the omission of all reference to the female genitalia, while including the mammary gland and the male genitalia, engenders the suspicion that perhaps *ennui* overtook the authors. What there is in the book is very good—the making of a really good book. A. K.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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A Clinical Procedure for the Recognition of Sugar in the Urine with Orthonitrophenylpropiolic Acid as an Indicator.—Bottu (*Soc. de biologie*, 1909, lxvi, 972) describes the method of using orthonitrophenylpropiolic acid as an indicator of sugar in the urine. He says that it is superior in sensitiveness and precision to the numerous copper or alkaline bismuth solutions in general use. The reagent is made by mixing 5 c.c. of fresh 10 per cent. sodium hydroxide with 3.5 grams of pure orthonitrophenylpropiolic acid powder and adding distilled water to make up to a liter. Its employment consists in adding 1 c.c. of urine to 8 c.c. of the reagent in a test-tube, and heating the upper layers. As soon as it comes to a boil, add drop by drop another cubic centimeter of urine. Then heat again. If the urine contains glucose, there will appear a blue color with more or less precipitation of small particles of indigo blue. If the reduction occurs spontaneously before the second boiling, the glucose content can be estimated to be 10 grams per liter or more. The reaction is trustworthy, very easy, and sensitive.

Statistics of Gonorrhœal Arthritis.—In the course of a fifteen-year practice, among 820 cases of gonorrhœa, Jordan (*St. Petersburger med. Woch.*, 1909, xxxiv, 59) has observed eighteen cases of gonorrhœal arthritis. This is a frequency of 2.1 per cent., and agrees well with the figures of other reports. The time of appearance of the arthritis has varied. Five cases occurred in five to seven days after the onset of the urethritis; 2 cases, in the course of the first two months; 11 cases, in the chronic stage. However, in most reports the figures of early occurrence are not so reliable, inasmuch as they depend solely upon the statements

of the patients as to the onset of the urethritis. One thing is certain, that is, arthritis may be an early complication of specific urethritis. As predisposing causes of the arthritis, exposure to cold, trauma of the affected joints, erection and trauma of the urethra must be considered. In addition, an individual predisposition to gonorrhœal arthritis is illustrated by two cases already so affected in previous attacks of urethritis. The age of the patient is no factor in the frequency of the complication. A single joint was affected in 7 of the 18 cases, 2 joints in 5 cases, 3 joints in 2, and 4 in 4 cases. Of the single joints, the knee was involved twelve times, the ankle eight times, the wrist four times, the elbow twice, the shoulder and the hip each once. Six cases showed simple hydrops, 11 serofibrinous exudate, and only 1 case phlegmonous. The fever was mild and intermittent. As to prognosis, 9 cases came to restitutio ad integrum. In 5 slight stiffness remained with swelling of a joint. Two had outspoken ankylosis and 2 refused treatment. The author adds nothing from his own experience as to the results obtained with Bier's hyperemia and the newer methods of treatment.

The Anatomical and Clinical Relations of the Sphenopalatine (Meckel's) Ganglion to the Nose and its Accessory Sinuses.—Sluder (*New York Med. Jour.*, 1909, xc, 293) calls attention to the intimate relation of Meckel's ganglion to the walls of the accessory sinuses of the nose and the common wall of the nose itself. It lies close to the top of the pterygopalatine fossa just below the sphenoidal sinus. The sphenopalatine foramen is accurately placed just posterior to and immediately below the posterior tip of the middle turbinate bone; and the ganglion is close to the plane of this foramen, from 2 to 9 mm. from the general membrane of the nose. With such intimate anatomical association, clinical manifestations from the extension of inflammation or its products would seem of almost necessary occurrence. Sluder has observed characteristic disturbances follow postethmoidal and sphenoidal inflammations which cannot be explained otherwise than by assuming that Meckel's ganglion had become involved by extension. This has also occurred from the nose proper, but never, so far as known, from the maxillary sinus. In such cases of grippe origin, the pain has persisted, neuralgic in nature. The typical picture, as drawn from 47 cases, is of pain which begins at the root of the nose, extends downward over the maxilla, and backward on the mastoid, to become severest about 5 cm. posteriorly to its tip. Then extending backward, it takes in the entire occiput, and down into the neck, shoulder blade, shoulder, and sometimes the axilla. With the severest attacks it extends into the arm, forearm, hand, and fingers. It rarely invades the upper part of the head. It may extend into the brow or the zygoma. Concurrent salivation, a "metalic" taste, earache, toothache, and pain behind the eye have been noted. Sluder has applied cocaine solution to the nasal mucous membrane overlying the ganglion with success, and has injected alcohol directly into the ganglion, aborting attacks successfully.

A New Method of Recognizing Ulcers of the Upper Alimentary Tract and Determining their Localization.—Einhorn (*Berl. klin Woch.*, 1909, xlvi, 742) has devised a method for recognizing positively the presence

of ulcers in the upper digestive tract, and especially their localization. At nine in the evening the patient swallows in a gelatin capsule the so-called duodenal bucket devised by the author, to which a thread of twisted silk is made fast. This thread can reach from the lips 75 cm. in the alimentary canal. The next morning at seven or eight o'clock, while the patient is fasting, the apparatus is withdrawn. In cases of ulcer, where the thread comes into contact with the latter there is a brown or black stain on that exact portion. The distance of this from the lips gives the situation of the ulcer. With this "thread-impregnation test," ulcers in the œsophagus, cardia, smaller curvature of the stomach, pylorus, and duodenum can be recognized, whereas it is not so practical for those in the greater curvature and fundus.

Cutaneous Pigmentation as an Incomplete Form of von Recklinghausen's Disease.—F. P. Weber (*Brit. Jour. Dermat.*, 1909, xxi, 49), in 1906, reported the case of a fifteen-year-old girl in whom the skin of the trunk and neck was mottled with numerous pigmented areas varying in size and shape. The earliest pigmentation was at the back of the neck, noted by the mother when the patient was only eighteen months old. Since then it had gradually developed, and relatively faint spots and patches had recently appeared on the extremities. The variety in color and form of the areas of pigmentation and their situation (mainly on the trunk) made it seem most probable that the case was an incomplete form of von Recklinghausen's disease. Only one small, flaccid, molluscous tumor was found on the lower back. In 1908 the diagnosis was confirmed by the formation of several other molluscous-like tumors in various parts of the skin. The pigmentation was darker and new brown spots had appeared. Though the term "von Recklinghausen's disease" should be confined to cases showing, (1) obvious neurofibromas in connection with nerve trunks, (2) molluscous tumors of the skin, and (3) cutaneous pigmentation, yet Weber feels that incomplete or anomalous forms certainly occur in which one or even two of this triad of morbid features may be wanting. He would classify these forms as follows: (1) Cases of plexiform neuromas unaccompanied by molluscous skin-tumors, with or without cutaneous pigmentation. (2) Cases of multiple molluscous tumors unaccompanied by any obvious neurofibromatosis of the nerve trunks, with or without any decided pigmentation. (3) Cases of pigmentation without neurofibromas of nerve trunks or cutaneous neurofibromas. (4) Anomalous cases of neurofibromatosis complicated by the co-existence of bony or epidermic changes.

Abolition of the Corneal Reflex as a Diagnostic Sign of Hemiplegia in Coma.—Miliau (*Progrès méd.*, 1909, xviii, 229) emphasizes the importance of the loss of the corneal reflex in differentiating a hemiplegia during coma. When the cornea is touched, the lids are normally contracted at once and the eye closed. While an individual is in coma, if he has a hemiplegia, this energetic reflex is abolished. This occurs on the side of the paralysis, and is absolutely constant. Miliau considers it valuable evidence and has found it most useful. In crossed paralysis, in bulbar lesions, the loss is on the side of the facial paralysis and

opposite to the paralyzed limbs. In Jacksonian epilepsy the reflex is lost on the side of the convulsive seizures, and on both sides when the convulsions become general. This is particularly useful in distinguishing hysterical convulsions from true epilepsy. If the comatose condition is due to an intoxication, the reflex is absent bilaterally. Chloroform narcosis is an example of this. In uremia, however, reservations must be made, inasmuch as this intoxication is occasionally accompanied by partial paralysis dependent upon cerebral lesions, either toxic or due to œdema.

The Clinical Value of Antitrypsin Determinations in the Blood.—Jacob (*Münch. med Woch.*, 1909, lvi, 1361) has studied the antitryptic content of the blood in various diseases, his material numbering about 100 cases. Fuld's method was employed in the experiments. His study leads Jacob to conclude that an increase of antitryptic ferment of the blood serum occurs in a relatively large number of diseases. It is practically constant in severe cachexia, but is also very frequently present in all diseases associated with changes in the white blood cells. Finally, it may be observed in a number of cases in which neither of these factors is present. The occurrence and the cause of the reaction are still quite obscure; a positive result is obtained in such a variety of diseased conditions that no diagnostic significance can be attached to it.

A Reaction in the Umbilical Blood of Newborn Infants: A Contribution to the Study of Much's Psychoreaction.—Bauer (*Münch. med. Woch.*, 1909, lvi, 1367) has studied the reaction described by Much in the blood of patients suffering with dementia præcox, etc., in which a positive result is indicated by inhibition of cobra hemolysis after the addition of the patient's serum to the hemolytic material. The author finds that serum obtained from the umbilical cord of the newborn gave a positive result in practically all of 17 cases. The blood serum of 16 older children and adults in no case gave a positive reaction, while of 9 sucklings examined, only one was positive. The results obtained by the author indicate that the reaction is not specific, and the name "psychoreaction" should not be applied to it. The author's investigations are being continued.

Vaccine Bodies.—Czaplewski (*Deut. med. Woch.*, 1909, xxxv, 1152) obtained human, somewhat "purulent" lymph from a vaccinated child for study. Cultures on various media showed the lymph to be free from bacteria; stains also failed to demonstrate any bacteria, but the author considered the stains unsatisfactory, for no leukocyte nuclei were found in this "purulent" material. (Gram-Weigert with carbol-fuchsin counterstain, gentian violet, carbol-fuchsin and Giemsa stains were tried). By a special modification of the staining technique the author obtained very unexpected objects, which he later demonstrated also in vaccine lymph from man, the calf, and the rabbit. Further study revealed new forms which reminded the author, both in appearance and staining reaction, of the cycles of certain protozoa, especially coccidia, hemosporidia, and microsporidia. The author believes he has observed both asexual (schizogomie) and sexual (sporogonia) cycles, corresponding to

the double cycles of coccidia and malarial parasites. The asexual and sexual forms are described in various stages. In a detailed paper, soon to appear (*Zentrabl. für Bakt.*), the technique will be fully described and microphotographs published.

The Treatment of Typhoid Bacillus Carriers.—Liefmann (*Münch. med. Woch.*, 1909, lvi, 509) discusses the problem of the disposal of chronic typhoid bacillus carriers who are themselves in good health, but a source of danger to others. It is important naturally to render them harmless to the community, and yet the many researches for this purpose have been without results. But it seemed to Liefmann that good results might be obtained with the Bulgarian sour milk, the so-called Goghurt, which is such a favorable medium for the growth of intestinal flora. He fed this to three bacillus carriers for a long period, taking care that the diet be not so rich in proteins as to interfere with a strong intestinal putrefaction. After seven weeks typhoid bacilli were found in the stools of one of the individuals, but never again since, although search was kept up diligently for a period of eleven weeks from the beginning of the treatment.

The Rare Forms of Cyanosis: Polycythemia, Methemoglobinemia, and Sulph-hemoglobinemia.—Clarke (*Med. Record*, 1909, lxxvi, 143) describes the commoner forms of cyanosis due to imperfect aëration of the blood, as in heart disease, pneumonia, pleurisy, emphysema, and tuberculosis. Polycythemia is the form occurring in conjunction with a high red blood count. But the rarest of all forms is that produced by an alteration of the blood pigment itself. If this abnormal pigment is methemoglobin, the condition may be due to drug poisoning (acetanilide, phenacetin, sulphonal, trional), or auto-intoxication by the absorption of nitrites from the intestines in cases of chronic diarrhœa. If the pigment is sulph-hemoglobin, it is probably associated with chronic constipation, and is the result of the hyperformation or hyperabsorption of H_2S or the presence in the blood of an abnormal reducing agent acting with a small trace of H_2S. The methemoglobin cases improve as the enteritis is cured, and the sulph-hemoglobin cases recover if the bowels are kept open.

The Ipecac Treatment of Amœbic Dysentery.—Dock (*New York Med. Jour.*, 1909, xc, 49) emphasizes the use of ipecac in chronic amœbic dysentery, in which he has had remarkably good results. From experiments, he suggests a direct destructive action of the drug. In addition there is a cholagogue and sometimes a purgative action. Clinically, in cases treated with ipecac a cessation of amœboid motion of the parasites occurs in a few hours. In one to three days they disappear completely. In the same time the stools lose their dysenteric character. Mucus and blood, later pus, disappear, and the stools resemble those of mild enteritis or even become formed. The effect upon the other protozoa in the stools varies. The patient should always be kept in bed. The ipecac is sometimes depressing. With the idea of getting the remedy into as close contact as possible with the parasites, there should be a preliminary purgation with salts. All food should be stopped while the ipecac is being taken, and only liquids

used after it. The dose of ipecac varies with the severity of the case, from 30 to 60 grains being given at first, and then 20 to 40 grains twice a day, for three days. This is less troublesome and just as efficient as smaller doses at frequent intervals. Coating the pill with salol or keratin prevents solution in the stomach and attendant emetic action. Salivation is occasionally produced. Griping may follow, but is easily controlled with atropine.

Chronic Cholecystitis as a Cause of Myocardial Incompetence.—Babcock (*Jour. Amer. Med. Assoc.*, 1909, lii, 1964) reports 13 cases in which the effect of cholecystitis is seen in myocardial incompetency; 5 of these cases suffered with dilatation, arrhythmia, and feebleness of the heart's action plus murmurs: in short, subjective and objective symptoms of serious heart disease. Two cases had attacks of pain that had been called angina pectoris or were of an anginoid character. Evidences of myocardial inadequacy followed the attacks. A group of 3 cases had intermittence of the pulse of long standing and very intractable. Two cases of valvular disease suffered a decompensation due to outspoken attacks of hepatic colic. The explanation of the effect of the gall-bladder disease upon the heart is difficult. Several theories may be advanced: (1) The chronic myocarditis may be the result of the chronic gall-bladder infection—the circulation of bacteria and their toxins. The acute myocarditis due to an acute cholecystitis is well known. (2) The bile constituents have a depressing effect upon the myocardium. Stewart and King have proved that biliverdin is toxic to heart muscle and that bile salts increase the blood pressure. (3) Inasmuch as the splanchnics are the regulators of the circulation, their disturbance may prove an added factor by unfavorably affecting an already weakened heart and so leading to its dilatation and incompetence. (4) The disastrous effect of biliary colic upon the heart is probably a reflex result of stimulation of the filaments of the pneumogastric distributed to the wall of the gall-bladder. It is probable that there must be a predisposing cause residing in the heart muscle, a chronic myocarditis, in consequence of which the heart is unfavorably affected by influences which a healthy myocardium would be able to resist.

Meat as a Source of Infection in Tuberculosis.—Littlejohn (*Practitioner*, 1909, lxxxii, 843) states that, to the community at large, the risk of contracting tuberculosis by eating the meat of tuberculous animals is not so great as generally believed. Imperfect inspection makes the risk greater than it should be, a risk which concerns especially those who buy cheap meat and eat such commonly infected organs as the lungs, udder, and mesenteries. The relative infrequency of tuberculosis in all but cattle and pigs makes them the only food animals at all dangerous to man. The flesh of tuberculous animals is not necessarily infectious, and when so, in many instances, it is due to secondary contamination by means of the knife, butcher's cloth, and dressing. Cooking is sufficient ordinarily to destroy tubercle bacilli on the outside of a roll or joint, but not in the centre. Finally, digestion, salting, or smoking have little or no disinfecting effect. However, the rarity of tuberculous foci in the flesh, even in advanced cases, makes ordinary cooking efficient if the thick rolls and joints are avoided and the meat is well done.

SURGERY.

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The Surgical Treatment of Benign Affections of the Stomach, Especially of Gastric Ulcers and their Sequels, with Particular Reference to their Late Results.—Busch (*Archiv f. klin. Chir.*, 1909, xc, 1) says that most German surgeons are agreed that gastro-enterostomy is the normal treatment for gastric ulcer, and that resection is justified only when there exists an indurated tumor, in which there is some suspicion of cancer. Kronlein considered that operation was absolutely indicated in the presence of pyloric stenosis, in atonic gastresia and gastroptosis, and in frequently recurring slight hemorrhages. Busch reviews 124 cases of benign affections of the stomach operated on by Körte, in which 129 operations were performed. Of 76 ulcer cases, 65 were completely free of trouble more than one year after operation, 8 were able to work with slight stomach disturbances, and in 2 cancer developed in the floor of the ulcer. In 85.5 per cent. of the cases in which long continued internal therapy had failed, a successful result was obtained after operation. In 12 resections the end-results were good in all, although in 2 cases another operation was necessary because of a recurrent stenosis. He concludes that for ulcer stenosis, as well as for ulcer persisting in spite of long-continued internal treatment, the posterior, retrocolic gastro-enterostomy, by means of a circular double row of sutures, is the operation of choice. Especially for an open ulcer it will be advisable to keep up, for several weeks after operation, treatment with food of reduced acidity, with later washing out of the stomach, and a careful selection of foods. Resection should be done when there is a suspicion of malignancy, or in callus ulcer, especially when they press upon neighboring organs, as the liver and pancreas. It should always be circular according to Riedel's suggestion. In many cases it will be doubtful whether radical resection or the less dangerous gastro-enterostomy should be done. The nervous diseases of the stomach should not be operated on. There are, however, cases in which only an exploratory laparotomy will determine whether an ulcer exists or not.

The Value of Intravenous Adrenalin-Saline Solution in the Treatment of Peritonitis.—Heinecke (*Archiv f. klin. Chir.*, 1909, xc, 102) says that the excellent results obtained by Heidenhain in peritonitis, by the intravenous infusion of adrenalin-saline solution, are remarkable, in view of the transitory influence of adrenalin in raising the blood pressure. No investigator has yet observed a lasting effect on the blood

pressure, even for hours, in animal experimentation; nor has anyone observed that its influence is exerted on the vasomotor centres, so that this theory must have a purely hypothetical basis. The disturbances depend upon a paralysis of the vasomotor centres. The intravenous infusion will raise the blood pressure in every stage of collapse in peritonitis, but only for a few minutes. Its effect is no greater on the blood pressure in collapse than in a normal condition. When the salt solution is added, the effect of the adrenalin will last somewhat longer, but this is probably due to the effect of the salt solution, not of the adrenalin. The influence of the latter must, therefore, be very limited, and the success of Heidenhain must have been due to the salt solution, the influence of which on the blood pressure is much less than that of adrenalin, but much more permanent. Its effect, however, is not constant. In those cases in which there has been a loss of much body fluid its effect is very marked, and may be life saving, but when this condition does not exist it is not very valuablee.

Operations for Recurrence in Trigeminal Neuralgia.—Büdinger (*Deut. Zeitsch. f. Chir.*, 1909, xcix, 164) says that the present views concerning the dangers to which the eye is exposed in extirpation of the Gasserian ganglion are too optimistic, and that it is desirable that some method be sought which may be substituted for it. He reports a case in which the neuralgia was confined to the left inferior dental and lingual nerves. There was continuous lacrymation from the left eye, but no other disturbances in it. The two nerves were exposed within the mouth near the entrance to the inferior dental canal, and a 1 cm. piece of each removed cranialward. About a half-year later the pain recurred and was soon as bad as ever, and the patient much depressed. About a year after the first operation the trunk of the inferior maxillary division of the fifth nerve was resected to the base of the skull, after a temporary resection of the zygoma. The attacks of pain were again relieved for nearly a year, when they began again and soon became of the old severe type. Extirpation of the ganglion was now proposed to the patient, and was declined because of the risk to the eye. About ten months after the second operation the base of the skull was exposed as in the second operation. Then the bone was chiselled away in an area about 1.5 cm. in diameter, from the lowest part of the temporal fossa. The medial border of the defect was about 1 cm. distant from the foramen ovale. The bridge of bone between the two was then chiselled away. The trunk of the inferior maxillary nerve in the region of the bony canal was thickened and club-shaped, and sent fine twigs into the old scar tissue. It was isolated, and by pressing outward the dura, was followed to the ganglion, cut through, and drawn out as a whole. The hemorrhage stopped after a short compression. A piece of the bone chiselled away was properly shaped so that it fitted exactly into the foramen ovale and was wedged into the foramen to prevent the regeneration of the nerve tissue. The external portion of the bone defect was left open, and the wound was closed in the usual way. The typical attacks of pain did not recur after this operation. The chief objects of the operation were: to open up the foramen ovale in order to remove the neuromatous formation from its narrow bony canal, to remove as

much as possible of the nerve trunk, and to close the canal with bone to prevent regeneration. The fact that there has been no recurrence up to the present time, three and one-half years after the last operation, shows its advantage over the other peripheral operations, and there is the possibility that the cure may be permanent.

Fracture of the Astragalus with Luxation of the Fragments.—Mauclaire and Pettiteau (*Archiv. gén. d. chir.*, 1909, iii, 575) say that these fractures do not include dislocations complicated by fracture, since the dislocation in such cases is the dominating condition, but those cases in which the fracture is the first condition to be noted, as when a displaced fragment is detected under the skin. These fractures are very rare. The displacement is due to indirect force, the traumatism inducing secondarily forced movements of the foot in which the bone is broken and the fragments displaced. When the fracture is anterior to the interosseous ligament uniting the astragalus to the os calcis, we have a fracture of the neck or a decapitation of the astragalus. In this case the posterior fragment is fixed by the ligament and retains its normal position. The anterior fragment is caught between the weight of the body above and the bones below, so that the anterior fragment is enucleated upward under the skin on the dorsum of the foot. When the fracture is posterior to the interosseous ligament, the body of the bone loses its strong attachment and is luxated. The line of fracture may be more complicated, so that there may be several fragments. In the treatment two objects are sought after—to place the foot in good position and to obtain good function. The reduction differs from that in a fracture of a long bone, in that the fragments are more or less removed from their original location and are maintained in their abnormal position by a ligamentous apparatus which is itself dislocated. In a number of the ten collected cases a non-operative reduction was attempted without success. Such a reduction may be successful rarely, but even then it cannot be maintained with the aid of compression, and vicious callus develops, interfering with the function of the tibiotarsal joint. The frequent communication with an important joint tends to defeat consolidation and to produce a concomitant arthritis. In three cases reduction was accomplished by operation, in all of which there was a transverse fracture of the neck. The joint was freely opened and the fragments coapted. The functional results appear to have been excellent. Astragalectomy has proved to be an excellent operation. With vigorous asepsis it is a benign operation, which leaves perfect functional results, except in running and jumping. In all the cases it gave good results, except in that of Frischen, which resulted fatally. This occurred previous to the period of antisepsis. A total astragalectomy is usually preferable to a partial. In a compound fracture an astragalectomy should be done as soon as possible.

The Technique of Intrathoracic Resection of the Œsophagus.—Tiegel (*Zentrabl. f. Chir.*, 1909, xxxvi, 1009) says that the end-to-end union of the œsophagus to the stomach has certain advantages over the lateral anastomosis as recommended by Sauerbruch. It saves a valuable portion of the œsophagus which must be sacrificed for a lateral anasto-

mosis, in which it is also difficult to place the sutures. The union is also more secure than in the latter. The method suggested by Tiegel is based upon experimental work on dogs, and makes use of a modified Murphy button. This consists in giving to the male portion of the button a more expanded bell-like shape. An obturator is made to fit into it and to catch by a spring which may be loosened by light pressure of the finger. The female portion is exactly the same as in the Murphy button. After the cardiac portion of the stomach has been completely separated from the diaphragm and the vagi nerves from the œsophagus, the œsophagus is divided by a Paquelin cautery between two clamps, about 4 cm. above the stomach. After compressing the cardiac end by a piece of gauze the clamp is removed and the female portion of the button is introduced into this portion of the œsophagus and the clamp again applied. The button is then directed into a portion of the fundus of the stomach by the finger, and is held there by a compress or a loose ligature. If there is a tumor at the cardia the button must be introduced through a small incision, which must be immediately closed by a clamp or suture. The œsophagus is ligated below the tumor, and this, with the attached piece of œsophagus, is removed by a Paquelin cautery and the edges of the cardiac opening in the stomach closed by turning in the edges with sutures. Then a movable piece of stomach is drawn into a cone with tenaculum forceps, and a purse-string suture introduced around and about 1.5 cm. from the point of the cone, including the serous and muscular coats. The ends of this suture are left loose. The oral end of the œsophagus is then turned forward by the attached clamp and a similar purse string suture placed in it, a loop being left opposite the free ends. Then a compress is placed on the œsophagus above and the end of the same cut through close to the clamp. With the aid of the ends of the purse-string suture and the loop opposite, the male portion of the button is easily introduced with the obturator. The suture is then tied around the cylinder of the button. The female portion of the button lying loose in the stomach is now forced into the cone already prepared for it, and an incision is made in the point of the cone over the button and just large enough to permit the cylinder to be forced through. The obturator is now removed from the œsophageal portion of the button and the two portions pressed together and locked. The purse-string suture in the stomach wall is drawn upward so that it may be tied above the male portion without constricting the œsophagus. This brings the serous surface of the stomach against a considerable portion of the outside surface of the œsophagus, and makes a good closure of the line of union between the œsophagus and stomach.

Cancer of the Kidney; Extirpation; Cure.—Pillet (*Ann. d. mal. d. org. gén.-urin.*, 1909, ii, 987) removed a cancerous kidney about the size of the fœtal head. The incision was made along the crest of the ilium, turning upward toward the thorax and ending in the epigastric fossa. Although extensive, this incision makes a good exposure of renal tumors. The musculocutaneous flap was turned over on the abdomen and the twelfth rib resected. The tumor was then enucleated after ligating the venous plexuses uniting the tumor to its capsule. The inferior pole of the kidney was enucleated with difficulty, the promontory being

nearly reached before this was accomplished. The abundant hemorrhage was controlled by a tampon. The tumor being brought out of the wound, the ligatures were tied and reversed several times about the pedicle, the tumor extirpated, and definite hemostasis provided. Drainage and sutures were then introduced. The operative results were normal. The urine passed on the first day was 450 grams, on the second day 1200 grams, and on the third day 1500 grams. From the eighth to the tenth day, 2.5 liters, a true polyuria of convalescence was present. Microscopic examination showed the tumor to be an epithelioma after the type of a hypernephroma. Five weeks after operation the patient left the hospital and took up his normal life.

Palpation of the Ureter at the Superior Straight of the Pelvis.—Taddei (*Ann. d. mal. d. org. gén.-urin.*, 1909, i, 904) says that the origin of the common iliac arteries corresponds to the umbilicus. The course of the common and external iliacs is, therefore, outlined on the abdomen by a line from the umbilicus to the femoral pulse under Poupart's ligament, or if the femoral pulse cannot be palpated, to a point 1 cm. internal to the middle of Poupart's ligament. The bowels should have been purged, and the patient should be in a comfortable position with relaxation of the abdominal muscles by flexion of the thigh on the abdomen and elevation of the head and upper part of the trunk. The patient should breathe regularly with the mouth open. The palm of the hand is placed on the line already traced. The hand will cover the greater part of the line which will run about under the middle of the palm, the tips of the fingers being directed toward the umbilicus and reaching to about 3 or 4 cm. from the umbilicus. By deep pressure the pulsation of the common iliac artery is sought. In a large number of cases this pulsation is easily perceived. The pulsation of the common and external iliacs can be distinguished from that of the mesenteric vessels over them by the difference in force determined by their caliber. After recognizing the iliac artery by the tip of the middle or ring finger already in place, the other hand is placed in a similar position under the first, and the tips of the same fingers of the second hand, just outside of those of the first, are made to sink into the abdomen until they perceive the pulsation of the iliac artery. This will then bring out the ureteral pain. The fingers of the first hand serve to recognize the pulsation of the iliac, and to maintain the depression of the abdominal wall. This makes more easy the finding of the ureter by the hand underneath. Under favorable circumstances, that is, with lax abdominal walls, empty intestine, and thickened ureter from periurethritis, the ureter can be palpated for several centimeters.

Jejunal and Gastrojejunal Ulcers following Gastrojejunostomy.—Paterson (*Annals of Surgery*, 1909, l, 367) says that the risk of jejunal ulcer following gastrojejunostomy is probably under 2 per cent. At the present time this complication apparently occurs less frequently than formerly. Clinically there are two groups of cases: (1) Those in which perforation into the general peritoneal cavity ensues; (2) those in which general peritonitis is prevented by the formation of adhesions. Pathologically the cases may be classified as follows: (1) Ulcers of the jejunum;

(2) gastrojejunal ulcers, or ulcers at the site of the anastomosis. Jejunal ulcers are in some instances of infective origin. In these cases ulceration commences within a very short time after gastrojejunostomy, and usually the ulcers are multiple. In a large proportion of cases the ulcer is single, and is probably the result of the toxic action of HCl, which injures the cells of the mucous membrane so that they are digested by the intestinal juice. Possibly agents other than HCl may play a part in injuring the mucous membrane. Gastrojejunal ulcers are the direct result of the wound made in effecting the anastomosis, and their persistence is probably the result of hyperacidity of the gastric juice. Closure of a gastrojejunostomy opening is the consequence of cicatrization of a gastrojejunal ulcer. It is more likely to occur when the pylorus is patent, not because of the patency of the pylorus, but because in such cases hyperacidity is usually marked. Any procedure or disease which diminishes the amount of bile and pancreatic juice in the jejunum, favors the occurrence of jejunal and gastrojejunal ulcers. For this reason operations of the "Y" type and entero-anastomosis are inadvisable, at any rate in cases in which free HCl is present in the gastric contents, as after these procedures the anastomosis and a portion of the jejunum are deprived of the protective influence of the alkaline bile and pancreatic juice. The reason that ulceration has followed the anterior operation more frequently than the posterior operation with a loop, is probably that in former times the anterior operation was more frequently performed. As no instance of ulcer after the posterior, no-loop operation has yet been recorded, we must for the present assume that its occurrence after this type of operation is less likely. It is possible, however, that this immunity is partly the result of improvement in technique and in the after-treatment of gastric operations in general. In cases in which perforation into the general peritoneal cavity occurs, immediate laparotomy offers the only chance of saving the patient's life. Inasmuch as there is some evidence that jejunal and gastrojejunal ulcers may heal, an operation should not be performed in the chronic cases until after a thorough trial of medical treatment. Even when surgical intervention is necessary, an attempt should be made to diminish hyperacidity, if this be present. Our aim should be to prevent the occurence of the complication of gastrojejunostomy. Preventive treatment consists, in (1) careful and appropriate surgical technique, and (2) prolonged after treatment. Lastly, every case of recrudescent pain of constant character after gastrojejunostomy, especially when associated with hyperacidity or hypersecretion, should be regarded as a case of potential ulcer, and treated accordingly.

THERAPEUTICS.

UNDER THE CHARGE OF

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Lactic Acid as an Agent to Reduce Intestinal Fermentation.—HEINEMANN (*Jour. Amer. Med. Assoc.*, 1909, lii, 372) says that the usefulness of lactic acid or lactic ferments as curative agents for intestinal fermentation is still problematical. Much clinical evidence has accumulated since Metchnikoff recommended the use of Bacillus bulgaricus. This bacillus digests some of the casein of the milk and also decomposes some of the fats, so that the taste of the milk becomes disagreeable. Metchnikoff advised the addition of the paralactic bacillus (Streptococcus lacticus) in order to obviate this difficulty. Heinemann thinks that the association of the two organisms leaves doubt as to which one is responsible for the beneficial effects claimed. Since Metchnikoff published his work, many other lactic acid producing organisms have been advised by different observers. Heinemann also believes that the experiments which have been reported in regard to finding the Bulgarian bacillus in the feces are incomplete in so far as no statements are made in regard to the number of the bacilli found in relation to other intestinal bacteria. Recently Herter and Kendall have investigated the fate of Bacillus bulgaricus in a monkey. They failed to establish the preponderance of the Bulgarian bacillus in the ileocecal region. They found that the number of these bacilli was relatively smaller in the large intestine. Their assumption is that the introduction of lactic acid bacilli reinforce the enfeebled action of the normal intestinal lactic acid bacilli against pathological varieties of bacteria. Heinemann believes that the question of the usefulness of bacteria in the digestive tract is a debatable one—although most authorities seem to agree that the production of acids in the colon is of value for the inhibition of the growth of putrefactive bacteria. However, Heinemann says that it has not been shown that lactic acid is produced in any considerable quantity by the presence of lactic acid bacteria in the intestine. It is true, though, that clinical evidence has shown that sour milk and pure cultures of lactic acid bacilli in connection with a diet in which milk is a prominent feature are of value for inhibiting intestinal putrefaction. Heinemann tested many of the commercial preparations for the artificial souring of milk. Samples were examined bacteriologically, their lactic acid forming properties were tested, and observations made with regard to the palatability of the artificial product. Ordinary sour milk, buttermilk, and the effect of the commercial preparations on pasteurized and boiled milk were also tested. Heinemann's experiments showed that the claims for the artificial souring of milk are not justified. Furthermore, it is largely questionable whether artificially soured milk possesses any therapeutic advantages over milk naturally

soured. He thinks that Streptococcus lacticus, the organism responsible for the natural souring of milk, produces about the right palatability. When milk has solidified from the coagulation of the casein brought about by Streptococcus lacticus it contains these bacteria in almost pure culture. Milk which has reached this stage is eaten by many people as a delicacy. Raw certified milk is preferable to boiled milk for use in the souring process, as boiling alters the taste. However, he thinks it better to boil or pasteurize the milk if certified milk cannot be obtained. If this is deemed necessary, an artificial starter must be used. Any of the commercial preparations serve for this purpose—lacto-bacilline, fermenlactyl, kefil, yoghurt, or lactone. A small amount of the artificially soured milk may be used for subsequent inoculations. Metchnikoff believes that the presence of yeasts is detrimental, yet all of the commercial preparations save one contained yeasts in large numbers. Yoghurt of the Bulgarians, kefir, koumyss, leben, and all the other fermented milk beverages contain alcohol-forming yeasts. Heinemann says that the question of taste is of considerable importance, and believes that the taste produced by the activity of Streptococcus lacticus with the addition of a yeast is without doubt the most preferable.

The Treatment of Obesity.—Von Noorden (*Med. Klinik*, 1909, i, 1) distinguishes two main forms of obesity. One form is due to overfeeding in proportion to exercise, and the other is due to deficiency of thyroid function. In the first form the power of oxidation is normal, but excessive eating or lack of exercise, or a combination of both, causes obesity. In the second form the power of oxidation is reduced, though the amount of exercise may be relatively normal. The influence of the thyroid may be primary or secondary. In the secondary forms the deficient oxidation may be due to an inhibitory action upon the thyroid secretion by diseases of other organs, such as the pancreas, ovaries, hypophysis cerebri, suprarenals, or thymus. Von Noorden divides cases of obesity into these two main classes, in order to facilitate their treatment. He treats the first class by the general principles of the usual obesity cure, which is a diet of low caloric value with an abundant supply of albumin. Exercise is a valuable adjunct, and oxidation may be increased by the withdrawal of warmth. If dietetic measures fail after a fair trial, he believes that thyroid insufficiency should be suspected. He thinks that a careful and systematic thyroid treatment is much less taxing to the patient than the ordinary dietetic treatment, especially the "starvation cases." He allows his patients on thyroid treatment a liberal diet, especially of proteins. He says that dietetic measures and exercise have no effect upon cases of obesity of thyroid origin except that of weakening the patient.

The Dietetic Treatment of Obesity.—Strauss (*Therap. Monat.*, 1909, i, 17) reviews the dietetic treatment of obesity as advocated by many different observers. He thinks that the Karell "milk cure," with its various modifications, and the Rosenfeld "potato cure" are practically starvation diets. They are only applicable to those patients who can be kept in bed or at rest. These diets are especially low in

protein, so that the nitrogen equilibrium is not maintained. Consequently part of the loss of weight in such cases is due to the loss of protein substances from the body. He says that it is unfortunate that the ease of their application should be one of the chief arguments advanced for their use. Strauss' opinion is that a more liberal though definite diet and a regulated manner of living have more permanent effects than the so-called short "cures." Since the course of the disease is essentially chronic, an endeavor should be made to regulate the diet and mode of life for an indefinite time. The selected diet should be both satisfying and nutritious and as like the usual diet of the patient as possible. In short, Strauss would prefer to treat the individual case of obesity rather than to treat by a definite plan. He gives some general rules, and tables of the caloric value of the different articles of food. He divides these into three classes—foods of high caloric value, those of poor caloric value, and those of moderate value. In a proper diet the protein content should be from 100 to 120 grams, which is equivalent to from 400 to 500 calories. In the Moritz milk cure the protein content is from 45 to 90 grams, which is equivalent to from 180 to 370 calories. The nitrogen-free food should be equivalent to from 1000 to 1500 calories. In the "quick cures" the nitrogen-free food is equivalent to less than 600 calories. The question whether the nitrogen-free food should be given in the form of carbohydrates or fats is a debatable one. Ebstein, Karell, Lenhartz, Tarnier, Schindler-Barnay, Debove-Gaval, Bouchard, and Moritz supply the bulk of the necessary calories by a preponderance of fats. In Hirschfeld's diet, the calories supplied by fats and carbohydrates are nearly equal. Bunting, Hart, Kisch, von Noorden, and others favor the use of carbohydrates to supply the necessary calories. Strauss favors the last method, since patients, as a rule, tolerate the reduction of fats more easily than that of carbohydrates, especially such carbohydrates as bread and potatoes. Furthermore, carbohydrates are most easily combined with the necessary protein. Strauss believes that certain procedures may be of value to reduce weight in certain cases of obesity. Among these are various modifications of the milk cure. Thus, Lenhartz has modified the usual Karell cure to hasten the loss of fat in the ordinary treatment of obesity. He gives 800 c.c. of milk for periods of from six to eight days. Roemheld uses 1000 c.c. of milk on two days of each week, combined with a restricted diet for the remainder of the week. Boas makes use of fast days, when patients receive tea with saccharin and lemon, fat-free bouillon, the whites of two or three eggs, a few sour apples, and approximately 100 grams of whole wheat or Graham bread. This diet contains about 470 calories. The Oertel treatment and also the various milk cures have the advantage of a restriction in the amount of fluids. Furthermore, the milk cures have a low sodium chloride content. Both of these tend to a loss of weight which is not due entirely to the loss of fat.

The tables which Strauss gives are valuable and offer a great variety of foodstuffs to choose from. Some general rules which he follows in diet are valuable. He uses meat in amounts not of more than 300 grams a day. The whites of hard-boiled eggs and fat-poor cheese may be added to supply the necessary amount of protein. Green vegetables

and various salads add variety to the diet. Potatoes are allowed, and are valuable because of their satisfying effect upon the appetite. Among the vegetables which he advises are green beans, spinach, cauliflower, asparagus, mushrooms, and the cabbage group. He forbids grapes, bananas, figs, dates, and nuts. Other fruits may be eaten raw or cooked with the addition of saccharin. Bread is given preferably in the form of whole wheat bread or diabetic bread containing albumen. Alcoholic beverages are forbidden. Other beverages should not be taken with meals. They should be restricted in amount and taken between meals. The intervals between feedings should not be long, and a cup of tea, bouillon, or some raw fruit taken between meals may lessen the appetite for the next meal. The sensation of an empty stomach is to be avoided, and this can easily be done by the use of some article of food poor in caloric value.

Tuberculosis.—Wright (*Jour. Amer. Med. Assoc.*, 1909, lii, 1227) believes that mercury has a specific action in tuberculosis and gives some statistics to support this belief. He compares the results obtained by the routine treatment with the results obtained by the addition of deep muscular injections of mercury to the routine treatment. In a series of 161 cases, 83 patients voluntarily chose the addition of mercury to the routine treatment, while 78 patients preferred the routine treatment alone. Of the 161 cases, 8 were cured. Of those cured, 87.5 per cent. took the mercury treatment, while 12.5 per cent. took the routine treatment. There were 43 cases that showed marked improvement; all of these took the mercury treatment. Sixteen cases were improved; of these 87.5 per cent. had the mercury treatment, while 12.5 per cent. had the ordinary routine measures. Seventeen of the cases were classified as slightly improved; of these, 58.82 per cent. were on the mercury treatment, and 41.18 per cent. were on the routine treatment alone. Nineteen cases remained stationary; all of these took the routine treatment alone. There were 42 cases that showed an advance in the tuberculous process. Of these, 9.52 per cent. took the mercury treatment and 90.48 per cent. took the routine treatment. Sixteen deaths occurred in the series; of these, 31.2 per cent. were on the mercury treatment, and 68.8 per cent. were on the routine treatment. Combining the items cured, marked improvement, improved, and slightly improved, under the heading improved, stationary as such, and combining the items failed and died under the heading, failed, the following percentages are obtained. Of the 83 patients on mercury, 89.16 per cent. improved; none were stationary; 10.84 per cent. failed. Of the 78 patients on the routine treatment, 12.82 per cent. improved; 24.36 per cent. remained stationary; 62.82 per cent. failed. Wright gives the mercury by deep intramuscular injections in gradually increasing doses until the therapeutic limit is reached. After the maximum dose is established, it is then divided by two and the injections continued with this dosage. During the intervals between the injections the use of potassium iodide, which Wright formerly advised, is discontinued.

PEDIATRICS.

UNDER THE CHARGE OF
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Acquired Venereal Infections in Children.—F. POLLACK (*Johns Hopkins Hospital Bulletin*, 1909, xx, 142) states that yearly in Baltimore there are about 1000 cases of acquired venereal infection in children, thus proving that they are far more common than the medical profession or the laity realizes. The cause of most of the outrages upon children is found in the superstition that a person infected with either syphilis or gonorrhœa may get rid of it by infecting another, and preferably an "untouched virgin." Thus, a defenceless child is the most natural victim. Pollack has seen 189 such cases personally in the last six years. The infections run a milder course as regards complications than in adults; the duration of the disease is as long. The complications are as follows: Urethritis, 36; bubo, 28; secondary syphilis, 29; peritonitis, 19; bleeding, 16; primary sores, 7; pregnancy, 6; Bartholinitis, 3; arthritis, 3; ophthalmia, 3; etc. As treatment does not seem to influence the disease, prophylaxis seems to be the most effective remedy. In the secretions gonococci can be demonstrated very readily.

The Treatment of Facial Paralysis Due to Mastoid Disease or to the Mastoid Operation.—F. SYDENHAM (*Brit. Med. Jour.*, 1909, i, 1113) reports the case of a boy, aged five years, in whom, during an operation for mastoid disease, the facial nerve was divided, resulting in complete paralysis of that side of the face. Two days later the wound was re-opened and an end-to-end anastomosis performed; when apposed a gap of about one-half inch was noticed; this was bridged with silkworm gut. Electricity was employed from the first, but no movement of any kind could be produced for three months, when slight movements of the alæ nasi of the affected side could be observed upon forced sniffing; the other facial muscles afterward gradually returned to full power. The boy has now recovered his normal expression; when at rest and when his facial muscles are thrown into action, it is difficult to tell that there has been any facial paralysis at all. Sydenham recommends this method of anastomosis in preference to the faciohypoglossal and to the faciospinal accessory, as being more easily performed and better for the patient.

A Clinical Study of the Children of Tuberculous Parents.—J. A. MILLER and I. O. WOODRUFF (*Jour. Amer. Med. Assoc.*, 1909, lii, 1016) have made a study of 150 children of parents under treatment for pulmonary tuberculosis. The factors taken under consideration were the tuberculin test, pulmonary symptoms and signs, sputum examination, joint, bone, and other non-pulmonary lesions, the state of nutrition, hypertrophied cervical lymph nodes, tonsils, and adenoids. The following

are their conclusions: (1) In the children of tuberculous parents, who live in close association with such parents, a large proportion (in their series, 51 per cent.) become infected with tuberculosis. (2) The earliest manifestations are found in the lungs, and not in the superficial glands, bones, and joints. (3) The physical signs in children under ten years of age consist usually of persistent fine rales just without the midclavicular line in the fifth and sixth intercostal spaces; persistent sibilant rales in various parts of the chest make the diagnosis doubtful, but such cases should be regarded with suspicion. (4) The tuberculin tests, more particularly the hypodermic test in doses up to 5 mg., furnish the most reliable means of diagnosis. Of the local tests, the cutaneous is more reliable in children than the ophthalmic; it presents none of its possible dangers. (5) The ordinary examination of the sputum is almost no aid in the diagnosis of early tuberculosis in children. (6) Malnutrition is sometimes the only appreciable evidence of tuberculosis in children. (7) Hypertrophied tonsils and adenoids do not incline the balance in a suspected case toward a positive diagnosis of tuberculosis; further investigations of this subject, however, should be made. (8) The evidence that enlargement of the cervical lymph nodes is of aid as a determining factor in arriving at a diagnosis of tuberculosis in children is not conclusive.

Constitutional Eczema of Infants.—E. Feer (*Münch. med. Woch.*, 1909, lvi, 113) calls attention to chronic eczema occurring oftenest during the first few years of life and most commonly during the nursing period. In contradistinction to chronic eczema the acute form is frequently a true dermatitis; intertrigo is a true dermatitis only if coexisting with a disposition to eczema. The eczemas of older children rest oftenest upon a tuberculous base, and a positive tuberculin reaction may be obtained in almost every case. The real chronic eczema is undoubtedly a constitutional disease, but secondary dermal disturbance is quite common in their course. The two factors of importance in the development of chronic eczema are an inherited disposition and the type of nutrition; either of them may be the more important. There are two forms of chronic eczema to be found: the weeping, scale-forming eczema of the head, and the disseminated dry form. The first form is found oftenest in fat, pasty children, but even the healthiest may be affected. It begins in the scalp, spreading to cheeks and ears; it may spread to other parts of the body; there is very little itching. Overfeeding and constipation are common with these children and a spontaneous cure may be noted, when at the end of the first year a mixed diet is ordered for the child. The second form occurs almost exclusively in artificially fed infants; overfeeding is common and the patients are usually pale, thin, and flabby; chronic nutritional or intestinal disturbances are frequent. It manifests itself as discrete, dry, red, desquamating, infiltrating islands; also as papular and pustular foci. Weeping and scale formation are uncommon; part or all of the body may be involved. It is slow in development, itches, and is difficult to eradicate. Both the gouty diathesis and auto-intoxications have been blamed for this condition. Sudden deaths in children with such eczemas must be referred to "status lymphaticus" rather than to sepsis. Feer's

treatment consists of softening the scabs with oil or vaseline; this must be done gradually. He treats weeping surfaces with dilute solutions of aluminum acetate for several days, following it by a paste of oxide of zinc, starch, and vaselin. He bandages the hands to prevent scratching. Naphthalan is added in slowly healing cases, sulphur in the seborrhoic forms. Bismuth, zinc, or Lassar's salve is used in dry cases. Fowler's solution internally and slight laxation are of advantage. The amount of food must be reduced to a minimum, the quality also must be reduced. Barley water may be substituted for some of the breast feeding. A reasonable loss in weight need not be feared. Starchy food should be given in reasonable quantities to infants more than four months old; after the sixth month vegetables are allowed. Eggs and soups should be avoided under all conditions. The diet which has brought about the cure should be continued for months. In underfed children cutting down of diet is not permitted; the children should be fattened.

The Distribution of Bacteria in Bottled Milk.—Hess (*Deut. med. Woch.*, May, 1909) says that in bottled milk for children and babies, it should be considered that most of the bacteria are found in the upper layers of the cream, and become less in number in successive lower layers. The top 60 c.c. of cream holds the greater portion of the bacteria, and this holds good for the tubercle bacillus as well as streptococci and other germs. He believes that the top 60 c.c. of cream should be removed from milk intended for babies and children, instead of using the whole top part. He points out that a milk with 4.2 per cent. fat will still have 3 per cent. fat after taking the 60 c.c. from the top, and that this procedure is of benefit to the child.

The Early Symptoms of Anterior Poliomyelitis.—L. E. La Fetra (*Archives of Pediatrics*, May, 1909) has studied 63 cases of anterior poliomyelitis, to determine the more important early symptoms. Vomiting occurred in 25 cases, as a rule only at time of onset. Restlessness and irritability were common, and definitely noted in 37 cases. Tendon reflexes were absent in the paralyzed limbs in 16 out of 20 cases, and present, but sluggish, in 3. In no case in the whole series were tendon reflexes exaggerated. Cough, tonsillitis, or sore throat was noted in only 6 cases. Delirium was present in but 2 cases; convulsions in 4 cases. Rigidity of the neck occurred in 11 cases. Pain and tenderness in the affected limbs was present in 32 cases. Pain was in the muscles in 5 cases, on movement of joints in 4 cases. The latter is important, as possibly confusing the diagnosis with cerebrospinal meningitis and neuritis. The paralyzed limb was flaccid in 58 cases. It was spastic or rigid in only 5 cases. Paralysis came on early. It occurred on the first day in 24 cases; on the second day in 9 cases; on the third day in 3 cases; after two weeks in 4 cases. Paralysis was occasionally noted in the muscles of the neck, face, back, and abdomen. Also paralysis of the bladder and general anesthesia. Leukocytosis varied from 13,000 to 20,000.

OBSTETRICS.

UNDER THE CHARGE OF
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Ligation or Excision of Thrombosed Veins in the Treatment of Puerperal Pyemia.—Williams (*Amer. Jour. Obst.*, May, 1909) reports 5 cases of puerperal pyemia in which the veins of the broad ligaments were ligated or resected. In some of these cases an enlarged septic tube was removed; 4 of the 5 cases recovered, one of them being complicated by pneumonia and pleurisy. Williams has collected from the literature 15 cases of extraperitoneal operation, with 12 deaths—a mortality of 80 per cent.; 10 cases of transverse peritoneal operation, with a mortality of 70 per cent.; 5 cases of ligation or excision of both spermatic veins after laparotomy, with 60 per cent. mortality; 4 cases of ligation or excision of one spermatic or hypogastric vein, with 25 per cent. mortality; 2 cases of ligation of both spermatics and one hypogastric vein, with 50 per cent. mortality; 8 cases of ligation or excision of both spermatic and hypogastric veins, with 25 per cent. mortality; 2 cases of ligation of one hypogastric vein, with 100 per cent. mortality; 3 cases, in which no details are given, of ligation of the broad ligament veins, with 66.6 per cent. mortality. Williams concludes that the average mortality of puerperal pyemia is in the neighborhood of 66.6 per cent., which justifies any operation offering a chance of improvement. His paper is based upon the study of 56 cases of thrombophlebitis treated by the excision or ligation of one or more pelvic veins: 15 operations by the extraperitoneal method gave a mortality of 80 per cent.; 41 cases by the transperitoneal method gave a mortality of 43.9 per cent. This is no especial improvement upon expectant treatment. If, from these cases, those are deducted which were not susceptible of cure, and in which the technique was faulty, the corrected mortality for extraperitoneal operation was 40 per cent., and for transperitoneal operation 21.4 per cent. In Williams' 5 cases the gross mortality was 20 per cent. Early operation for thrombosis limited to the spermatic vein should not give a mortality exceeding 10 per cent., as compared with 25 per cent. when other vessels are involved. A positive diagnosis can be made when a worm-like mass can be palpated at the outer portion of the broad ligament in patients suffering from chills and hectic temperature. Operation should then be undertaken. Excision of the thrombosed vessels is rarely necessary, and when the vessel appears likely to rupture or is surrounded by periphlebitic inflammation, excision should be undertaken; otherwise ligation only should be performed. The transperitoneal is preferable to the extraperitoneal method. The transperitoneal operation is easier, gives a better view of the vessels, and scarcely increases the danger of peritoneal infection. Laparotomy should be done in all cases in which interference seems indicated, because the vaginal route is applicable only to a small class of cases in which thrombosis is limited to the vessels of the broad ligament

exclusively. A diagnosis of this condition can rarely be positively made.

Exophthalmic Goitre Complicating Pregnancy.—Stowe (*Amer. Jour. Obst.*, May, 1909) reports a fatal case of exophthalmic goitre complicating pregnancy. The patient was aged twenty-four years, and had the appendix removed at the age of twenty-one. At this time the peritoneum was the seat of miliary tuberculosis. She afterward aborted at two and a half months, probably due to the goitre, as the symptoms had made their appearance a few months before. When pregnancy again occurred the characteristic signs of goitre were present. The thyroid gland was slightly enlarged, chiefly on the right side. Under rest in bed and appropriate treatment, the patient's general condition improved, but the ocular signs and enlarged gland remained unchanged. Shortly after pregnancy occurred the patient vomited incessantly for eleven days, and her condition rapidly became desperate from acute toxemia. The uterus was emptied so far as possible, under ether, but the patient died in coma three days afterward. An autopsy could not be obtained.

Symphysiotomy.—Prentiss (*Amer. Jour. Obst.*, May, 1909) reports the case of a white woman whose first labor terminated after a very difficult forceps extraction, with the death of the child. The pelvis was symmetrically contracted 2 cm. in each diameter. At the eighth month of pregnancy rapid dilatation was performed under ether, the forceps applied above the superior strait, and traction made for about one-half an hour when the head failed to descend. The pubic joint was then opened, the bones separating 6 or 8 cm., the child being readily delivered. No injury to the bladder, urethra, or vagina occurred, and the bones were wired together. The patient died suddenly on the third day, after being moved. At autopsy no evidence of sepsis was present, but thrombosis had developed in the veins of the uterus, left broad ligament, left ovarian vein, and right heart. The child survived. This case draws renewed attention to the danger of unsuccessful attempts at forceps delivery.

The Clinical Value of Opsonins in Pregnancy and the Puerperal State.—Guggisberg (*Zeitschr. f. Geb. u. Gynäk.*, 1909, lxiv) reports the results of his experiments in pregnant and parturient women. The spontaneous phagocytosis of the human leukocytes is present in staphylococcic and streptococcic infection. In normal human serum substances are found which increase the phagocytosis of the staphylococci and strepcococci through the cells of the human tissue. In pregnant patients the opsonic index is not the same, and varies in a number of pregnant patients, as regards both bacteria, from the average of the normal human being. In some pregnant patients the opsonic index is normal. In the puerperal period the opsonic indices for staphylococci and streptococci are not similar, but vary greatly in different patients. It was impossible to estimate precisely the cause of this variation. As a general result of his investigations Guggisberg could not find a definite value for the opsonic index in prognosis and diagnosis in septic infections of pregnant and parturient women.

The Results of Hebostiotomy from the Study of Seven Hundred Cases.—Schläfli (*Zeitschr. f. Geb. u. Gynäk.*, 1909, lxiv) obtained the following results in the study of 700 hebostiotomies: In 510 cases there was considerable hemorrhage during the operation in 10.98 per cent.; the bleeding became profuse in 2.94 per cent., and abundant in 1.37 per cent. Taken in all, hemorrhage was greater than normal in 15.3 per cent. In 664 cases, hemorrhage occurred during operation in 0.3 per cent.; in 510 cases there was hematoma or laceration communicating with the vagina in 15.49 per cent. This was most frequently observed among primiparæ in 32.5 per cent.; among multiparæ less frequently, 11.88 per cent. Simple lacerations of the birth canal occurred in 17.84 per cent. The mortality of those patients having lacerations communicating with the vagina was 12.66 per cent. A fatal result could be directly traced to the lacerations in 31.25 per cent. In fatal cases, lacerations of the genital tract were a potent factor in 40.6 per cent. Injuries to the bladder occurred in 12.35 per cent.; fever during the puerperal period in 31.76 per cent.; and thrombophlebitis in 8.23 per cent. There was hernia between the edges of the cut bone in 7.5 per cent. after the operation; prolapse of the vagina in 24.17 per cent.; and incontinence of urine in 4.17 per cent. The corrected mortality among the mothers following the operation was 4.37 per cent.; the corrected mortality for the children was 9.18 per cent.

Correlation of the Uterus and Ovaries.—Bell and Hicks (*Brit. Med. Jour.*, March 13, 1909) report, from their studies of the correlation of the uterus and ovaries, that the removal of the ovaries during pregnancy was followed invariably by abortion. The internal secretion of the ovary is evidently favorable to the continuation of pregnancy—because of the relation between the ovary and the calcium metabolism of the body. In the human subject, however, this secretion is not absolutely essential in all cases.

Elective Cesarean Section.—Porter (*Jour. Amer. Med. Assoc.*, March 20, 1909) has collected the report of 126 cases of elective Cesarean section performed by thirteen American operators upon uninjured, uninfected, and unexhausted women. The maternal mortality of the series was 1.58 per cent., with no fœtal mortality. The maternal mortality of the high forceps and version is stated at 1.14 per cent., with a fœtal mortality of 17.3 per cent. The maternal morbidity of Cesarean section in this series was 12.69 per cent., and that of high forceps or version, 42 per cent. The fœtal morbidity in Cesarean section was nothing, compared with the fœtal morbidity of high forceps and version of 12.2 per cent. Porter urges that in contracted pelves Cesarean section is the operation of choice, especially in primiparæ. If the child is viable Cesarean section should be performed in central placenta prævia. In eclampsia, at or near term, in a primipara, abdominal Cesarean section should be chosen, and when the child is small, and the vagina and pelvis capacious, vaginal Cesarean section should be selected. In cases requiring abdominal section at term, or the removal of uterine ovarian tumors, the child should be delivered by Cesarean section immediately before or after the removal of the tumor. In elderly primiparæ at term, with a vigorous child, a normal pelvis, but

rigid soft parts, unusually sensitive to pain, in bad physical condition, and with bad nervous equilibrium, early Cesarean section offers the best chance for life and health to mother and child. The fact that Cesarean section entails no risk upon the child should cause the operation to be limited to those cases in which the child is in good condition.

GYNECOLOGY.

UNDER THE CHARGE OF

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Menstruation and Menorrhagia.—Florence E. Willey (*Jour. Obst. and Gyn. Brit. Emp.*, 1909, xv, 236) says we may still regard the monthly cycle as a preparation of the uterus for the development of an impregnated ovum, but we must realize that the important physiological requirement is an efficient blood supply to the uterine musculature. The changes in the mucosa are such as would occur in any congested state of the uterus with overfilled capillary vessels, and are probably secondary, the primary changes being in the muscular tissue. Anatomical studies of the capillary system of the uterus during menstruation and during the time of rest show that parts of this system are functional, that is, many capillary fissures are closed during the intermenstrual period and become filled with blood, and act as part of the general vascular system during menstruation and pregnancy. Clinical observation shows that the uterus increases in size during menstruation. Willey also says that uterine muscle possesses, in addition to its function of contractility, a possibility of hypertrophy unknown in any other muscle. Assuming all the above is true, Willey then suggests that menorrhagia should direct attention to the blood supply to the muscular wall of the uterus rather than to changes in the endometrium. Therefore, in the treatment of those cases of menorrhagia, in which newgrowths of the uterus or tubal disease may be excluded, general and local conditions that bring about uterine congestion or inefficient action of the uterine muscle must be sought. Massage of the uterus is condemned.

Tuberculous Peritonitis.—Broun (*Amer. Jour. Obst.*, 1909, lix, 812) discusses the various factors in the etiology of tuberculous peritonitis especially mentioning the dictum of J. B. Murphy that the chief avenue of infection is through the Fallopian tubes and from coitus with tuberculous men. From Leuret the following conclusions are adopted: (1) Genital lesions are the most frequent cause of peritoneal tuberculosis in women, in particular the clinical type known as idiopathic ascites in young women. (2) Tuberculosis of the adnexa is always accompanied by tuberculous peritonitis; the form occurring being one of two types—ascitic peritonitis, free or encysted, and dry pelvic peritonitis with adhesions. (3) The type of pelvic peritonitis with adhesions is

subject to frequent exacerbations. In the surgical treatment of this lesion Broun recommends ablation of both Fallopian tubes, and, if suspected, after careful palpation and inspection, the vermiform appendix. Complete closure of the abdominal wound without drainage is advocated. With hygienic after-treatment the use of tuberculin is advised.

Aneurysm of the Uterine Artery.—Reymond (*An. de gyn. et d'obst.*, 1909, xxxvi, 117) presented to the Société Anatomique de Paris a specimen of aneurysm of the uterine artery removed by an abdominal operation. The symptoms were those of cancer of the corpus uteri after the menopause. To touch, the uterus was found displaced slightly to the left and the pulsations of the aneurysm in the right lateral part of the posterior fornix vaginæ were evident. The aneurysm extended from the origin of the uterine artery to the uterine cornu, and was exsected. The dilatation was the size of the thumb. Hemorrhage did not occur after the operation.

Resection of Seventy Inches of Intestine after Perforation of the Uterus.—Barton and Smalley (*Jour. Amer. Med. Assoc.*, 1909, lii, 1665) add another to several cases recently appearing in medical literature of perforation of the puerperal uterus and pulling through the opening a considerable length of the intestine. The case reported was one of perforation of the uterus with ovum forceps in using them to complete an abortion. Barton and Smalley were at once called and opened the abdomen, encountering much free fluid, blood, and a hole one and a half inches wide through the uterus. From the uterus the incarcerated intestine was drawn back into the peritoneal cavity. A four or five months fœtus and placenta were then withdrawn from the uterine cavity by enlarging the opening. A portion of the jejunum and of the ileum, seventy inches in length was found to be detached from its shrunken and black mesentery. It was resected and an end-to-end anastomosis of the intestine was made successfully. An interesting incident was that a long-standing intestinal indigestion has not recurred during the eighteen months elapsed since the operation.

Treatment of Myoma.—Kurt Torkel (*Monatsschr. f. Geburtsh. u. Gynäk.*, 1909, xxxi, 338) reports 303 cases of myoma observed at Breslau during 1902-1908. A study of these cases shows that radical measures are not invariably indicated, while, on the other hand, too much reliance should not be placed on palliative measures which may unnecessarily prolong the condition. Curettage is indicated in suitable cases, but atmocausis and internal medication are to be denounced. Operative intervention should always meet with the requirements of the individual case. With the exception of special cases, conservative methods are of value only when retention of the uterus would prove of value to the patient. In all other cases, as a rule, supravaginal amputation should receive preference over abdominal total extirpation.

The Diagnosis of Tuberculous Peritonitis.—J. N. West (*Amer. Jour. Obst.*, 1909, lix, 820) states that in fully one third of the cases of tuberculosis of the peritoneum operated upon the previous diagnosis was

ovarian cyst, and that in another third the diagnosis was pyosalpinx of the other forms. Quoting C. H. Mayo, West states that in 78 per cent. in the United States and in 89 per cent. in Germany of all cases in which necropsies have been held, there is shown the presence of, or the evidence of, past existence of tuberculosis. Of 13,922 necropsies collected by Grawitz and Brun, 2 per cent. showed tuberculous peritonitis. The proportion of women to men as victims of tuberculous peritonitis is said to be 5 to 1, and the most common period of infection is from twenty to thirty years. The chief points given to establish a clinical diagnosis are recurrent attacks of pelvic peritonitis extending over several months; gradual accumulation of fluid in the abdomen; hectic, afternoon or evening, temperature; gradual and progressive loss of weight and strength, and sensitiveness of the abdomen. The clinical diagnosis failing, the aid of other tests may be applied. Of these the skin inoculation test of von Pirquet, is preferred to that of the Calmette eye installation or the skin inunction test of Moro. While these are not infalliable tests, the data thus obtained are valuable. Curiously enough, when considered superficially, the absence of reaction is more reliable than the occurrence of a reaction.

Perforation of the Stomach and Small Intestine as a Sequel to Ovariotomy and Hysterectomy.—J. Bland-Sutton (*Jour. Obst. and Gyn. Brit. Emp.*, 1909, xv, 197) reports three cases of perforation of the stomach or the small intestine after ovariotomy or hysterectomy that have come under his observation. Two of them died on the eighth day after operation. The third died forty-six hours after hysterectomy. Bland-Sutton refers to casual reference to the personal experience of von Rosthorn and Olshausen with this complication, and appeals to gynecologists to report similar experiences.

Hernias through the Pelvic Floor.—Barrett (*Amer. Jour. Obst.*, 1909, lix, 553) believes that in the treatment of hernias of the pelvic floor consideration should be given to the following principles: (1) The abdominal organs need for support, in addition to their own ligaments, a limiting wall. (2) The limiting wall should grow stronger from above downward, as gravity of the organs exerts a force not exerted above. (3) The pelvic floor, being the lowest part of the abdominal wall, needs to be strongest. (4) Abdominal organs herniate through the walls by reason of increased pressure, insufficient supports, and weak places in the walls or "faults." (5) The pelvic floor, does not act as a direct support to the uterus, as the uterus does not lie against it. (6) The pelvic floor by reason of its importance in man has developed an additional diaphragm—the levator ani muscle and its fascia. (7) This diaphragm is weakened by "faults" (the rectum, vagina, and urethra). (8) The vaginal canal, by reason of extreme dilatation which lacerates the surrounding levator muscle, becomes a frequent site for hernia. (9) Atavism of the levator ani muscle occasionally furnishes a congenital predisposing cause of hernia through the rectum in the male, and the rectum and vagina in the female. (10) The cure of these hernias, due either to traumatism or congenital defects, should look toward, (*a*) lessening intra-abdominal pressure; (*b*) correcting the pelvic floor defect; (*c*) putting the vagina out of a vertical line;

(*d*) putting the uterus out of line with the vagina. (11) The posterior segment of the pelvic floor is repaired by uniting the levator ani muscle and its fascia, and the lower end of the vagina is thereby pushed forward. (12) The anterior segment may be corrected by anterior colporrhaphy and, if occasion demands, a shortening of the sacro-uterine ligaments. The upper end of the vaina is thus pushed backward. (13) The uterus is got out of line with the vagina, (*a*) by shortening the round ligaments; (*b*) by vaginofixation in cases of mild prolapse past the menopause; (*c*) by vaginal hysterectomy in cases of marked prolapse, especially with a pathological uterus; in case of hysterectomy the ligament should be implanted into the vaginal vault; (*d*) by bringing the lower end of the broad ligaments in front of the cervix, according to Alexandroff, which operation is slightly modified in this country by Dudley and others. Barrett emphasizes that the pelvic floor is an important factor in abdominal support and that the chief structure is the levator ani muscle; that the tendency of the vagina to become a hernial canal is greatly increased by traumatism and congenital defects. In slight cases rest and lessened intra-abdominal pressure are recommended; in exaggerated cases repair of the pelvic floor, lessening of the vertical position of the vagina, removal of the uterus, or increasing its angle to the vagina should be done.

OTOLOGY.

UNDER THE CHARGE OF

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Ear Disorders in Diseases of the Urogenital Apparatus.—Dr. J. Sendziak (*Archiv f. Ohrenhlk.*, 1909, lxxiii, 55), presents an interesting summary of the several ear affections that may complicate diseases of the genito-urinary organs. Considering first the kidneys, he points out the occasional occurrence of hemorrhages into the tympanum or labyrinth in the course of severe acute or chronic parenchymatous nephritis; sometimes these hemorrhagic spots can be observed on the drumhead, particularly in the anterior upper portion. The clinical symptoms, which suggest an aural examination in nephritis, are sudden defects of hearing, subjective sounds, and giddiness. He lays especial stress upon the frequency with which purulent otitis media is associated with that form of nephritis that follows scarlet fever, pointing out a prognostic significance of this connection; it having been repeatedly observed that an increase in the otorrhœa accompanies an exacerbation of the kidney trouble, and vice versa. Regarding the relationship between aural affections and diseases and abnormalities of the genital organs, he finds such instances much more frequent among females than males. Vicarious menstruation, when hemorrhage from the ear sub-

stitutes the normal discharge, is said to have been noted six times in 200 vicarious cases. The appearance of a mild dermatitis of the auricle or canal at the monthly period, or during pregnancy has been often observed, and the stubbornness of such inflammations when occurring about the climacterium is dwelt upon. Perhaps the most important point mentioned in the paper refers to the unfavorable influence sometimes exerted upon aural affections by pregnancy and childbirth; any existing pathological process in the ear may suffer a considerable exacerbation during the period of gestation, and only exceptionally does there follow a reclamation of the hearing power lost in such cases.

A Pure Transudate in the Middle Ear.—Walb, of Bonn (*Archiv f. Ohrenhlk.*, 1909, lxxiii, 317), refers to the discussion which took place at the last session of the German Otological Society, at Bremen, as to the possibility of a sterile exudate or transudate into the tympanic cavity occurring. He holds, with Kümmel, Schiebe, and others, that there are many instances of pure intratympanic exudate, resulting from negative intratympanic pressure, and that such fluids, naturally, are sterile. In support of his contention, he cites clinical cases in which the middle ear disturbance arises from closure of the tympanopharyngeal tube, and in which an incision of the tympanic membrane disclosed the following conditions: Sterile fluid evacuated; the tympanic mucous membrane presenting characteristic appearances of tissue under negative pressure; an incision that remained sterile, even though unhealed, for weeks; and, in some cases wherein the causes of negative pressure had not been removed, a recurrence of the transudate shortly after healing of the incision. Walb's views coincide with the commonly accepted views here that the simple exudative otitis medias, occurring without inflammatory symptoms, usually in elderly persons or in those who have stricture or obstruction of the tympanopharyngeal tubes, are not of bacterial, but rather of mechanical, origin.

The Surgery of the Labyrinth of the Ear.—During the past five years operations upon the labyrinth have been quite frequently performed, and the revived study of the physiology and anatomy of this portion of the organ of hearing, incited by these surgical investigations, together with the clinical experience resulting therefrom, is giving a broader knowledge of the internal ear affections and holding out some promise of a more enlightened treatment in the future of these previously ignored or neglected conditions. It was quite natural to pursue further the excellent surgical work that has developed in the treatment of suppurative otitis media, and to open the semicircular canals or the cochlea when, in the course of a mastoidotympanal exenteration, they were found to be diseased. The symptoms of labyrinthine invasion associated with purulent otitis media are not yet so clearly defined as to make it always possible to diagnosticate the condition prior to operation, but great progress is being made in that direction. Meanwhile, all cases of chronic suppurative otitis that present evidences of labyrinthine disturbance, such as frequent or continued headaches, severe tinnitus, nausea and vomiting, or great deafness, are being carefully investigated during the course of the operative intervention that is necessary to cure the middle-ear disease.

In an interesting paper on "Indications for Labyrinth Opening in Complicated Middle-ear Suppurations and New Suggestions as to Methods," W. Uffernorde (*Archiv f. Ohrenhlk.*, 1909, lxxiii, 227) discusses these questions at some length. He points out the fallibility of all known tests to indicate invasion of the labyrinth by purulent processes, and the dangers of operating in this region; so great is the risk, and so frequently do what were apparently symptoms of labyrinthine disease disappear after eradication of the disease from the middle ear and mastoid cells, that he cautions against needless or unnecessary operations upon the internal portion of the ear. Even when carious openings in the bony wall of the semicircular canals are discovered during an operative exploration, it must be remembered that this process may be superficial, may not yet have broken through the membranous capsule, and so a curettement of the labyrinth may not be called for. In the cases of pronounced purulent labyrinthitis the results of surgical treatment have been brilliant; such conditions are practically always fatal unless treated by operation. The difficulty at present lies in determining which cases shall be submitted to operation.

Much has been recently reported concerning the clinical value of the caloric tests of equilibrium, and it might be supposed that these, added to von Stein's tests (walking, jumping, rotation, etc.), would give certain rules by which to ascertain the location and extent of a labyrinthine lesion. Uffernorde discourages dependence upon these tests, while encouraging further study of them, and says that he has seen all the rules clinically reversed. If at the present the situation seems to be somewhat doubtful, it is certainly hopeful and there is prospect of important discoveries in this field. It will not be surprising if in the very near future the labyrinth of the ear becomes quite as much an open book to the otologist as is the tympanic cavity now.

In this connection it is interesting to note that much attention is being given to the study of aural vertigo and tinnitus aurium. These are conditions that have long baffled observers and about which very little is known with certainly. Matte (*Archiv f. Ohrenhlk.*, 1909, lxxiii, 142), in an article on "The Surgery of the Labyrinth," considers an operation for the cure of distressing tinnitus. He recites the character of the serious type of this affection, the noises sometimes being so serious and persistent as to drive the victim to the contemplation of suicide, and recommends in such cases operative ablation of a portion of the labyrinth. He believes that by a careful study of the nature of the sound heard, and comparison of it with other measurable sounds under control of the investigator, it may be possible to localize the lesion, and thus determine what part of the vestibule or cochlea to attack. He reports a successful operation of partial ablation of the labyrinth, the details of which are interesting as showing what may be conservatively effected. Section of the auditory nerve has been successfully done for the relief of subjective sounds, but this totally destroys hearing, while Matte's suggestion, if it further proves to be practicable, may serve to relieve the patient of a distressing affection without completely destroying the hearing.

PATHOLOGY AND BACTERIOLOGY.

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The Action of Narcotics and Alkaloids on the Complement.—Goldschmidt and Pribram (*Ztschr. f. exp. Path. u. Ther.*, 1909, vi, 211) have studied the action of various narcotics and alkaloids on hemolytic sera and have obtained the following results: The narcotics of the aliphatic and aromatic groups as well as powerful brain and nerve poisons in general (especially the alkaloids) exert a group of actions which stand in causal relationship to one another. (1) The action on lecithin suspensions as well as on other colloidal suspensions is either solution or precipitation; subsequent solution of the precipitate may take place. (2) Fresh hemolytic sera lose their power to destroy red blood cells when subjected to the action of the above-mentioned poisons. This action is due to a change in the physical condition of the medium and can be reversed by again adding sufficient fresh serum. The phenomenon is probably similar to the inactivation by heating to 60° C., since both procedures are accompanied by lowering of the surface tension. (3) Red blood cells are laked by powerful brain and nerve poisons with few exceptions. (4) The action of erythrocytic and cerebral bacterial toxins (tetanus, diphtheria) is weakened or prevented by these poisons. (5) All of these actions are of physical nature and are due to the intense physical activity of the above-mentioned substances. (6) Only poisonous alkaloids produce this phenomenon, and they probably stand in close relationship to their chemical constitution.

The Origin of Nephritic Œdema.—Julius Bence (*Zeit. f. klin. Med.*, 1909, lxvii, 69), in order to elucidate certain problems in connection with œdema in nephritis, studied the fluid content of the blood in normal and nephrectomized rabbits, and in rabbits in which a nephritis had been produced by injections of uranium nitrate. When rabbits whose kidneys are normal are deprived of water, there is a slow but constant loss in weight with a decrease in the fluid content of the blood. If such rabbits are given sufficient water by the stomach to keep the weight at a constant level, that is, from 100 to 150 grams of water a day, the fluid content of the blood is not perceptibly altered. Nephrectomized rabbits when deprived of water also show a gradual loss in weight, but present interesting changes in the fluid content of the blood. At first this rises slightly, then falls temporarily, but finally increases steadily until the animal dies. With the hydremia, which continues in spite of the fall in body weight, œdema may develop, since three of the eight nephrectomized animals showed ascites. The ingestion of 100 to 150 grams of water a day, sufficient to keep the body weight at practically a constant

level, gives rise to a more pronounced hydremia and uniformly to œdema. Rabbits in which a nephritis has been produced by means of uranium nitrate, deprived of water, lose weight slowly during the first day; on the second day there is a sharp fall in weight synchronous with the diuresis produced by the nephritis. On the third day anuria makes its appearance and the body weight remains about the same. A drop in the fluid content of the blood accompanies the diuresis, but with the onset of anuria hydremia sets in and increases during the production of œdema and until death. In three of the eight animals there was ascites. Injection of water in the same amounts as was used in the other experiments gave rise to more hydremia, which started before the development of anuria, and was accompanied constantly by œdema. Uranium nitrate injected into nephrectomized animals did not influence the course of events, which seemed to show that the effects obtained by the use of chemicals are not due to injury to the vessel walls, but simply to the disturbance which it sets up in the kidney. The experiments show that when the kidneys are removed or made functionless through disease, there is an interchange of water from the tissues into the vessels giving rise to hydremia. A second interchange of fluid takes place between the blood and tissue spaces or serous cavities. These phenomena occur even though there is no addition of water to the body, though if water is obtained from outside the body, the effects, that is, the œdema, are much more marked.

The Relation of Typhoid Bacilli to the Gall-bladder.—Another evidence of the importance of the role played by bacillemia in typhoid fever is afforded by the experimental work of Chiarolanza (*Ztschr. f. Hygiene u. Infektionskr.*, 1908, lxii, 1) on the relation of typhoid bacilli to the gall-bladder and gall-ducts. He found that if bacilli are injected into rabbits subcutaneously, they do not reach the gall-bladder; but after intravenous injection they were found in 17 out of 23 cases (74 per cent.) The shortest time for them to reach the gall-bladder was two hours; the longest period of their excretion from it, fifty-eight days. To determine in what way they entered the gall-bladder, the cystic duct was ligated shortly before injecting the bacilli. In these cases also, bacilli were found in the bladder, showing that they had entered not with the bile, but through the capillaries of the mucosa and submucosa. Also, if the common duct was ligated, bacilli could be found in the intestine, showing that here also they might invade by way of the capillaries. On histological examination the infected gall-bladder showed degeneration of the epithelium in acute cases; proliferation of the epithelium with thickening and infiltration of the submucosa, in the more chronic forms. Necrosis of the liver was never found.

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ORIGINAL ARTICLES.

INTESTINAL PERFORATION DURING TYPHOID FEVER IN CHILDREN.

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The modern conception of the symptomatology, diagnosis, and treatment of typhoid perforation in general would seem to be firmly based upon the mass of carefully studied material which is now available. Aside, however, from text-book articles, limited series of personal observations, and scattered case reports, there was but little systematic work done on the subject of typhoid perforation in children with respect to treatment by operation, until the appearance of the paper by C. A. Elsberg[1] in 1903. This paper was based upon a study of 25 cases of perforation operated upon in children, all that were available at that time. While the number of cases was small, the deductions which Elsberg was able to draw were of great interest and importance. It has seemed to us worth while to study the cases occurring in children, and reported since 1903, with a view to confirming, if possible, the rather remarkable findings of Elsberg; and, at the same time, to consider briefly those points in

[1] Annals of Surgery, 1903, xxxviii

the clinical course of typhoid fever in children that have a bearing on the diagnosis of perforation and to compare these with the symptoms commonly observed in adults.

Frequency. Perforation in children is usually considered to be relatively infrequent. That this is true of infants and very young children is undeniable. Typhoid fever in young children is not often attended by the grave intestinal lesions which are found in older children and adults. Ulcerations of the bowels are few in number and superficial in character, and we find what is commonly spoken of as the "nervous type" of the disease rather than the abdominal type, which becomes the rule as we approach the age of puberty. Hemorrhage and perforation in the earliest years of life are, therefore, very infrequently encountered. It is not until after the age of four or five years that the intestinal lesions become of a character to favor perforation of the bowel, but from this time on the accident becomes of correspondingly greater frequency. We have collected from various sources 2274 cases of typhoid fever in children in which perforation occurred 35 times (1.54 per cent.).

In some of the series studied, notably that of Berg,[2] 154 cases with 4 perforations, or 2.6 per cent., the percentage was higher. Morse[3] collected 284 cases with no perforations. Griffith and Ostheimer,[4] in 302 cases under two and one-half years, found 2 perforations (0.66 per cent.). Two cases mentioned by Griffith and Ostheimer, which occurred at the ages of twenty and twenty-one months, respectively, are the youngest authentic examples of which we have knowledge.

We find that the percentages of perforation in typhoid fever at all ages vary from 1.5 (Skutesky) to 3.03 (Murchison) and 3.66 (Armstrong). Harte and Ashhurst,[5] in an analysis of 8881 cases, estimate the frequency of perforation at 2.54 per cent. We have collected 4947 cases, with a percentage of perforation of 2.19.

The relative frequency of typhoid perforation in children and adults is somewhat difficult to estimate, as the figures just quoted include cases of all ages; but it may be safely said that while perforation occurs less frequently in children, the disparity is less than is commonly supposed, the complication being probably slightly more than half as frequent in the junior class.

Age. In collecting cases of perforation in children for study we have fixed the usual age limit of fifteen years; we have analyzed only cases in which operation was performed and perforation proved; we have not considered cases of peritonitis occurring without perforation, nor have we studied cases of perforation of the appendix in which this lesion was manifestly due to a preëxisting or coincident

[2] Deutsch. Archiv f. klin. Med., 1895, vol. liv.
[3] Boston Med. and Surg. Jour., February 20, 1896.
[4] Amer. Jour. Med. Sci., November, 1902.
[5] Annals of Surgery, 1904, xxxix.

appendicitis not due to typhoid ulceration. Reported since the publication of Elsberg's paper in 1903, we have found 45 cases in subjects fifteen years of age and younger, the exact age being given in 44. These are as follows:

Case I.—Staunton. Med. Press and Circular, 1903, 609.
Case II.—Cotton. Boston Med. and Surg. Jour., 1906, clv, 151.
Cases III.—Russell. Montreal Med. Jour., 1903, 584.
Cases IV and V. Hays. Jour. Amer. Med. Assoc., 1905, 1267.
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Case XXVII.—Stewart. Amer. Jour. Med. Sci., May, 1904.
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Case XXIV.—Kaehler. Deut. med. Woch., 1907, No. 34, 1370.
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Cases XXXVI and XXXVII. Griffith and Hutchinson. Unpublished, Children's Hospital Histories.
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The youngest were aged five years, of which there were four; two recovered, and two died. There were three patients aged fifteen years, all of whom died. The numbers and ages were as follows:

	Years.		Years.
4 cases at	5	2 cases at	11
4 cases at	6	4 cases at	12
2 cases at	7	3 cases at	13
7 cases at	8	5 cases at	14
4 cases at	9	3 cases at	15
6 cases at	10		

Of 21 cases under ten years of age, 12 recovered and 9 died, a mortality of 43 per cent.; of 23 cases ten years of age or over, 10 recovered and 13 died, a mortality of 56.5 per cent.

This shows apparently a less mortality in young children, which is further corroborated by an analysis of Elsberg's table. In his

series, of cases under ten years of age, the mortality was 20 per cent.; in cases ten years of age and over, the mortality was 46.6 per cent. As we approach the age of fifteen, the mortality approximates that of adult life.

Sex. The sex was given in 43 of our 45 cases: 28 were males and 15 were females, a disparity much less than that observed by Elsberg, of whose cases, 18 were males and 6 females.

Of 3071 cases of typhoid fever in children that we have collected from various sources, 1659 were males and 1412 were females, a proportion of 1.17 males to 1 female.

Of 7848 cases of typhoid fever at all ages gathered from the statistics of Murchison, Curschmann, and others, 4452 were males and 3396 were females, a proportion to 1.31 males to 1 female. Except for Elsberg's and our own figures, we have found none which give the proportion of male to female in regard to the occurrence of perforation in children. Combining our figures with Elsberg's, there are 36 males and 21 females, a proportion of 1.7 males to 1 female.

In adults the preponderance of males over females who have been operated on for perforation is admittedly large. Harte and Ashhurst give the proportions as about 4 to 1; Finney,[6] 3 to 1; and Scott,[7] men and boys, 77; women, 7 (11 to 1). Curschmann[8] considers that this disproportion in adults is not due to any inequality in the development of typhoid ulceration, but probably to the fact that in men convalescence is unfavorably influenced by previous derangements of digestion due to diet and mode of life; that they submit to treatment at a later stage of the disease than women; that they are more impatient and more careless during convalescence. In view of the almost equal proportion of sexes attacked by the disease, the disparity in the number of adult males to females who suffer from perforation might be satisfactorily explained on these grounds, as we find in children, in whom such etiological factors are absent, that the disparity in the sexes is greatly lessened.

Type of the Disease. In 31 cases of our series, the type of the disease previous to perforation was mentioned. Eight were mild, fifteen were moderate, and eight were very severe; of the mild cases, five recovered and three died; of the cases of moderate severity, eight recovered and seven died; of the very severe cases, two recovered and six died. The influence of the severity of the attack upon the prognosis in cases of perforation as shown by these figures seems to be considerable. The child who is toxic and exhausted by prolonged hyperpyrexia is in poor condition to resist the additional complication and operative intervention.

That children, as a rule, suffer from a milder type of the disease

[6] Johns Hopkins Hosp. Rep., vol. viii.

[7] New York Med. Jour., 1907, lxxxv; Univ. Penna. Med. Bull., May and June, 1905.

[8] Nothnagel's Encyclopedia, American edition.

than adults is generally admitted; and it is probable that this, in part, explains their lower mortality after perforation. To this rule, of course, there are well recognized exceptions. The age is an important factor. Children under three years of age and over ten years, show a much higher comparative mortality, in the former exceeding, and in the latter approaching, that found in adult life. The influence of the severity of the disease upon the occurrence of perforation, however, has not been universally agreed upon. In adults, the majority of recent writers admit that the severe cases are most prone to the accident of perforation. The most noteworthy exceptions exist in the ambulatory cases, in which the occurrence of perforation is comparatively frequent. Of 68 cases of perforation reported by Finney, 39 were ambulatory, 10 moderately severe, and 19 severe. Only 8 of the 31 cases of our own series, in which the type of the disease was mentioned, were severe.

Time of Perforation. In 44 cases the time of perforation was definitely stated. In the second week there were 11 perforations, the earliest on the ninth day; 6 occurred at the end of the second week (thirteenth or fourteenth day); in the third week there were 12; in the fourth week there were 6; in the fifth week, 3 (one relapse); in the sixth week, 4 (three relapses); in the seventh week, 3 (all relapses); in the eighth week, 1 (a relapse); and in the ninth week, 1 (a relapse). One occurred one month after convalescence. Among the cases that perforated in the second week there were 6 recoveries and 5 deaths; in those perforating in the third week there were 3 recoveries and 9 deaths; in the fourth week, 2 recoveries and 4 deaths; in the fifth week, 1 recovery and 2 deaths; in the sixth week, 3 recoveries and 1 death; in the seventh, 1 recovery and 2 deaths. The cases perforating in the eighth and ninth week and one month after convalescence all recovered. If these figures prove anything, it is that the severity of the disease, rather than its protracted course, contributes toward its mortality. The danger of perforation is greatest at the end of the second and during the third week—42.8 per cent. of all cases occurring during this period. This proportion corresponds rather closely with that of Elsberg (10 out of 24 in the third week). In adults also perforation occurs most frequently during the second and third weeks.

Relapses. Of our cases, 9 were in relapse at the time of perforation; in another, a probable relapse, the duration of the disease was not stated, thus making 10 cases in all. The date of the perforation ranged from the seventh to the fifteenth day of the relapse. Of these cases, 6 recovered and 4 died, a percentage of recoveries higher than that observed in unrelapsed cases. This, again, seems to confirm the conclusions mentioned above in regard to mortality being influenced by the severity rather than by the duration of the disease.

Harte and Ashhurst found a mortality in 15 cases of relapse of 46.6 per cent.; and in the convalescent cases, 36.6 per cent.—which is

much lower than at any stage except in the first week (33.3 per cent.). Their highest mortality was observed in cases perforating in the third week (78.1 per cent.).

Pain. The importance of pain as a symptom of perforation is shown by its occurrence in practically every case of our series of which details were given. In only eight cases, taken from tables, or discussions, in which scanty data were furnished, was it not mentioned; and in one of these the child was under the influence of morphine. It may be considered in its relation to the time of development, its severity, location, and persistence.

In 87.5 per cent. (21 out of 24 cases) it was an initial symptom; in one case it was mentioned as not being initial; in two other cases it had been present during the illness to such an extent as to constitute a difficulty in diagnosis. In 89 per cent. (25 out of 28) the pain was severe. In the remaining 11 per cent. it was stated to be not severe. In 58.6 per cent. (17 out of 29) the pain was general in distribution. In 24.1 per cent. (7 out of 29) it was confined to the right side of the abdomen, especially the right iliac region. In 10 per cent. (3 out of 29) it was noted in the left side. In one case it was noted in the "upper," and once in the "lower," abdomen. In 10 out of 17 cases the pain was persistent, in four of which it continued to increase in severity. In 3 out of 17 it was intermittent in type; and in 4 it was distinctly stated to be "not persistent." In practically all of Elsberg's cases pain was the initial abdominal symptom. It was usually localized to the lower abdomen, especially the right side, and was generally paroxysmal; in a few cases it was constant.

Pain is thus seen to be a very characteristic and important symptom. In the great majority of cases it is an initial symptom and of marked severity. It is more often generalized than local, although it favors the right side in a considerable percentage of cases, and it is usually persistent, often increasing as time advances.

In Scott's statistics, pain at the time of perforation was observed in 75 per cent. of the cases. Other authors state that it is usually present, but may be entirely absent, although practically all agree in placing it with tenderness and rigidity as one of the three cardinal symptoms.

Except in a phlegmatic, delirious, or semicomatose patient, whether child or adult, the occurrence of pain seems to be almost a *sine qua non*, either of the perforation itself or of the beginning of the localized peritonitis. In such cases it would precede the rigidity, and possibly even the tenderness; and it might even appear to be for a time, at least, an isolated symptom. It would seem to be of great importance to determine the frequency with which sharp pain, not connected with perforation, occurs during typhoid fever.

In the case of children, Adams[9] found it to be an "infrequent"

[9] Archiv. Pediat., February, 1904.

symptom throughout the course of the uncomplicated disease. Edwards[10] found it in 10 per cent. of his cases under ten years of age; and in 40 per cent. between ten and fifteen years. Butler[11] found it in 22.4 per cent. Hand and Gittings[12] record pain or tenderness in 37 per cent. Barthez and Sanne[13] "believe that pain in children is commonly seen in connection with diarrhœa, but is usually a transient symptom." Biedert and Fischl[14] also believe that pain and meteorism are neither frequent nor severe in children. In the well-known statistics of McCrea[15] pain was found in about one third of 500 adult cases at some time during the course of typhoid fever. It was most constantly present with perforation and was closely simulated in cases of hemorrhage, phlebitis, and in some cases of unknown origin. In two-fifths of all the cases of pain, no adequate cause for it was found, and in 14 per cent. the pain was due to some condition other than the intestinal lesions. Shattuck, Warren, and Cobb[16] found a record of pain in only 5 out of 70 cases without perforation or peritonitis.

While the figures of various observers both of cases in childhood and adult life vary from 7 per cent. to 37 per cent. it is, nevertheless, apparent that a real difficulty in interpreting the symptom of pain will often exist. That its occurrence can never be safely disregarded is equally obvious. The various conditions which, by giving rise to pain, may simulate perforation will be considered under the heading of diagnosis.

Tenderness. Tenderness was noted as being present in 36 out of 45 of our cases. Its absence in the others may depend largely on insufficient data. It was recorded as "general" in 15 cases; prominent in the right iliac fossa in 9; confined to the "lower abdomen" in 2; and once to the hypogastrium.

A study in regard to the time of its occurrence showed its presence both in the early and in the late stages. In only one case was it absent "early," and in this one it developed later. Elsberg found tenderness in every case studied, and in six cases it was most marked in the right iliac fossa. He emphasizes the interval of time which occurs between the development of pain and the appearance of tenderness. As has been said, tenderness forms one of the tripod of cardinal symptoms, upon which the majority of observers rest the diagnosis in adults, although its absence in rare cases has been noted by competent authorities.

Rigidity. Rigidity may be regarded as another important diagnostic symptom. It is almost invariably present. It was noted as being absent in only 1 of 32 cases of our series in which full data

[10] Archiv. Pediat., September, 1907.
[11] Jour. Amer. Med. Assoc., 1905, xlv.
[12] Archiv. Pediat., June, 1906.
[13] Rev. des malad. des enfants, 3d ed.
[14] Lehrbuch. der Kinderkrankheiten, 12th ed.
[15] New York Med. Jour., May 4, 1901.
[16] Boston Med. and Surg. Jour., June 21, 1900.

were given; and in one it was absent early but appeared later. Elsberg found it more or less marked in 14 cases. Its late appearance often depends upon general peritonitis; but when found early, it becomes, in children, as in adults, a most valuable aid to diagnosis.

Vomiting. In 26 cases, vomiting was present twenty times, and absent six times. Of the 20 cases, it occurred as an initial symptom in 4; and in 4 others it was noted within four hours of perforation. In a large number of cases in which it was not mentioned, it was probably absent; and in the positive cases, it must often be a symptom of peritonitis rather than of perforation per se. Elsberg found it present early in four cases, occurring more frequently as the case progressed, but sometimes absent even in the presence of advanced peritonitis. In adults, Finney found vomiting with perforation in 26 of 68 cases. It will be seen, therefore, that, while vomiting is frequent and a symptom of some value always to be inquired for, it cannot be considered as characteristic.

Distention. Distention was noted as being present in 19 cases and absent in 11. In 9 early cases (within six hours of perforation) it was present in 3 and absent in 6, while in 21 late cases (after six hours) it was present in 16 and absent in 5. In the other cases it was not recorded.

Distention was present in only 50 per cent. of the cases that recovered and in 91 per cent. of the cases that died. Some degree of distention was observed in 19 out of 22 cases in Elsberg's tables, although in only 10 was it of more than moderate degree. As one explanation of its early occurrence, it must be noted that distention is often present in an uncomplicated case of typhoid during the second or third week; while its frequency as a late symptom, taken in connection with its presence in so large a proportion of the fatal cases, simply indicates its significance as a symptom of peritonitis. Of 749 cases of typhoid fever in cildren collected by Morse, Adams, and Hand and Gittings, 209 (26.5 per cent.) showed the presence of distention during the course of the disease, apart from the complication of perforation. Curschmann believes that, while distention is more common in children than in adults, it is generally more moderate in degree in the former than in the latter. Distention, therefore, seems to be of scant value in the early diagnosis of perforation in either children or adults.

Liver Dulness. In sixteen cases the normal area of liver dulness was diminished or almost obliterated; 5 times this was noted in early cases. Elsberg noted it in only 5 cases, and emphasizes the fact that distention alone may cause a diminution in the normal area of dulness. Its value as a diagnostic sign is certainly greatly lessened by this fact. In adults Finney found obliteration of hepatic dulness in only 5 of 68 cases, and Shattuck, Warren, and

Cobb, in only 2 of 24 cases. Harte and Ashhurst consider it a most unreliable symptom. Diminution of liver dulness thus will rarely give material aid in the early diagnosis of perforation.

Effusion. Effusion, as indicated by movable dulness in one or both flanks, was noted as being present in eight of our cases. The failure to record it may be explained partly by the difficulty often experienced in determining the presence of small amounts of fluid, and partly by the impossibility of distinguishing between fluid within the bowel or free in the peritoneal cavity, without movement of the patient to his side, a procedure which is manifestly attended with risk of spreading the peritoneal infection. The absolute determination of a rapidly increasing movable dulness in the flanks which were previously tympanitic, is of undoubted value in corroborating a diagnosis, but will rarely be available for establishing it. Other abdominal symptoms not noted in our cases demand attention. Curschmann refers to the condition of paralytic ileus and consequent constipation, which is apt to develop soon after perforation. While this would seem to depend upon a rapidly spreading infection of the peritoneum, yet it may be noted some hours before the patient's case has reached a surgically hopeless condition. Taylor[17] considers that inhibition of peristalsis forms one of the most important symptoms of the perforative accident. In very few of the case records which we have consulted was the presence or absence of peristaltic sounds noted. More careful attention to this point might furnish valuable aid to diagnosis.

Temperature. In 12 cases information as to the temperature was entirely wanting. In 10 cases the temperature was given at varying times subsequent to perforation, but without statement as to its height before perforation. In 7 of these 10 cases it varied from 100° to 103.8° F. In the remaining 3 cases it was "subnormal." In 6 cases it was stated that there was no change in the temperature curve at the time of perforation. In 5 cases there was a distinct rise varying from 1° to 5°. In 11 cases there was an immediate drop; this averaged from 3° to 4° one of 7° and another of 10° being recorded. In one case there was a late drop of 6°. Analyzing these figures, we find that of 33 cases, there were 15 in which the temperature was depressed at some time subsequent to perforation, and that in 11 of these it was specified that there was an immediate drop. In 6 cases it was definitely stated that there was no change. In 5 cases it was stated that there was a rise at the time of, or shortly after, perforation; and in 7 cases the temperature given, presumably at the time the diagnosis was made, was fairly high. In these there may have been a slight drop, or one more pronounced with a subsequent rise. In only 33 per cent. of the cases was it specifically stated that there was a pronounced initial drop in temperature; so that, while this is

[17] New York Med. Jour., February 1, 1902.

suggestive of perforation, it is probably absent in more than half the cases occurring in childhood.

We are more inclined than Elsberg to lay stress on an initial drop as a feature of significance in diagnosis. He found it recorded in only 4 of his 25 cases. It must not be forgotten that a drop in temperature may be a symptom of intestinal hemorrhage with or without perforation. In 2 of these 4 cases of Elsberg both hemorrhage and perforation were associated with the fall of temperature.

When we compare the conditions found in adults, we find authorities differing as to the frequency of this initial drop. Finney found it recorded in 14 of 68 cases. Harte and Ashhurst consider it significant but not diagnostic, while Harte,[18] in another communication, considers that a fall in temperature associated with sweating is a fairly constant accompaniment of perforation. The necessity for recording the temperature promptly after the first symptoms is obvious, as the subsequent rise may occur so rapidly as to mask the initial fall.

Chill. The interesting observation of a chill at the time of perforation was recorded in 6 cases of our series. In view of the rarity of chills during an uncomplicated attack of typhoid in children, their occurrence during the second or third week should lead to careful examination of the patient for other symptoms of impending or existing perforation.

In adults, Scott found that in 16.9 per cent of his cases the occurrence of perforation was marked by a chill. Harte considers a chill infrequent, and Curschmann says that it occurs occasionally, especially in those cases with subsequent elevation of temperature.

Pulse. In 34 cases more or less satisfactory observations upon the pulse rate were furnished. In 8 there was no material change; in 8, there was an average rise of 39 beats per minute, the increase ranging from 12 to 70; in 9 cases the pulse was rapid when they came under observation, the rate ranging from 140 upward. Others were simply described as weak, rapid, or running. This condition was sometimes explained by an advanced peritonitis, but more often by initial shock. In 4 cases the rate varied only from 112 to 124; presumably in these cases the pulse showed no change. In 2 cases a slight slowing of the pulse was subsequently recorded, probably after an initial rise. Elsberg found decided change in the pulse in 15 cases; the rate increased and the quality diminished. In 5 cases this change was a sudden and early one. The majority of cases, therefore, in children, as in adults, show an increase in the rapidity of the pulse rate; and in a considerable number, a weakening in the quality of the pulse may be looked for as an accompaniment of intestinal perforation.

All writers seem to lay stress on the quickening of the pulse in

[18] Boston Med. and Surg. Jour., July 18, 1907.

adults after perforation. Harte and Ashhurst found a rise within fifteen and twenty minutes in the vast majority of cases. Briggs considers the change of pulse of the greatest importance, and Curschmann states that it may be the first indication of the grave significance of abdominal pain of sudden onset.

Shock and Facies. Does shock occur as an early symptom of perforation in children? Elsberg believes that they "seldom show the sudden symptoms of collapse that are so frequent in adults." The results we have obtained in our studies are not in accordance with this view. Shock was distinctly stated to be a marked symptom in 10 out of 45 cases, the most prominent alteration being in pulse and facies, and sometimes temperature—a rapid increase in the first; a pinched, pale, anxious expression in the second; and a fall in temperature, being the concomitant features. In three cases in which there was no shock the facies showed no change. In this connection we may mention the facies which is found in cases of advanced peritonitis, the well known hippocratic type. In 12 out of 14 cases of perforation observed late (after six hours) this appearance was noted. Curschmann considers that the change in facial expression corresponds approximately with the local manifestations.

Taken in connection with the change in pulse and temperature, change in facies assumes some importance; but alone, it is doubtful whether it could be satisfactorily interpreted.

Respiration. The study of the data on respiration was unsatisfactory. Change in the rate is probably not of very great moment. In five cases, some seen early and some seen late after perforation, absence of abdominal respiration was noted—a symptom which is not often looked for, or, at least, recorded, but one which we think is probably of some importance. In two cases it was stated that abdominal respiration was present. In only 7 of the 45 cases, therefore, are any records available. We would emphasize the necessity of more careful observation on this point.

Leukocytosis. Leukocytic counts were recorded in only 14 cases in our series; in 6 of these repeated observations were made. In 2 of these 6 cases an increase of from 2000 to 3000 leukocytes was observed after perforation, both cases ending fatally. In two others which recovered a rise, respectively, of 2000 and 7000 was found. In the remaining 2, both of which were fatal, a falling count was recorded, which is in accordance with what has been observed by other writers, namely, that the outpouring of leukocytes in the effusion of peritonitis seems to act, at times, as a depleting agent to the circulation. Of the single counts taken within a few hours of perforation, 3 were under 8000. Of these cases, 2 recovered and 1 died. Of the cases showing counts of 10,900 and 12,440 respectively, one recovered and one died; while of the three which showed a count in excess of 18,000, all recovered.

The number of these cases is too small to permit of conclusions being drawn, but they suggest that a falling count is unfavorable, while a high count or a rapidly rising count is of better prognostic significance. Elsberg found a sudden or gradual rise in 5 cases, and believes that frequent counts in the early stages may be of confirmatory value, but even here may lead the surgeon astray. We may conclude, therefore, that leukocytosis often accompanies the accident of perforation in children; and without being an infallible sign, yet it forms one of the features in the symptom complex. The attempt to establish a normal average of leukocytes throughout an uncomplicated attack of typhoid in childhood is difficult. Hand and Gittings found counts in 71 cases, which ranged from 5000 to 10,000; while 31 cases gave counts varying from 10,000 to 16,000. All of these occurred in uncomplicated cases.

Many authors, in writing of typhoid fever in the adult, are inclined to attach little importance to the leukocytic count in relation to perforation, owing largely to the wide range found in uncomplicated cases; others consider that the subject needs further study before any conclusion can be drawn. Shattuck, Warren and Cobb, Cushing,[19] Thayer,[20] and others attach much importance to a rise shown by hourly estimations.

Blood Pressure. The question of blood pressure at the time of perforation has recently received attention, but we have found no records of estimations in children. In adults results so far recorded show a distinct rise within two hours of perforation. After this time the pressure falls.

Duration of Perforation Before Operation. There were 10 cases which were operated upon within six hours of perforation, of which 7 recovered and 3 died; of 10 cases operated on between six and twelve hours, 5 recovered and 5 died; of 8 cases operated on between twelve and twenty-four hours, 3 recovered and 5 died. Between twenty-four and forty-eight hours there were 8 cases, of which 3 recovered and 5 died. After forty-eight hours, there were 2 recoveries and 1 death.

There is here evident a steadily rising mortality after six hours. The recoveries in 2 of the late cases can be explained only on the theory of personal resistance to infection, lessened virulence of the intestinal bacteria, or more probably to partial localization of the infecting material. The same observation is made in late cases by Harte and Ashhurst.

Elsberg's figures show 100 per cent. of operative recoveries in the first eight hours, and 86.6 per cent. of recoveries in the first sixteen hours. After this time only 44.4 per cent. of recoveries are noted. All statistics confirm the value of early operation. The third hour was the most favorable period according to Harte and Ashhurst's tables; and after this a rising mortality was observed.

[19] Johns Hopkins Hospital Report, viii. [20] Ibid.

Description of Perforation. *Number.* In 3 of our fatal cases there was a double perforation at the time of operation, in one of which one of the perforations was overlooked and discovered at autopsy. In a fourth case there were two perforations in the ileum and two in the appendix. This case recovered. In 8.9 per cent. of the cases, therefore, the perforations were multiple at the time of operation. Comparing with adults, we find that 12.5 per cent. of Harte and Ashhurst's cases showed multiple perforations.

Situation. In our cases of single perforations the site in 20 cases was within twelve inches, and in 5 cases within two feet, of the ileocecal valve. In 2 cases the notes record it "within a short distance," and in one case in the appendix; 81.5 per cent. of the cases, therefore, were in the ileum within a foot of its termination. In adults, Harte and Ashhurst found that in 73 per cent. the perforation occurred within twelve inches of the ileocecal valve.

Size. Nineteen of the perforations in our cases were of small size (pin point, one-sixteenth to one-tenth inches), and 10 were large.

Secondary Perforation. Three cases of secondary perforations were recorded in our series, all of which were fatal; of these, one was in the jejunum. This gives a percentage of 6.6 per cent. of secondary perforations. In Elsberg's series there were four instances of multiple perforation, and of these probably two, and possibly three, were secondary. This is interesting when it is noted that in Harte and Ashhurst's 362 cases there were only 12, or 3.3 per cent., of secondary perforations.

Complications. There were three cases of secondary perforation after operation. One had hemorrhages on the second and fifth days after operation; a second case had pleurisy, bronchopneumonia, and a fecal fistula. Another case, already mentioned, died of an undiscovered perforation, which was present at the time of operation. There were four other cases of fecal fistula developing after operation, all of which recovered, as did 2 cases of Elsberg's. These are of interest in connection with the observations of Hays[21] and others who have commented on the favorable course of cases developing fecal fistula after operation.

In the first 10 recoveries in Hays' practice there were 5 cases of fecal fistula, and in 16 similar cases of Harte and Ashhurst's series the mortality was only 12.5 per cent. There were two fatal cases of suppurating parotid bubo, one of which was bilateral, and three cases in which secondary abscess in the abdominal cavity developed, two of which were fatal. The latter were probably due to deficient drainage or unavoidable pocketing of infected collections of fluid. Two cases of postoperative intestinal hemorrhage were recorded, one of which recovered. In another case (authors') there was a hernia of several loops of bowel from the wound, following the removal

[21] Jour. Amer. Med. Assoc., 1905, 1267; Penna. Med. Jour., January, 1908.

of loose gauze drainage five days after operation. Etherization was required for its reduction. We know of another case in an adult in which this accident occurred; caution should be exercised in the removal of gauze, for this reason. There does not seem to be a tendency for the formation of extensive adhesions in these cases when we consider that only 3 deaths out of 89 in Harte and Ashhurst's series were due to subsequent intestinal obstruction; no example of this sequel occurred in our own series and only one in Elsberg's.

Analysis of Deaths. In our series there were 23 recoveries and 22 deaths, a mortality of 48.8 per cent. In Elsberg's 25 cases there were 16 recoveries and 9 deaths, a mortality of 36 per cent. Combining these figures, we find a total mortality in children of 44.28 per cent. The average mortality in adults as given by various authors equals about 75 per cent. It remains to consider fatal cases with respect to the time at which death followed operation. There were no deaths on the table. Eight patients (38 per cent.) died within twenty-four hours after the operation, as contrasted with 36 per cent. for this same period in Harte and Ashhurst's series. Two patients died on the second day and three on the third day. This gives 64.7 per cent. of the total mortality for the first three days, as compared with 76 per cent. for the same period in the Harte and Ashhurst tables. One died on the fourth, one on the fifth, one on the seventh, and one on the eighth day; and four patients lived respectively twelve, fifteen, twenty-three, and thirty-one days after operation. In these late cases death was due to secondary perforation, pneumonia, parotid bubo, secondary abscess, etc., some cases showing multiple complications. Of the three cases of secondary perforation, death occurred on the fourth, eighth, and twelfth days, respectively.

Diagnosis. The important diagnostic symptoms which are shown by a study of our cases are: pain, tenderness, and rigidity, which in children, as in adults, are early in their development and fairly constant in appearance. Next in importance, because less frequent, but of significance when present, are a drop in the temperature, a rise in the pulse rate, and some degree of shock, often preceded or accompanied by vomiting, with perhaps a preceding or accompanying chill. A wave of leukocytosis will sometimes be recorded, and is at least as significant, if not more so, than in adults. Distention, and obliteration or diminution of liver dulness are of little value in making the diagnosis in children, while effusion is mainly of value in confirming it. Other points are an absence of abdominal respiration, inhibition of peristalsis, and possibly a rise of blood pressure, hitherto unstudied in early life. The accident of perforation may be simulated in children, as in adults, by a number of other conditions.

Appendicitis. We have operated for acute perforative appendicitis in typhoid fever in a child under the diagnosis of perforation. The

diagnosis between the two may usually be made from the fact that in appendicitis in typhoid fever, while the local symptoms are the same, their development is not usually marked by the same degree of constitutional involvement in the way of shock, fall of temperature, and rapid rise of pulse-rate as in perforation; nor is the course toward a fatal issue so rapid, if operation is not done. We have seen a case, in the service of a colleague (Hodge), of peritonitis without perforation, in a child who recovered from the operation. Such a case would be difficult or impossible of diagnosis from perforation, and operation would be equally indicated.

Hemorrhage. This may simulate perforation, or may precede or follow it. When pain is not present, it will not be difficult to distinguish. When pain and collapse are present before the appearance of blood in the stools, local and general symptoms must be carefully studied. The occurrence of previous hemorrhage would put the physician on his guard. The absence of rigidity and marked tenderness, and of leukocytosis, vomiting, and chill, are points in favor of hemorrhage.

Retention of Urine. While we know of no cases in childhood in which a distended bladder has caused pain suggestive of perforation, the occurrence of this condition is as easily conceivable, or more so, than in the adult, in whom it has to be reckoned with as a source of possible error in diagnosis.

Pneumonia and Pleurisy. A right-sided pleurisy or pneumonia very frequently simulates an inflammatory process in the right iliac fossa in childhood; examination of the base of the chest posteriorly should invariably be made when pain and rigidity are observed in the right side of the abdomen.

Flatus and Intestinal Colic. The local and constitutional symptoms of an inflammatory process will usually be wanting.

Rarer affections, perhaps simulating perforation, such as suppuration of mesenteric glands, intestinal obstruction of mechanical origin, intussusception, venous thrombosis, etc., need only be mentioned in this connection. Finally, it may be assumed that the accident of perforation is quite as likely to be overlooked in children as in a ults. The difficulty in eliciting the symptoms in young children, both objective and subjective, is the greatest stumbling block in the making of a diagnosis.

The Technique of Operation. It remains to analyze our statistics with regard to the technique employed: In the large majority of the cases the usual right iliac incision was employed. There is little difference in opinion as to its merits and the preference to be shown it. In three cases, probably on account of the condition of the patient, local anesthesia was employed. All of these cases ended fatally. While local anesthetics have undoubted advantages at times in adults, and are used practically as a routine by some operators (Hays), their employment in the young is open to

serious objection on account of the excitability of children and the difficulty in controlling them. Elsberg prefers chloroform anesthesia in children, but our own preference is for ether. In two cases the appendix was removed for perforation. There are no special data in our tables as to the type of sutures used to close the perforation. A double row of interrupted Lembert sutures, applied in a direction longitudinal to the bowel, is usually sufficient. A purse-string suture, for a small perforation, reinforced, if necessary, and omental grafting, to reinforce the sutures, for larger perforations may be used. As Elsberg points out, primary resection of the bowel in cases with a very large ulcer should never be done, if it is possible to avoid it. Indeed, we question whether it should ever be considered in children. When a perforation is too large to suture without the danger of occluding the lumen of the bowel, it will be far better to establish an artificial anus at once by primary enterostomy.

Primary Enterostomy. The favorable influence of the development of a fecal fistula upon the prognosis, and the arguments based thereupon in favor of primary enterostomy instead of suture of the perforation at the time of operation, have been touched upon in the consideration of complications. Hays recommends enterostomy as a routine measure. Two cases in our series in which an artificial anus was established died. Without doubt, enterostomy is an operation which is growing in favor, both for obstruction and for peritonitis.

The question of its routine employment may still be considered an open one. The near future will determine whether its disadvantages are offset by a decrease in mortality when it is adopted. We are heartily in favor of anchoring the sutured segment of the bowel in the wound, which was done in two of our cases with favorable issue.

Treatment of Effusion. The fluid was removed by irrigation in at least 19 cases in our series, with 11 recoveries; and by wiping or sponging in 6 cases, with 4 recoveries Surgeons are still divided as to the relative merits of irrigation and non-irrigation in the treatment of abdominal effusions of infectious material. Most of the arguments commonly used against it are valid in the consideration of infections of a different type, especially those of appendicular origin, when the infection is a slowly spreading one from a limited focus, and when a large area of the peritoneum is not involved. In perforations of the bowel of other types, and in gastric perforation, especially from ulcer, when the peritoneum is often flooded with intestinal or gastric contents and inflammatory exudate, we believe irrigation furnishes the quickest and most effective means of removing this infectious material. Evisceration should never be employed in children, whose susceptibility to shock is well known.

Rapidity of operation is even more essential in children than in adults, on account of the danger of fatal shock. Our experience in operations upon children leads us to indorse the opinion of Elsberg

that children bear abdominal and other operations well, if manipulations are not prolonged. Moreover, their recuperating powers after operation are greater than adults, and the reaction is more rapid and more complete.

Drainage. At least three cases were closed without drainage of the abdomen; and of these, two recovered. Tube or tube and gauze were used in the majority of cases, in which the character of the drainage was described. Since the widespread adoption of the principles of the Murphy treatment of diffuse peritonitis, we hear less about leaving infectious collections to the tender mercy of the peritoneum. A tube in the pelvis for existing or potential collections can do no harm, and a gauze strip to the region of the sutured ulcer, unless the bowel be anchored in the wound, is a wise precautionary measure.

Position After Operation. The Fowler position, either alone or in combination with continuous enteroclysis (Murphy), was occasionally mentioned as being practised, and was probably more frequently used. Its advantages are manifest. Nevertheless, the warning sounded by J. Alison Scott as to the wisdom of always employing it is to be borne in mind. He believes it to be occasionally harmful in typhoid cases, and in exhausted or delirious children it may well be found at times inexpedient. Unless contra-indicated by these conditions, however, we would advise it, as we have used it and seen it used to advantage.

In children, we cannot withhold either water or nourishment from the stomach as long as we can in adults; nor is it necessary, in the majority of instances, to do so. If the continuous enteroclysis is employed, the patient will suffer less from thirst. After six or eight hours, if there is no vomiting, we see no objection to administering small amounts of water by the mouth; and after twenty-four hours we may begin the administration of albumin water and pasteurized milk by the mouth.

Conclusions. Typhoid perforation is very rare under five years of age; after this period it is not infrequent, being about half as common as in adults. The favorite time of perforation is at the end of the second and during the third week. The diagnostic symptoms, in the order of their importance, are pain, tenderness, rigidity, fall in temperature, rise in pulse rate and collapse, vomiting, chill, and rising leukocytosis. The mortality after operation is influenced by the severity of the disease, rather than by the protracted course. It is lower under ten years of age than after this time. The mortality is lower in relapsed than in unrelapsed cases. The average mortality is somewhat less than 50 per cent. and at least 25 per cent. lower than in adults. The earlier the operation is performed, the better the prognosis. The technique of the operation does not differ materially from that advisable in adults, except in the use of a general anesthetic and the even greater necessity for rapidity in operation and avoidance of meddlesome surgery.

THE GENERAL MOVEMENT OF TYPHOID FEVER AND TUBERCULOSIS IN THE LAST THIRTY YEARS.

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Professor Finkelnburg, of Bonn, estimates that the average length of human life in the sixteenth century was only between eighteen and twenty years, and at the close of the eighteenth century it was a little over thirty years, while today it varies in different countries from less than twenty-five to more than fifty years. The span of life since 1880 has been lengthened in civilized countries about six years. No two factors have contributed so much to the general results as the improvements of the air we breath and the water we drink. Indeed, we have ample evidence that with the introduction of public water supplies and sewers the general mortality in numerous cities during the past fifty years has been reduced fully one-half, the good effects being especially shown by a marked decrease in the number of deaths from typhoid fever, diarrhœal diseases, and consumption. The vital statistics of Great Britain furnish the proof,[1] and our experience with American cities confirms this conclusion.

The death rate in the city of Berlin has been reduced from 32.9 in 1875 to 16.4 in 1904; in Munich, from 41.6 in 1871 to 18 in 1906; and in Washington, from 28.08 in 1875 to 19.25 in 1907. The death rate in the city of New York in 1804 was 28 per 1000; from 1850 to 1854 it was 38 per 1000; while in 1906, in spite of the density of

[1] The following table shows the death rate from certain diseases per 10,000 of population in the English cities before and after the introduction of sanitary works (see Cameron, A Manual of Hygiene, 1874, p. 129):

	Typhoid fever	Diarrhœa.	Consumption.
Bristol, before sanitary works	10.0	10.5	31.0
Bristol, after sanitary works	6.5	8.1	25.5
Leicester, before sanitary works	14.7	16.0	43.3
Leicester, after sanitary works	7.7	19.3	29.3
Cardiff, before sanitary works	17.5	17.2	34.7
Cardiff, after sanitary works	10.5	4.5	28.6
Macclesfield, before sanitary works	14.2	11.0	51.5
Macclesfield, after sanitary works	8.5	9.0	35.3
Warwick, before sanitary works	19.0	5.7	40.0
Warwick, after sanitary works	9.0	8.0	32.3
Stratford, before sanitary works	12.5	11.2	26.6
Stratford, after sanitary works	4.0	5.7	26.5
Ashby, before sanitary works	13.3	4.0	25.5
Ashby, after sanitary works	5.7	8.3	31.3
Dover, before sanitary works	14.0	9.5	26.5
Dover, after sanitary works	9.0	7.0	21.2
Croydon, before sanitary works	15.0	10.0	. . .
Croydon, after sanitary works	5.5	7.0	. .

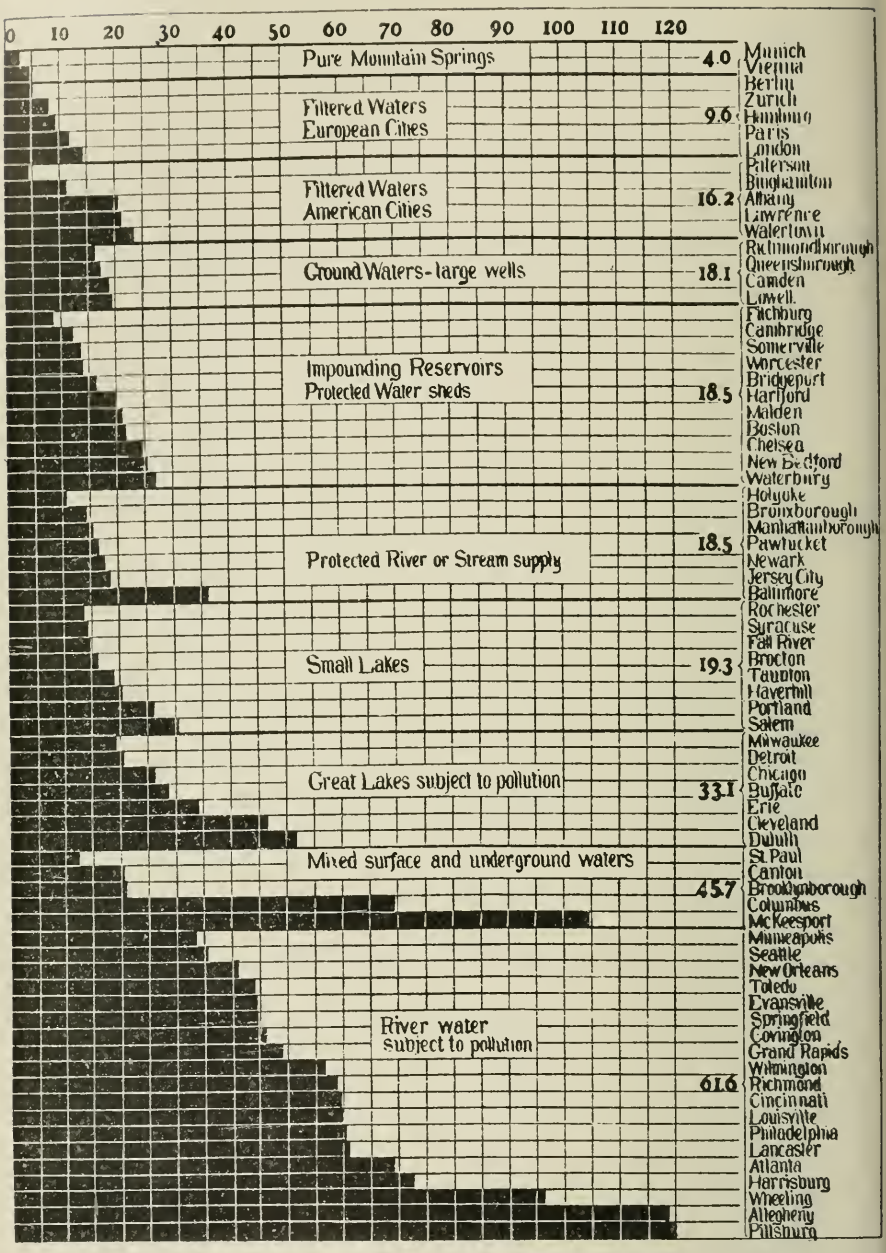
the population, it was 18.9 per 1000, practically a reduction of 50 per cent., which, according to Walter F. Wilcox,[2] of Cornell University, means a saving of something like 46,000 lives each year in that city alone.

The mortality in the registration area in the United States has been reduced since 1890 from 19.6 to 16.2 per 1000 in 1905. Taking the census figures of a population of 33,757,811 in the registration area as a basis, the number of deaths in 1905 was 544,533; whereas at a rate prevalent in 1890, they would have been 662,654, a reduction of 17.8 per cent. and a saving in one year of 118,121 lives. If the same rate is applied to the entire estimated population in the United States of 82,574,195, the saving of human lives during 1905 alone would be over 290,000.

It has long since been known that rivers are always purer near their source; the amount of impurities increases as we descend the stream, since the water courses are the natural drainage channels of the country and the wastes of human life and occupations find their way into the streams. It is also well known that our large American rivers are the sewers and at the same time the source of water supply for nearly all the cities located on their banks. These cities show, moreover, a marked prevalence of typhoid fever, thus confirming what has been observed over and over again, that this disease, as also cholera, dysentery, and diarrhœal diseases can be carried from one town or city to another by means of inland waterways. Indeed, the question is one of extreme interest even to the residents along the Great Lakes; we know that large cities like Buffalo, Erie, Cleveland, Detroit, and Milwaukee discharge their sewage into the lakes, and we also know how Chicago and Cleveland suffered from typhoid fever visitations by contaminating their own water supplies. It is also a well-known fact that many of the river cities were obliged to resort to purification of their water supplies in order to arrest the ever-increasing typhoid fever wave.

Influence of Water Supplies Upon Typhoid Fever Death Rates. For the purpose of determining the influence of public water supplies on the typhoid fever death rates in general, Mr. M. O. Leighton, Chief of the Water Resources Branch of the U. S. Geological Survey, very courteously complied with my request for a list of the principle American cities with a population of over 30,000 classified according to their water supply. Dr. Cressy L. Wilbur, Chief Statistician of Vital Statistics, Bureau of the Census, with equal promptness and accuracy, has computed the mean rate (not the average annual rate, which, however, differs only slightly for the five years 1902 to 1906), and has arranged them in a diagram (Chart I). The statistics, in spite of the many factors concerned in the dissemination of typhoid fever, conclusively show that the water

[2] Monthly Bulletin, New York State Dept. of Health, March, 1908.



MEAN DEATH RATES FROM TYPHOID FEVER, 1902 TO 1906, IN 66 AMERICAN CITIES AND 7 FOREIGN CITIES. GROUPED, AFTER FUERTES, ACCORDING TO THE QUALITY OF THEIR DRINKING WATER. THE RATES FOR FOREIGN CITIES ARE TAKEN FROM JAMES H. FUERTES.

CHART I.

supply plays the most important role in the spread of the disease. A summary of the typhoid fever death rates is here given:

	Mean typhoid fever death rate from 1902 to 1906 per 100,000 of population.
4 cities using ground water from large wells	18.5
18 cities using impounded and conserved rivers or streams	18.5
8 cities using water from small lakes	19.3
7 cities using water from the Great Lakes	32.8
5 cities using both surface and underground water	45.7
19 cities using poluted river water	61.1

The rates for cities using unpurified river water fluctuate from 33.1 at Minneapolis to 122.1 at Allegheny, and even 133.1 at Pittsburg.

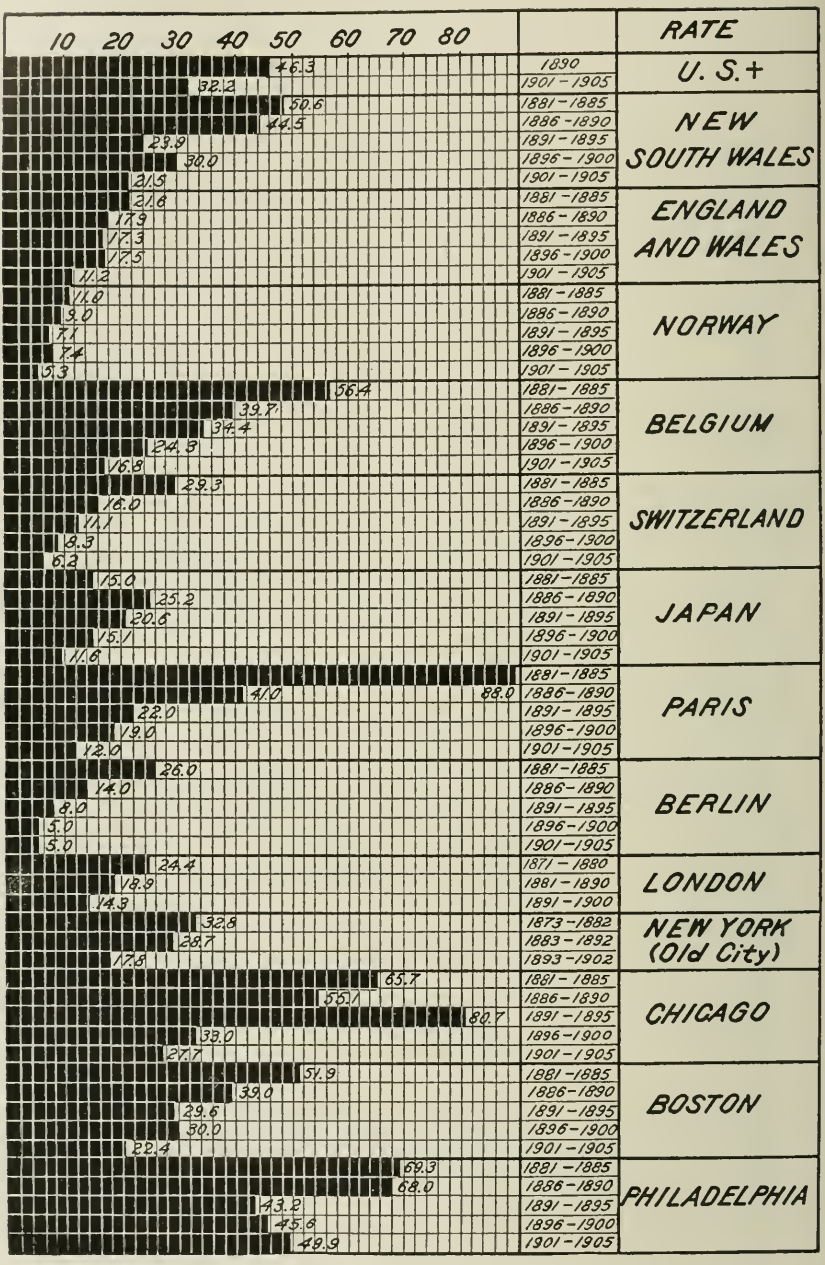
General Movement of Typhoid Fever. Chart II illustrates the general movement of typhoid fever in different countries and cities, showing the percentage of decrease from the first to the last period shown. The period covered by Dr. Wilbur is (nearly) the last quarter of a century, and the rates are usually given for the successive five-year periods, beginning with 1881. The table shows that during the last twenty-five years the death rate from typhoid fever has fallen in these fourteen countries and cities from an average of 42.3 to 18.1 per 100,000, a reduction of 54.3 per cent. A more striking reduction could have been made if statistics going back as far as 1870 had been included. The typhoid rate in Berlin in 1872, at a time when that city was riddled with cesspools and supplied with polluted water, was as high as 142 per 100,000. On account of the incomplete mortality returns everywhere prior to 1881, we have deemed it best to exclude all older foreign statistics, and for similar reasons Dr. Wilbur begins his statistics for the United States with 1890.[3] We have likewise excluded Mr. Whipple's statistics, which tend to show that the death rate from typhoid fever in twelve States, including all of the New England States, New York, New Jersey, Maryland, California, Minnesota, and Michigan, has fallen from 55 in 1880 to 21 per 100,000 in 1905.

The Hygienic Value of Pure Water: Annual Cost of Typhoid Fever in the United States. According to the Census of 1900, there were 35,379 deaths from typhoid fever during the

[3] Dr. Wilbur obtained the foreign statistics from data compiled from the international figures given in the last report of the Registrar General of England and Wales, from which report the rates for London are also taken. The rates for the cities of Paris and Berlin are given in the Annuaire Statistique of the City of Paris for the year 1904, and the rate of Berlin for the period 1901 to 1905 was supplied by Dr. Wilbur from data in his office. Dr. Wilbur laments the fact that "it is even now difficult to obtain a satisfactory statement of the number of deaths from such an important disease as typhoid fever in certain foreign countries, and the difficulty of securing comparative data increases as we go back. The disease was first accurately compiled by the Registrar of England in 1869."

TYPHOID FEVER

DEATH RATE PER 100,000 OF POPULATION



+Registration area

CHART II.

census year throughout the United States; and based on an estimated mortality of 10 per cent., it is within reason to assume a yearly prevalence of 353,790 cases of this disease. If we calculate the average cost for care, treatment, and loss of work to be $300, and the average value of a human life at $5,000, we have a total loss in the United States of $283,032,000 from one of the so-called preventable diseases. Mr. George C. Whipple[4] presents some striking evidence to indicate that a loss of $10,000 for every death from typhoid fever is a conservative estimate, in which case the decrease in the "vital assets" during the census year of 1900 would amount to $353,790,000. Reduce the prevalence of the disease one-half (which has been accomplished in Europe and our own country), and the question of the hygienic value of pure water will be answered from an economic point of view.

The Effect of Improved Water Supply on Typhoid Fever Death Rates. Chart III shows clearly the effect of change in water supply on typhoid fever death rates in seven American cities. Dr. Wilbur, of the Bureau of the Census, has given the death rate for a considerable time before and after the date of change, and also the average annual death rate before and after purification, and the percentage of reduction. From this table we learn that the combined average annual death rate from typhoid fever in cities with a contaminated supply was 69.4, and after the substitution of a pure supply it fell to 19.8 per 100,000, a reduction of 70.5 per cent.

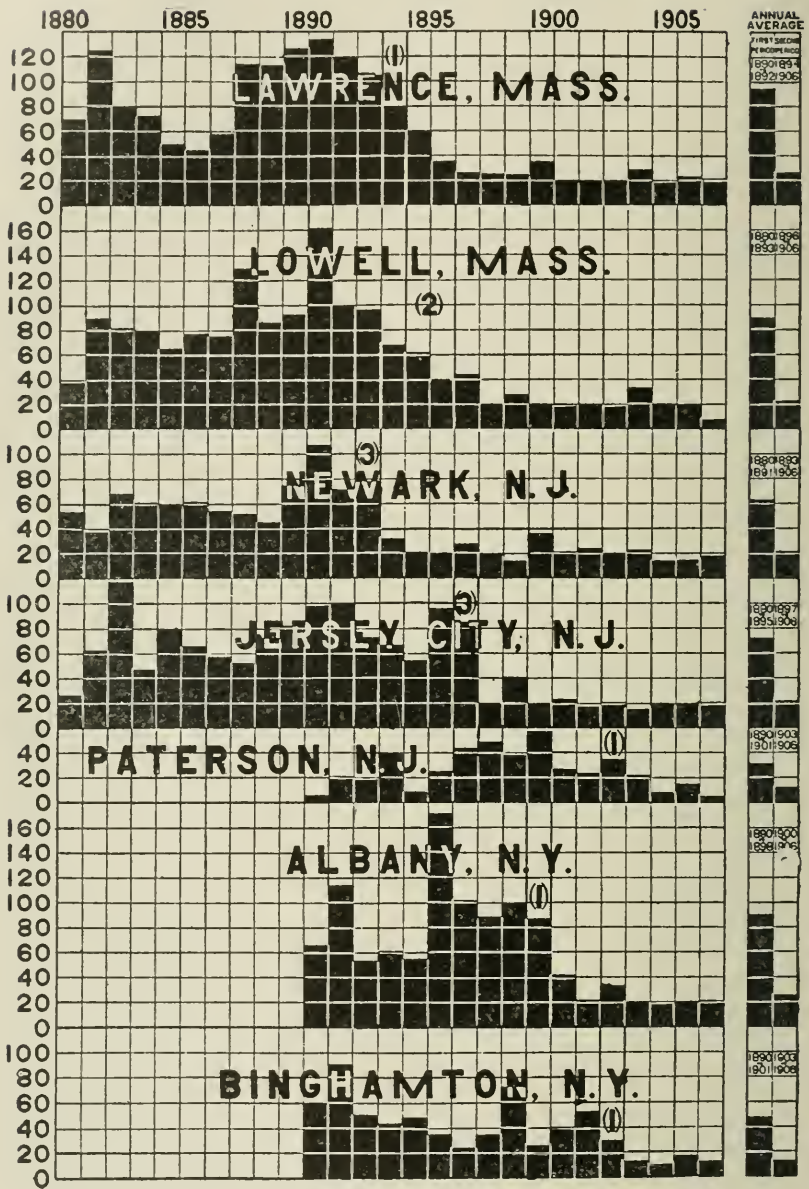
The *Bulletin* for the month of April, 1908, of the New York State Department of Health contains an interesting article showing that the death rate from typhoid fever in ten cities of that State has been reduced 53.4 per cent. by improved water supplies.

It may be urged that improved methods of medical treatment are responsible for a considerable reduction in the death rates from typhoid fever, but when we see such a striking change immediately after the installation of filtration plants as in the case of the American cities shown in Chart III, and also more recently in Cincinnati and Philadelphia, we are forced to the conclusion that water purification plays the most important role by diminishing primarily the number of cases. It should be stated, however, that the effects are still more marked when combined with a good system of sewerage. The history of every sewered town shows a lessening of the typhoid death rate, and that the typhoid rate is always higher in sections of the same city supplied with makeshifts. In 1895 I pointed out that typhoid fever prevailed in Washington in 1 of 81 houses supplied with privies, and in only 1 in 149 of those connected with sewers, and offered as the only reasonable explanation that the sewers carry away the filth and germs that otherwise would contaminate the soil and ground water; but even if there were no wells,

[4] The Value of Pure Water, New York, 1905, p. 5.

TYPHOID FEVER

DEATH RATES PER 100,000 OF POPULATION



CHANGE IN WATER SUPPLY

(1) From unfiltered river supply to filtered river supply
(2) From unfiltered river supply to wells
(3) From polluted river supply to conserved river supply

CHART III.

these makeshifts are still a source of danger in so far as they favor the transmission of the infection by means of flies; nor can the possibility be ignored that the germs in leaky or overflowing boxes may reach the upper layer of soil and with pulverized dust gain access to the system.

I believe that about 80 per cent. of the cases of typhoid fever are water- and milk-borne, and about 20 per cent. may be spread through the agency of flies,. personal contact, the consumption of raw oysters and shell fish raised in sewage polluted waters, or the eating of strawberries, radishes, celery, lettuce, and other vegetables and fruits which have been contaminated with infected night soil.

Other Water-borne Diseases. What has been said of typhoid fever is equally true of other water-borne diseases, like cholera, dysentery, cholera morbus, diarrhœal diseases, and the transmission of intestinal parasites, because the germs or ova of these diseases are present in the intestinal tracts and presumably also in sewage contaminated water. Mr. Allen Hazen,[5] one of the most distinguished experts on water purification in America, has conclusively shown that as the result of filtration plants in five cities supplied previously with an impure water, there was not only a reduction of 81 per cent. in the deaths from typhoid fever, but also a marked reduction in the general death rate. His computations clearly indicate that where one death from typhoid fever has been avoided by the use of a better water, a certain number of deaths, probably two or three, from other causes have been avoided. The truth of Hazen's theorem has recently been confirmed by Professor Sedgwick. It is a difficult matter to explain how water is connected with the deaths other than those from water-borne diseases, yet when we consider that water enters into the composition of the human body to the extent of 60 per cent., we are in a position to appreciate the sanitary acumen of Aristotle when he wrote in his *Politica:* "The greatest influence on health is exerted by those things which we most freely and frequently require for our existence, and this is especially true of water and air."

The general importance of the subject is now fully appreciated, and the North American Conservation Conference, on February 23,

[5] See a paper read at the International Engineers' Congress at St. Louis in 1904. Mr. Hazen found in five cities where the water supply had been radically improved:

	Per 100,000
A reduction in total death rate with the introduction of a pure water supply	440
Normal reduction due to a general improved sanitary condition, computed from average of cities similarly situated, but with no radical change in water supply	137
Difference being decrease in death rate attributable to change in water supply	303
Of this, the reduction in deaths from typhoid fever was	71
Leaving deaths from other causes attributable to change in water supply	232

1909, in the declaration of principles adopted the following in reference to Public Health:

"Believing that the conservation movement tends strongly to develop national efficiency in the highest possible degree in our respective countries, we recognize that to accomplish such an object with success the maintenance and improvement of public health is a first essential.

"In all steps for the utilization of natural resources consideration of public health should always be kept in view.

"Facts which cannot be questioned demonstrate that immediate action is necessary to prevent further pollution, mainly by sewage, of the lakes, rivers, and streams throughout North America. Such pollution, aside from the enormous loss in fertilizing elements entailed thereby, is an immediate and continuous danger to public health, to the health of animals, and, when caused by certain chemical agents, to agriculture. Therefore, we recommend that preventive legislation be enacted."

Having studied the effects of pure water supplies upon mortality rates, let us next consider the influence of pure air, removal of dampness, and general sanitation upon the movement of tuberculosis.

The Influence of Sewers in the Prevalence of Tuberculosis. The records of the Health Office of the City of Washington show that during the last thirty years 14.5 per cent. of all the deaths occuring in the District of Columbia have been caused by pulmonary tuberculosis. The death rate, however, has gradually and constantly fallen from 446 per 100,000 in 1880 to 280 in 1907. The death rate from this disease in New York City has fallen in like manner from 433 to 271; in the United States at large, from 326 in 1880 to 183 in 1907 (Chart IV). These reductions began long before the combat of the disease was a subject for popular education. The question naturally arises, if these reductions have resulted independently of any attempt to control the source of infection, what are the chief factors concerned in bringing about this gratifying result? I know of no sanitary reforms which could have exerted a greater influence upon our general well-being than the introduction of sewers, improved water supplies, and the erection of sanitary homes. The marked reduction in the prevalence of consumption after the introduction of sewers observed in England over forty years ago, and also in the cities of Washington and New York and the country at large, may fairly be attributed to the prevention of air pollution and dampness. It is noteworthy that while the reduction in Washington, coincident with the introduction of sewers, amounts to 37.3, the reduction in Baltimore, an unsewered city, is only 24.7 per cent. It has been estimated by Erismann that a cesspool with 18 cm. contents is capable of polluting the atmosphere in the course of twenty-four hours with 18.79 cm. of impure gases, composed of carbonic acid, ammonia, sulphuretted and carburetted hydrogen,

and volatile fatty acids. In view of this fact, it requires no great stretch of the imagination to calculate the amount of air pollution which resulted from the 30,000 cesspools and other makeshifts prior to the introduction of the sewerage system in the city of Washington. The influence of impure air upon our physical well-being and the powers of resistance cannot be over-rated. As a matter of fact, individuals who contract tuberculosis in cities often arrest the disease by removal to the country, showing that an abundance of pure air is a very important factor in the treatment of the disease, simply because it promotes oxygenation of the blood, stimulates the appetite and nutrition, and thereby increases the general resisting power of the system. There can be no doubt as to the curative virtues of pure air, and hence we ought not to under-rate its preventive properties.

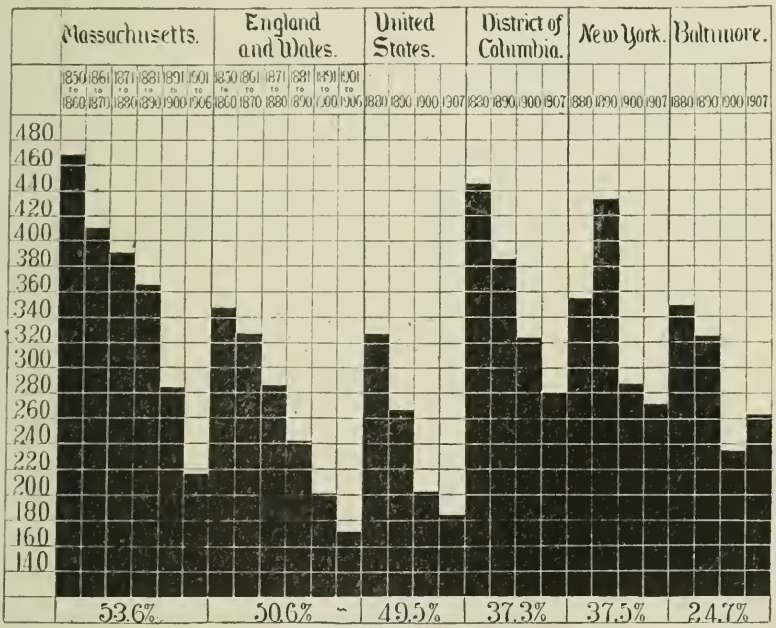


CHART IV.—General movement of tuberculosis. Death rates per 100,000 of population and percentage of reduction.

The observations of Dr. Bowditch, of Boston, as early as 1862, clearly indicate that there is a relation between dampness of soil and pulmonary consumption, and Dr. George Buchanan, the Medical Officer of the Privy Council of Great Britain, in 1867, supplied ample statistical proof that consumption became less frequent in certain towns after they had been sewered and the soil consequently drained. In towns like Worthing, Rugby, and Salisbury the deaths from consumption were reduced by 36 to 49 per

cent. It is true that such a marked reduction did not always follow, but in these instances it may be fairly assumed that the soil was previously quite dry and could not be materially affected by increased drainage.

The importance of a dry, healthful building site was appreciated by Hippocrates, since he, as well as Vitruvius, the father of architecture, referred in their writings to elevation as a desirable factor. One of the most striking illustrations of damp habitations as a predisposing cause to consumption has been recorded by Nowak, in the case of a prison in the vicinity of Vienna, containing on an average 200 inmates. Every convict is examined before his transport, and if found affected with incipient tuberculosis he is sent elsewhere. In spite of this precaution, the deaths number about fifty per annum, and the majority die from consumption. The prisoners are better fed in this institution than elsewhere, but the building rests on a wet soil, the walls are reeking with moisture, and the rooms smell musty.

The relation of dampness to consumption may be explained as follows: Dampness of soil, unless special precautions have been taken, extends by capillary attraction to the walls and renders the entire house damp. Damp air abstracts an undue amount of animal heat, lowers the power of resistance of the inmates, and predisposes to catarrhal affections, and these in turn render the mucous membrane, more vulnerable to the invasion of the tubercle bacilli. There is also reason for believing that the tubercle bacilli retain their vitality for a greater length of time in such an atmosphere on account of its humidity and excess of organic matter.

At all events, it has long been known that tuberculosis is far more prevalent in damp, dark, and insanitary houses. The children are anemic and as puny as plants raised without the stimulating effects of sunlight. The death rate is often double and treble that of other localities. While there are doubtless other factors which determine the frightful mortality, none are more potent than dampness, and deficient sunlight and ventilation. The tubercle bacillus clinging to floors and walls in carelessly expectorated sputum or droplets would be destroyed by a few hours' exposure to sunlight, but it finds in damp and dark basements, back-to-back houses, and yard and alley tenements suitable environments for its vitality and growth, and the other insanitary factors alluded to, together with the more intimate contact, materially increase the chance of infection.

If it should appear from the foregoing that I believe in the ubiquity of the tubercle bacillus, and that the question of environment should receive first and foremost consideration, I desire it to be understood that I am convinced, from the splendid labors of Professor Carl Fluegge, that the tubercle bacillus is not ubiquitous, and hence the task of stamping out the primary sources of infection is by no means hopeless. I believe, however, that until this is

accomplished, in this disease as in other infectious diseases, due attention should be given to all the causes likely to influence their spread, so that in our efforts to combat tuberculosis they may receive proper consideration.

I can scarcely do better than to conclude with the following quotation from the Report of the Conservation Commission:

"Since the greatest of our national assets is the health and vigor of the American people, our efficiency must depend on national vitality even more than the resources of the minerals, lands, forests, and waters. The average length of human life in different countries varies from less than twenty-five to more than fifty years. This span of life is increasing wherever sanitary science and preventive medicine are applied. It may be greatly extended. Our annual mortality from tuberculosis is about 150,000. Stopping three-fourths of the loss of life from this cause and from typhoid and other prevalent diseases would increase our average length of life fifteen years. There are constantly about 3,000,000 persons seriously ill in the United States, of whom 500,000 are consumptives. More than half this illness is preventable. If we count the value of each life lost at only $1700, and reckon the average earnings lost by illness at $700 a year for grown men, we find that the economic gain from mitigation of preventable disease in the United States would exceed $1,500,000,000 a year. This gain, or the lengthening and strengthening of life which it measures, can be had through medical investigation and practice, school and factory hygiene, restriction of labor by women and children, the education of the people in both public and private hygiene, and through improving the efficiency of our health service, municipal, State, and national."

EXPERIMENTS RELATING TO THE BACTERIAL CONTENT OF THE FECES, WITH SOME RESEARCHES ON THE VALUE OF CERTAIN INTESTINAL ANTISEPTICS.

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The great number of bacteria present in the intestinal tract has for many years been a source of great interest to the physicians, and many investigations have been undertaken to determine the number and nature of these organisms. It has been demonstrated that the great proportion of the organisms consists largely of but

a very few varieties. The question as to the specific importance of the flora in the intestines has also attracted considerable attention.

It was Pasteur who first expressed the view that the bacteria of the intestinal tract are essential to life. Nuttall and Theirfelder[1] fed pigs obtained by Cesarean section on sterile food. The animals lived and increased in weight. These investigators therefore concluded, that the intestinal microörganisms are not essential to the maintenance of life. Levin[2] has likewise shown that in the Arctic regions the intestinal tract of certain mammals is entirely free from germs. On the other hand, Schottelius[3] has demonstrated that in the chick the intestinal bacteria are essential to the normal nutrition, and when fed on sterile food the chicks were retarded in their growth and development. Madame Metchnikoff[4] obtained the same results in experiments on tad-poles and Moro[5] on the larva of the turtle.

If a certain number of bacteria are an essential factor in the intestine in order to maintain life, it must also be conceded that a large proportion of organisms present occasion fermentation and putrefaction. It is easily conceived how an antagonism between the cells of the intestines and the bacteria constantly exists, which ends in the destruction of a certain number of organisms, and keeps the bacterial growth within a certain limit. It is of interest to know something of the number of bacteria in the intestinal tract under normal conditions, so as to determine whether in certain diseases there is an increase or diminution. With the object of diminishing the number of intestinal bacteria, intestinal antiseptics have been utilized for some time. In order to determine their value scientifically, however, it is important to know something as to the number of bacteria infesting the intestinal tract in health. Two methods of solving this problem are feasible. The first consists in making plate cultures from a measured quantity of feces in various dilutions, and counting the number of colonies. Investigations of this character have been made by Sucksdenf,[6] Fürbringer,[7] Stern,[8] Gilbert et Dominic,[9] Salkowski,[10] Sehrwald,[11] Hammerl,[12] and Mieczkowski.[13] The difficulty arising from this method of investigation is due to the fact that even under normal conditions the number of bacteria

[1] Zeitschr. f. phys. Chem., 1895, xxi, 109; and 1897, xxii, 231.
[2] Ann. de l'institut Pasteur, 1901, p. 558.
[3] Archiv f. Hygiene, 1902, Band xlii, Heft 1.
[4] Annales d l'Institut Pasteur, 1901, xv, 631.
[5] Jahr. f. Kinderheilk , 1905, xii, 467.
[6] Archiv f. Hygiene, 1886, Band iv.
[7] Deut. med. Woch., 1887, Nos. 11 and 12.
[8] Zeit. f. Hygiene u. Infect.. Band xii, S. 88.
[9] Comptes rendus de la Société de Biologie de Paris, 1894, pp. 117 and 277.
[10] Virchow's Archiv, Band cxv, S. 339.
[11] Berl. klin. Woch., 1889, S. 413.
[12] Zeit. f. Biol., Band xxxv, S. 355.
[13] Mittheil. aus den Grenz. der Med. und Chir., 1902, Band ix, S. 405.

vary greatly, inasmuch as only a small proportion of those organisms sown in cultures develop at all, a large proportion dying.

Much more useful, therefore, is the method of counting not only the live, but also the dead organisms. This method was first introduced by Eberle[14] and further developed by Hellström.[15] The method was employed by Klein and de Lange,[16] who found 13 per cent. of the dried substance of the feces to be composed of bacteria. The value seemed so low that Strasburger[17] determined that the subject needed further investigation.

The method he employed is based on the principle that if the feces be macerated with water and then centrifugalized, the bacteria remain suspended in the fluid, while all the heavier substances fall to the bottom. If the fluid is now decanted, treated with alcohol so as to diminish its specific gravity, and again centrifugalized, the bacteria are thrown to the bottom and can be collected. Strasburger utilized this method in determining the number of bacteria in the feces, by weighing a certain quantity of feces, and afterward drying the bacteria obtained and weighing again.

From his investigations Strasburger arrived at the following conclusions:

1. The former methods of determining the number of bacteria in the feces are enveloped with great possibilities of error, and cannot give even relatively approximate results.

2. By means of this method it is possible to separate the bacteria from the feces, and weigh the same.

3. Under normal conditions one-third of the dried substance of the feces of a healthy individual ingesting a medium diet consists of bacteria.

4. The quantity of daily bacterial waste, dried, consists, in adults: (*a*) Under normal conditions, an average of 8 grams. (*b*) In dyspeptic intestinal conditions, an average of 14 grams, and even as much as 20 grams. (*c*) In chronic constipation, 5.5 grams, and at times even as little as 2.6 grams.

5. In chronic constipation the dried substance of the feces is usually abnormally small in quantity. The utilization of the food is far better, too, than under normal conditions. It is evident that the absence of the proper nutrient medium for the growth of the bacteria in the large intestine is the cause of this diminished growth.

6. In infants under normal conditions there is practically the same percentage of bacterial growth as in adults.

7. The total number of bacteria, estimated for a normal individual per day is 128,000,000,000.

8. Knowing the quantity of bacteria in the feces gives an insight into the bacterial development of the whole intestine.

[14] Centralbl. f. Bakt., 1896, i, S. 2.
[15] Ibid., 1896, S. 661.
[16] Ibid., 1900, Band xxvii, S. 834.
[17] Zeit. f. klin. Med., 1902, Band xlvi, s. 413.

This method affords us a means of determining the influence of intestinal antiseptics or the disinfecting activity of purgatives, and leads to the question, after all, whether it is possible to disinfect the intestine. In regard to the value of intestinal antiseptics, Strasburger concludes that the most effectual method of reducing the bacterial growth of the intestine is by means of diet. The value of intestinal antiseptics is quite doubtful. It cannot be doubted that under certain conditions there may be a diminution in the growth of the bacteria, especially in the small intestine by means of intestinal antiseptics; but the effect is slight, and is rarely pronounced. Other observers have come to conflicting views regarding the effect of intestinal antiseptics.

Bouchard[18] found that naphthalene, iodoform, and charcoal diminished the toxicity of the urine and stools, while Friedrich Müller[19] expressed the opinion that it is hardly possible to expect to disinfect the intestinal canal by drugs, and that there were no useful intestinal antiseptics. Stern[20] found that calomel diminished the bacterial content of the feces in some instances and not in others.

R. Schuetz[21] observed the action of various drugs by passing certain easily recognizable saprophytic bacteria through the bowel, and noting the number of organisms that passed through alive during the period of administration of various antiseptics. He found no special effect produced by the antiseptics. Mieczkowski[22] found that menthol and tannopin given by the mouth diminished the number of colonies in specimens of feces from a fistula at the lower end of the ileum; with itrol and bismuth this effect was not produced. Schönenborn[23] found that naphthalene, itrol, thymol increased the bacteria, and that salicylic acid diminished the bacteria of the feces. Hoffman[24] showed that isoform when taken by the mouth is an excellent intestinal disinfectant and decreases the number of bacteria in the feces. Herter[25] observes: "I have made some observations on the action of so-called antiseptics, and I have reached the conclusion that most of them do very little good in effecting a diminution of the putrefactive anaërobes of the intestines. I have found in certain instances that salicylates, aspirin, and salol have exerted some action in diminishing the output of indican, but beyond this I have not been able to satisfy myself that the effect of intestinal antiseptics is pronounced." The results of former investigations concerning the value of intestinal antiseptics differ to such an

[18] Leçons sur les autointoxicationes dans les maladies, Paris, 1887.

[19] Autointoxications intestinalen Ursprunges. Verhand. des Congress f. Innere Med., 1898, S. 148.

[20] Verhand. des Congresses f. Innere Med., 1898, S. 198.

[21] Archiv f. Verdauungskr., 1901, S. 58.

[22] Mittheilung aus den Grenzgeb. der Medicin und Chirugie, 1902, ix, 405.

[23] Inaug. Diss., Bern, 1903.

[24] Mittheil. aus den Grenz. der Med. und Chir., 1906, xv, 605.

[25] Bacterial Infections of the Digestive Tract, New York, 1907, p. 331.

extent that it appeared important to J. Dutton Steele[26] to investigate this subject more thoroughly than had been heretofore done. Steele points out the difficulties besetting this problem. Inasmuch as the intestinal antiseptics are seldom given alone, but usually combined with drainage of the stomach and intestine, and regulation of the diet, it is for this reason difficult to determine their true clinical value. Steele employed Strasburger's method of estimating the entire bacterial content in the stools in his researches, which he considers far in advance of all other methods. Bismuth salicylate and beta-naphthol, were the two antiseptics employed, and the subjects were free of any indications of intestinal disorders. His results were as follows: "The bacterial growth in the intestines is greatly diminished in normal individuals with the use of bismuth salicylate and beta-naphthol, and there is sufficient evidence to indicate that it is possible to check bacterial activity in the intestines by antiseptic drugs." Steele also expresses the opinion "that evacuation of the bowels combined with regulation of the diet are by far the most efficient means at our command to check excessive bacterial activity in the intestines."

The observations of Steele were not sufficiently extensive to draw very definite conclusions, so that it seemed most important to us to continue the investigations in the same direction. The technique employed in estimating the bacteria was that of Strasburger as modified by Steele. Steele describes this method as follows:

"The possibility of separating the bacteria from the rest of the feces depends on the fact that the bacteria are so nearly of the same specific gravity as distilled water that they cannot be centrifugalized out of a watery suspension of the feces, but remain suspended in the supernatant fluid. Taking advantage of this, the bacteria can be removed by washing with the centrifuge. Then, if the specific gravity of the wash-water is lowered by the addition of large amounts of alcohol, the relation of the bacteria to the fluid is changed to such an extent that the microörganisms can be readily centrifugalized out, separated, and weighed." "Unless the period of passage of the feces has been ascertained to be normal, it is better to mark the beginning and end of each period of examination by carmine. The use of the Schmidt diet is not necessary. The whole stool is saved. Unless the feces are liquid they are rubbed up with a known amount of distilled water until they are smooth and semiliquid and as homogeneous as it is possible to make them.

"Two portions of 5 c.c. are measured off with a pipette of large caliber, using for this purpose an ordinary 5 c.c. pipette with the tapering end cut off, and with the necessary correction made at the upper mark. One of these portions of 5 c.c. is put into a porcelain dish and dried over a water bath and later in a drying oven, in order

[26] Trans. of Assoc. Amer. Phys., 1907; and Jour. Amer. Med. Assoc., August 24, 1907.

to determine the dried weight. The addition of a little alcohol and thorough mixing will hasten the process of drying and prevent caking of the feces."

"The second portion is washed free from bacteria. This is done as follows: The wash-water is 0.5 of 1 per cent. HCl solution in distilled water. The acid increases the solubility of the salts and soaps of the feces. 100 c.c. of this solution is employed at the beginning of the washing. The feces are thoroughly mixed with the wash solution and then centrifugalized. The use of the water motor or electric centrifuge is almost essential. Each tube is centrifugalized for about one and one-half minutes, then the cloudy supernatant liquid is poured through a layer of gauze. This fluid contains the bacteria in suspension. All of the mixture (the wash-water and the feces) is centrifugalized the same way, and then the residue in the tubes is shaken up with more of the wash-water and centrifugalized again. This is repeated until the supernatant liquid after the centrifugalizing is transparent, showing that approximately all the bacteria have been washed out. If a smear is made of the residue at this point, it will be found that the bacteria are not entirely washed away, but are evidently very much reduced. They occur singly, while in the unwashed feces they are in great lumps and masses." "The suspension of bacteria is then mixed with a liberal portion of alcohol, and evaporated down slowly at a temperature of 40° to 50° C. until it amounts to not more than 50 c.c. in all. This takes approximately twenty-four hours. It is then mixed with at least twice its volume of alcohol, preferably absolute alcohol, although this is not absolutely necessary. This lowers the specific gravity of the fluid to such an extent that now the bacteria readily centrifugalize out." "The mixture is then centrifugalized until the supernatant liquid is quite clear. This takes thirty minutes or more for each tube. The residue, which consists of the bacteria, is washed with pure alcohol and is shaken up with ether to remove the fat; then it is again washed with alcohol. All of this washing is done by means of the centrifuge. The bacteria are next washed out of the tube with a little alcohol and evaporated to dryness and dried in the oven at moderate heat, dried in the desiccator, and weighed. Smears of the final preparation show that it consists of bacteria with a very few minute particles of other material. These particles are only visible with high power, and are very few in number, perhaps two to each field of the $\frac{1}{12}$-inch objective. They stain with methylene blue; Strasburger suggests that they are cellulose, which they may well be. At any rate, the error arising from the inclusion of these small particles in the dried weight of the bacteria must be very small, and is probably balanced by the bacteria that it is not possible to wash out of the residue in the first washing. During the preparation of the bacteria the first portion of 5 c.c. has been dried and weighed. We then know the dried weight of 5 c.c., the weight of the dried

bacteria in 5 c.c., the original volume of the stool, and the volume after the addition of a known amount of water. It is then easy to calculate the data that we desire, namely, the volume of the stool, its dried weight, the weight of the dried bacteria, and the percentage of bacteria in the dried weight."

In our experiments, extending over a considerable period of time, many examinations were made, both in normal individuals and patients suffering with digestive disturbances. Control experiments were made for several days without drugs, and the results compared with a period when the drugs had been taken for several days. The following drugs were employed: Beta-naphthol, bismuth salicylate, salol, aspirin, ichthalbin, lactobacilline, thiocol, bichloride of mercury, and thymol. In a certain number of cases the effect of diet alone was observed. The result of these examinations are summarized in the accompanying tables.

In order that the results of the various experiments may appear more clearly, the findings obtained by means of the same agent, in any particular individual, have been combined by us into a single average.

Normal Cases. These cases are represented in Table I.

The greatest reduction in the bacterial content was observed by means of diet alone. By the aid of a liquid diet, consisting mainly of fermented milk, this reduction averaged 16 per cent., and with a Schmidt diet, 13.7 per cent. Beta-naphthol reduced the organisms 9.9 per cent., whereas bismuth salicylate reduced them 8.8 per cent. Aspirin made an average reduction of 4.6 per cent., ichthalbin 4.2 per cent., while salol did not effect any reduction.

Individuals Affected with Gastro-intestinal Disturbances. In Table II are represented the cases, suffering with gastrointestinal disorders. The results are rather conflicting. In a case of intestinal catarrh, neither salicylate of bismuth nor thiocol reduced the bacteria to any degree, while ichthalbin reduced them only 7.2 per cent. In another instance, a case of chronic colitis, bichloride of mercury reduced the bacteria 7.1 per cent.; they were not influenced by the use of salol; and a slight increase was observed after the use of bismuth salicylate. In a third instance, a case of marked hyperacidity with intestinal catarrh, a very marked reduction was observed under a restricted semisolid diet, a reduction of 18 per cent., whereas no reduction was effected with salol, aspirin, or thymol, which clearly indicates that we cannot assume that the action of the so-called antiseptic drugs is the same in diseases of the gastro-intestinal tract as in normal individuals. From these observations we believe we are justified in concluding:

1. Regulations of diet, together with the evacuation of the bowels, is the most effectual method that we have at hand of reducing the excessively high bacterial content of the intestine.

TABLE I.—Normal Cases.

Name.	Diet.	Drug.	Number of examinations.	Average total feces.	Average dried weight.	Average dried weight (Bacteria).	Average per cent. (Bacteria).	Reduction.	Remarks.
				gms.	gms.	gms.			
J. D.	Regular	None	4	120	12.16	2.71	22.3		
J. D.	Regular	Beta-naphthol	3	200	36.40	4.51	12.4	9.9	
B. E.	Regular	None	4	150	12.10	1.11	19.2	...	Observation only after stool was black.
B. E.	Regular	Bismuth salicylate	3	175	23.18	2.41	10.4	8.8	
B. E.	Regular	Salol	3	91	10.08	1.95	19.4	None	
F. L.	Regular	None	3	138	13.30	3.21	24.2		
F. L.	Lozak	None	4	85	12.70	1.04	8.2	16.0	
F. L.	Schmidt	None	3	102	10.40	1.09	10.5	13.7	
F. L.	Regular	Lactobacilline	3	62	7.90	0.94	11.9	12.3	
G. H.	Regular	None	3	210	18.02	3.78	21.0		
G. H.	Regular	Ichthalbin	3	150	12.80	2.15	16.8	4.2	
R. S.	Regular	None	4	148	12.60	2.49	19.8		
R. S.	Regular	Aspirin	3	112	11.80	1.79	15.2	4.6	
R. S.	Regular	Salol	3	220	38.50	8.12	21.1	None	

TABLE II.—Individuals Affected with Gastro-intestinal Disturbances.

Name.	Diet.	Drug.	Number of examinations.	Average total feces.	Average dried weight.	Average dried weight (Bacteria).	Average per cent. (Bacteria).	Reduction.	Remarks.
				gms.	gms.	gms.			
D. R.	Regular	None	3	218.0	37.10	11.98	32.3	...	Case of intestinal catarrh.
D. R.	Regular	Bismuth salicylate	3	87.5	12.80	3.81	31.6	0.7	
D. R.	Regular	Thiocol	4	105.0	12.80	3.63	28.4	3.9	
D. R	Regular	Ichthalbin	3	114.0	17.40	4.36	25.1	7.2	
J. D.	Regular	None	3	76.0	10.50	2.89	27.6	...	Chronic colitis.
J. D.	Regular	Bicoloride mercury	4	120.0	15.70	3.71	20.5	7.1	
J. D.	Regular	Bismuth salicylate	3	208.0	26.50	9.06	34.2	None	
J. D.	Regular	Salol	3	172.0	19.40	4.88	26.1	1.5	
L. E.	Regular	None	4	69.0	12.60	4.80	38.1	...	Marked hyperacidity with intestinal catarrh.
L. E.	Regular semisolid	None	5	108.0	9.02	1.81	20.1	18.0	
L. E.	Regular	Salol	3	230.0	39.80	15.99	40.2	None	
L. E.	Regular	Lactobacilline	4	112.0	11.10	3.99	36.0	2.1	
L. E.	Regular	Aspirin	3	94.0	12.40	4.37	38.3	2.8	
L. E.	Regular	Thymol	3	85.0	10.60	3.84	36.3	1.8	

2. Beta-naphthol and bismuth salicylate appear to be our most effectual intestinal antiseptic drugs in normal individuals, while aspirin and ichthalbin effect slight reduction, and salol gives no results whatever.

3. The results produced by means of intestinal antiseptics in patients suffering with gastro-intestinal disturbances, do not seem to be marked, whereas the best results are obtained by regulation of the diet.

In conclusion, we wish to express thanks to our laboratory assistant, Mr. A. Burton Eckerdt, for his valuable assistance in carrying out the experimental portion of this work.

A CASE OF CARCINOMA ON DIVERTICULITIS OF THE SIGMOID.

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Clinical Report by H. Z. Giffin. In a previous paper,[1] Dr. W. J. Mayo and the authors of this paper reported the operative technique, the pathological findings, and the clinical histories in five cases of acquired diverticulitis of the sigmoid. Since that time several important articles have appeared upon this subject, notably those of Telling, Brewer, and Franke. Telling's paper[2] is an exhaustive study of 105 cases collected from the literature, including those cases operated upon, those discovered at autopsy, and a number found upon reëxamination of numerous museum specimens. Judging from the abstracts of these cases, 27 were operated upon. In only 12 of these was the condition recognized as diverticulitis at operation, or shortly afterward. Eleven of the 27 were thought to be malignant. In 5 of the 12 cases recognized as diverticulitis resection of a portion of the bowel was done. In 3 cases an inflamed diverticulum was resected, in 2 others a perforation was sutured, in 1 an abscess was drained, and in 1 an anastomosis was made. Brewer's paper[3] report as second operation upon a patient who had previously had an abscess drained (included in Telling's paper). This was the resection of a diverticulum acutely inflamed and about to rupture. The diverticulum was treated like an acutely inflamed appendix.

[1] Surgery, Gynecology, and Obstetrics, July, 1907; Trans. Amer. Surg. Assoc., 1907, pp 240–244.

[2] Lancet, March 21 and 28, 1908.

[3] Jour. Amer. Med. Assoc., August 15, 1908.

Franke[4] reports one undoubted case, diagnosticated before operation and confirmed by pathological examination, in which resection was done. This case is not included in Telling's collection.

In the above list of cases there is but one instance of operation upon what proved to be carcinoma developing upon diverticulitis. The following case, which, through the kindness of Dr. C. Graham and Dr. W. J. Mayo, we have the privilege of reporting, is, therefore, of importance.

H B., aged sixty-eight years (A 18,633), male, gave a four years' history of intermittent complaint. At first there was a bloody dysentery of four or five days' duration, with some low abdominal pain. Then for a year or more the patient felt well. Three years previous to operation he was sick three days with quite a severe pain low in the left side of the abdomen, without vomiting, and with little other complaint. Six months previously he had a similar attack, with vomiting and chill, the pain being well localized in the left lower quadrant. There had been no complaint whatever between these attacks. One month before operation another attack occurred and a mass was palpated. A very slight loss of flesh had occurred in the latter weeks, and there was more or less soreness in the side and an occasional chill. The general condition was quite good. The mass persisted to the time of operation, and was as large as an orange. The patient had noticed some blood in the stools during the last month, and a few times the passage of small hard masses of feces, which stained the water about them a reddish color. In attempting to arrive at a clinical diagnosis Dr. Graham placed carcinoma first and diverticulitis second.

The patient was operated upon January 21, 1909, by Dr. W. J. Mayo. A carcinoma was found at the juncture of the descending colon and sigmoid. It had evidently had its origin in a preëxisting diverticulitis. A number of diverticula were also found without carcinomatous change. The tumor had perforated into the left iliac fossa, and an abscess, very much like an appendiceal abscess, existed, with extensive adhesions. It contained a few drams of sterile pus. The entire descending colon and iliac portion of the sigmoid, including the infected peritoneum, were removed. A lateral anastomosis between the splenic flexure and the pelvic sigmoid was made. Drainage was provided by rubber tissue leading through the incision down to the anastomosis and by two stab wounds, each with two rubber-tissue drains.

The case from the literature above referred to is reported by Hochenegg. The following abstract is quoted from Telling's paper (Hochenegg,[5] 1902): The patient, aged seventy years, had suffered from constipation. A carcinoma of the sigmoid flexure was resected; the flexure showed for a great distance numerous cherry-

[4] Deut. med. Woch., xxxv, No 3.

[5] Verhand. der deut. Gesells. f. Chir. 31

sized diverticula, filled with somewhat hard, plastic fecal matter. The carcinoma was regarded as resulting from the chronic irritation and ulceration of the diverticula, due to the retained feces.

Two other cases are reported by Telling which illustrate the association of carcinoma and diverticulitis. These were discovered at autopsy (Telling,[6] 1907): Male, aged fifty-eight years. Constipation and flatulence for many years. Symptoms worse, with loss of weight, and for some time previous to admission. Shortly after this the patient developed general peritonitis and died. At the necropsy there were found the following: (1) Perforating appendicitis. (2) Multiple diverticula of the lowest four inches of the sigmoid flexure. This was thickened, increasingly from above downward; it was fibrofatty in nature, and was most marked opposite the mesosigmoid. The diverticula were, for the most part, currant-sized, contained fecal concretions, and in many cases entered the appendices epiploicæ. But all were not so situated; some were between the layers of the mesosigmoid. In some, ulceration had occurred, and this had led to the chronic perisigmoiditis, and consequent partial narrowing of the gut. The diverticula had "lipped" openings. (3) At the splenic flexure was a narrow, stenosing carcinoma, with a papillary surface toward the lumen of the bowel. It practically encircled the gut and caused partial obstruction. In it were to be made out several "pockets," suggesting those noted in the sigmoid flexure, but this origin could not with certainty be determined. At the edges of the growth were several "cysts" filled with mucus. The gut between the splenic and sigmoid flexures presented no diverticula. "This case is interesting from the presence of three different lesions which may, however, be connected. Assuming that the chronic constipation caused the diverticula, the thickening of the bowel from the chronic diverticulitis would increase the constipation, and the resulting irritation to the bowel may have occasioned the carcinoma above, even if this was not directly due to the irritation of diverticula at this spot. The increasing obstruction of the bowel from the carcinoma was probably a predisposing cause to the perforating appendicitis." The second case, an aged female, was reported by Stierlin:[7] Multiple diverticula were found above a stenosing carcinoma of the sigmoid flexure.

Diverticulitis of the sigmoid has been brought to our attention frequently enough within the last few years to make us think of its possibility in every case of abdominal cramp, especially when symptoms or signs are later localized in the left lower abdominal quadrant. Most of all should it be considered in a history of recurrent attacks over a period of years, and when a mass is palpable. One is quite safe in excluding a primary carcinoma if the history of attacks runs back for a year or two, thus arriving at a tentative diagnosis

[6] Loc. cit.

[7] Corresbl. f. Schweitzer Aerzte, xxxii, p. 749.

of carcinoma on diverticulitis in case the signs point to present malignancy. Only a provisional diagnosis will, of course, be possible in any case. The weight may remain close to normal even after malignancy has developed, but the general condition of the patient will be better if carcinoma be developing on a preëxisting inflammatory condition than one would expect with a primary cancer of equal size.

If one can ascertain how long a tumor has existed, the relation between this and the patient's general condition may be of assistance in pointing either to a primary carcinoma or carcinoma on diverticulitis.

Not infrequently cases are met clinically which give a history of one or two attacks of abdominal cramp localizing quite definitely in the left iliac fossa. No mass is palpable, but pressure on the gut is painful. Most likely a number of these cases are diverticulitis, and their future history will bear careful watching. Again, if surgical pathologists will carefully examine all specimens of carcinoma of the large bowel for the possible occurrence of diverticula, something will be done toward clearing up the question of the actual frequency of malignant change in diverticulitis.

Pathological Report by Louis B. Wilson. When the specimen was opened longitudinally a fungoid carcinoma about 6 cm. in transverse diameter presented itself. The base of the tumor was on the mesenteric side of the viscus. In examining the specimen it was found possible to pass a probe into a deep pocket which penetrated the carcinoma well within its margin. The specimen was fixed in Kaiserling's fluid and then sectioned longitudinally. Four diverticula were found. The one which had been determined by probing before fixation is shown in Fig. 1. Its lining mucosa had been somewhat damaged by the insertion of the probe, yet its character can be readily seen in the photograph. The carcinoma completely enveloped the diverticulum. It is possible that another diverticulum may have been present at the point occupied by the centre of the tumor mass, as is indicated by some fine dark lines at this point in Fig. 1. It is, however, impossible to say that this is the case, since the mucosa has been so completely destroyed. Fig. 1 also shows very nicely the enlarged glands within the mesentery, which were found, on microscopic examination, to be filled with carcinomatous metastases.

The section knife revealed two diverticula placed laterally to the one just described, and just within the border of the carcinoma. These are shown in Fig. 2. It will be seen that they have penetrated the muscularis, and a large amount of inflammatory tissue has been thrown out in the subserosa, causing adhesions which have been torn away by the operative procedure. In these two the mucosa is well preserved, and it is only their inner ends that are affected by car-

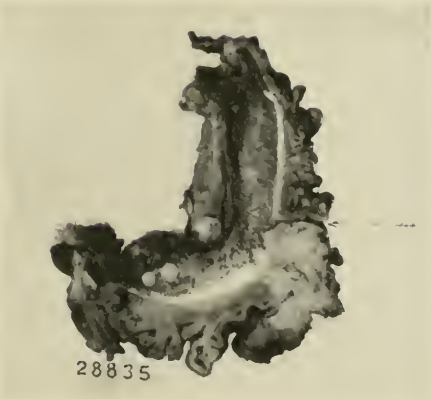

FIG. 1.—Carcinoma on diverticulitis of the sigmoid. The specimen was fixed in Kaiserling's solution, sectioned longitudinally and photographed, immersed without staining. The arrow points to the diverticulum, most of the mucosa of which has been destroyed by necrosis. Above the arrow is seen the main mass of the carcinoma, within the centre of which are some faint lines indicating what was probably a diverticulum. To the left are seen the transverse sections of glands containing carcinomatous metastases. × 2/3 diameters.

FIG. 2

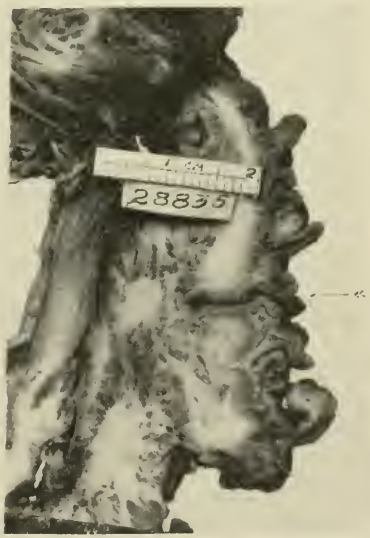

FIG. 3

FIG. 2.—Carcinoma on diverticulitis of sigmoid. The same specimen and technique as Fig. 1. The arrows point to two diverticula just in the edge of the carcinoma. Much of the connective-tissue adhesions have been torn away, as is shown by the gap in which the arrows are placed. × 2/3 diameters.

FIG. 3.—Carcinoma on diverticulitis of sigmoid. The same specimen and technique as Fig. 1. The arrow points to the diverticulum lying completely outside the carcinoma. × 1½ diameters.

cinomatous tissue. A fourth diverticulum was found on the side opposite the preceding two, and completely outside of the carcinomatous area. This is shown somewhat magnified in Fig. 3. This diverticulum is exactly like certain ones previously described by me.[8] It is a false diverticulum, that is, all the walls of the viscus are not carried outward, but the muscularis is penetrated by the coats lying within it. A large amount of inflammatory connective tissue has developed around the base of the diverticulum. The distal end of the pocket contained a small, dark, fecal concretion when it was opened. This specimen is particularly interesting in that it shows various stages in the destruction of previously formed diverticula by a carcinoma developing thereon. The abundant connective tissue formed in the wall of the sigmoid about the outer extremities of the diverticula was, no doubt, the result of a peridiverticulitis, which long preceded the carcinoma. It is, of course, impossible to say whether the chronic irritative process was the only etiological factor in the development of the malignant growth. The conditions, however, would seem to be somewhat analogous to those in the stomach when a chronic ulcer has become the base of a developing carcinoma, a process which pathologists and surgeons are now coming to recognize as the usual association in gastric carcinoma. It would seem important that we go over most carefully all carcinomas of the colon for remains of diverticula. There is no doubt that these anomalies are much more numerous than we have hitherto supposed, and their presence may account in some measure for the relative frequency of carcinoma of the colon as compared with that of the small intestine. Difficulty in determining their presence is due to the fact that most cases of carcinoma coming to operation or autopsy are so far advanced that evidence of preëxisting diverticula may have been destroyed in the necrotic centre of the tumor. Certain specimens of colon carcinoma which I have examined recently strongly suggested the presence of diverticula, but it was impossible to determine their exact etiological relationship owing to the mentioned fact.

CERVICAL RIB AND ITS RELATION TO THE NEUROPATHIES.

By S. P. Goodhart, Ph.B., M.D..

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The congenital anomaly, development of a cervical rib in human beings, may reasonably be interpreted as an evidence of atavism, since in embryos of lower vertebrates every vertebra has a rib

[8] Loc. cit.

attached to it. In reptiles, these embryonic elements proceed to the formation of true ribs; in man, the first seven cervical ribs remain rudimentary. The occasional abnormal formation of a cervical rib originally possessed purely an anatomical and morphological interest, and passed as a curious anomaly, sometimes discovered in autopsies as accidental findings. Clinical significance was given to the condition when hard tumors were demonstrated in the supraclavicular fossa of living subjects, giving rise to symptoms of pressure upon vessels and nerve trunks, with, in many cases, prompt subsidence of these phenomena after extirpation of the tumors. These osseous formations were found to be cervical ribs. More recent research in this direction includes cervical rib among the so-called stigmas of degeneration, or rather anomalies of development, and assigns it a certain place as a combined factor in various neuropathic disturbances. The occurrence of this anomaly was known to Galen, but the knowledge of the present is essentially based upon the monograph of Wenzel Gruber,[1] published in 1869, which contains a compilation of the recorded cases, supplemented by personal observations. The oldest description of cervical rib is probably given by Hunauld,[2] in 1742.

In the great majority of cases the bearer of a cervical rib is not conscious of its presence; but an originally harmless malformation of this kind may act as a compressing tumor, as the result of traumatic periosteitis.[3] Aside from traumatism, the late and slow development of the disturbances, which rarely manifest themselves before adult age, has been referred to loss of "padding," through constitutional diseases, such as anemia and rheumatism (bringing the bony protuberances in direct contact with the nerves and subclavian artery, followed by disturbances in the corresponding area of innervation and blood supply), and to the retraction of the pulmonary apices in healed tuberculous lesions. In each of the four cases observed by Nickol[4] in Senator's clinic, a tuberculous affection of the pulmonary apex was found on the same side as the cervical rib, and in the bilateral cases of cervical rib catarrhal affection was revealed over both apices.

The symptoms caused by a cervical rib may be: (1) Of purely nervous origin; (2) neuromuscular; (3) of circulatory character; (4) of a local nature. The first two groups of symptoms, which are closely related to each other, are those to be especially discussed in this paper and illustrated by my own observation. As is usual in bilateral cases, the symptoms were originally confined to the side of the larger rib.

The patient is aged twenty-eight years. Her history, both family

[1] Ueber die Halsrippen des Menschen, St. Petersburg, 1869, Mém. de l'acad. imp. d. sc., vii Series, 13, No. 2.

[2] Mém. math. et phys. d. l'acad. r. d. sc., lvii, 1742.

[3] Wien. klin. Woch., 1896, No. 6.

[4] Inaugural Dissertation, Leipsic, 1906.

and personal, is entirely negative from a neuropathological point of view. Symptoms referable to the cervical rib date apparently from the patient's eighth year. She then began to suffer pains in the left shoulder radiating down the arm; the entire left upper extremity would become paroxysmally numb. She noticed also gradual development of an indefinite sense of discomfort of the left shoulder; likewise, a protrusion of the left shoulder blade, which, when pressed upon, gave rise to varying degrees of pain and numbness through the left arm. These symptoms persisted. In the course of a few years there was added a certain inability properly to use the left index finger, noticed particularly in piano playing, and gradually a certain lack of dexterity of the left hand. When about eighteen years old, the patient noticed that certain movements of the arm, for example, arranging her clothing, necessitating placing the arm behind her, caused flexor spasm of the muscles of the forearm and hand. At this time there was no weakness and no wasting of the muscles. In October, 1904, the patient noticed weakness in grasp and in finer movements of the left hand. Steadily progressive atrophy of the small hand muscles developed; both thenar and hypothenar surfaces were involved. Soon after, a coldness of the left upper extremity, particularly from the elbow to the finger tips, was at times subjectively felt and objectively demonstrated. It was noticed that when the hand became temporarily cold, muscular weakness became more pronounced for the time being. Paroxysmal attacks of objective pallor and coldness occurred independently of the position of the extremities.

My examination in 1904 revealed areas of hypesthesia and hyperalgesia irregularly distributed over the left forearm and hand. These areas varied, and finally disappeared, leaving, as the only sensory disturbance, an area of hypesthesia for all forms of sensation in that part of the forearm and hand corresponding to the inner cord of the brachial plexus, particularly the ulnar distribution (Fig. 1). In 1904 the left hand presented in appearance the typical *main à griffe*. The following comparative measurements (1904 to 1908) show that the atrophy itself had remained about the same for the past four years (previous to operation):

	Inches. 1904	1908
7 inches inferior to the acromial end of the clavicle, circumference of the upper forearm:		
left	9⅜	9½
right	10½	10½
5 inches below the bend of the elbow:		
left	6½	7¼
right	8⅛	8¾
2 inches above the wrist joint:		
left	5½	5½
right	5¾	6
Circumference of the palm, fingers extended:		
left	7⅛	6¾
right	8	8

I noted some atrophy of the left pectoral muscles in 1904. The same condition existed in 1908 (Figs. 2, 3, and 4).

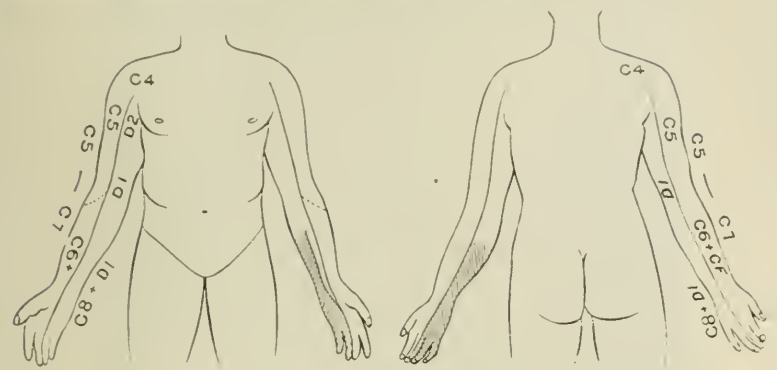

FIG. 1.—Diagram showing the peripheral distribution of the roots of the brachial plexus. *C*1 to *C*8, area of nerve supply of the first to the eighth cervical roots; *D*1, area of supply of the first dorsal root. The shaded area on the left forearm shows the area of diminished sensation—eighth cervical and first dorsal roots—in author's case of cervical rib. This is the area usually affected in cases of cervical rib.

FIG. 2.—Showing muscular atrophy of the left forearm and hand. (Author's case.)

Physical examination revealed an osseous tumor on the left side two inches outside of the sternal insertion of the sternocleidomastoid muscle and extending vertically two inches from the upper clavicular

Fig. 3.—Showing atrophy of the left palmar muscles. (Author's case.)

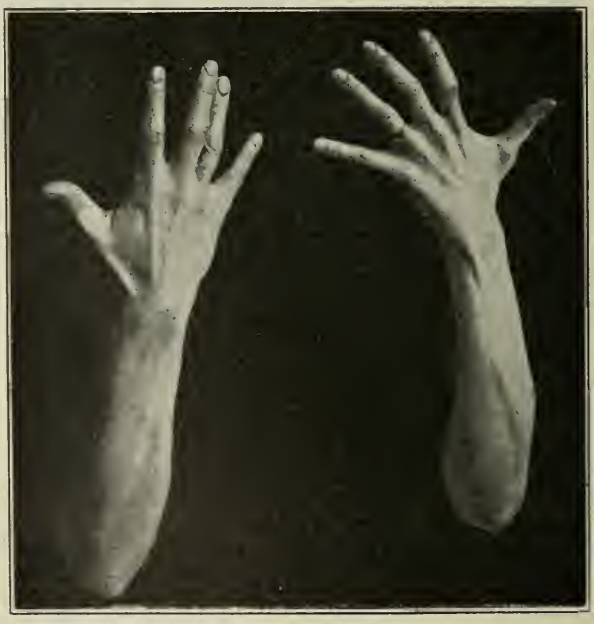

Fig. 4.—Showing particular atrophy of the interossei of the left hand. (Author's case.)

border. On the right side a far smaller eminence was found in about the same position (Figs. 5 and 6). The *x*-rays disclosed scoliosis

FIG. 5.—Showing the positions above the clavicles at which the osseous growths (false ribs) were seen and felt. (Author's case.)

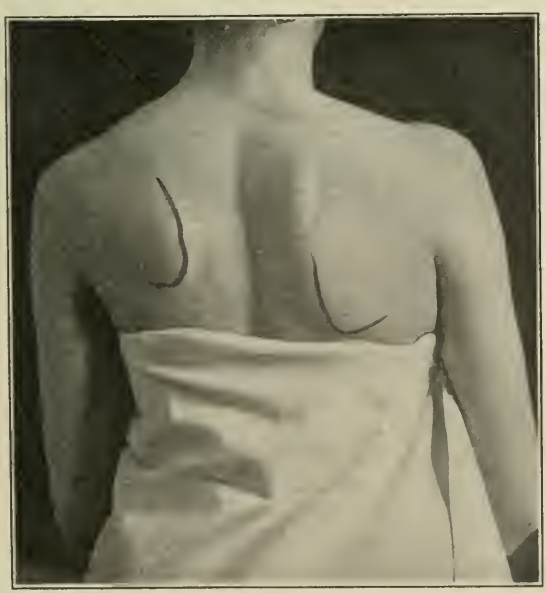

FIG. 6.—Showing the relative positions of the inner and lower borders of the scapulæ. The high scoliosis is also evident. (Author's case.)

extending from the sixth cervical to the fifth dorsal vertebra, with convexity to the left side; compensatory scoliosis below. Bilateral cervical ribs were seen attached to the bodies of the seventh cervical vertebræ, articulating at their distal ends with a facet from the upper border of the first dorsal ribs. On the left side the supernumerary rib measured about one and three-quarters inches; the right one, about two inches in length. At the angle of the cervical rib there was an upward protuberance far more marked on the left side. The scoliosis, as well as the angular displacement of the rib, give it greater prominence and greater interference with the plexus on that side. The third normal rib on the left side was markedly smaller in diameter than its fellows. The radiogram was made by Dr. L. G. Cole; by means of it I was able in 1904 to confirm my diagnosis of cervical ribs (Fig. 7).

Fig. 7.—Radiogram of the author's case made before removal of the false ribs. The supernumerary ribs are indicated by arrow heads. Note the greater prominence of the left cervical rib. Scoliosis is evident. (The radiogram was made by Dr. L. G. Cole, New York.)

In my examination, in 1904, the radial pulse on both sides showed no change in volume. I failed to observe the position of the subclavian artery at that time. During the last year I have observed a diminution in the volume of the radial pulse on the left side; the subclavian artery pushed upward by the false rib, crosses the rib anteriorly; slight compression of the vessel against the rib obliterates the pulse. A distinct bruit but no thrill is heard over the subclavian on the right side only. As will be observed, the vascular symptoms are slight. These observations were made also just prior to operation for removal of the ribs.

It was clinically possible to demonstrate upon careful palpation the exact location of the three roots formed by the fifth, sixth, seventh, and eighth cervical and first dorsal nerves in their relation to the supernumerary rib on the left side. The fifth and sixth were felt lying superiorly about at the summit of the rib and somewhat anteriorly, freely movable. The seventh and eighth were more inferiorly, although pushed upward by the rib. The eighth cervical and first dorsal were deep between the supernumerary and the first normal rib, and could hardly be reached. Pressure with the finger tip upon each of these bands of fibers produced a numbness in the area of the arm supplied by each. The position of the eighth cervical and first dorsal pressed upon as the fibers were, by the cervical rib, explained doubtless the hypesthesia in the ulnar distribution, as well as the muscle atrophy of that nerve supply.

The weight of the patient since 1904 has remained unchanged. In 1906 she gave birth to a child. During the course of her pregnancy, her subjective sensory symptoms, she tells me, were more troublesome. The confinement was normal; the offspring normal, except showing apparent secondary lesions of specific infection. Shortly before the delivery, specific manifestations appeared on the mucous membranes, buccal and vaginal, of the mother. The history of the case points to infection after a second marriage in 1905. Periodic outbreaks of specific symptoms affecting the mucous membranes have since appeared, yielding rapidly to treatment. The patient began, about six months before operation to observe a weakness in the right index finger and a certain want of dexterity in executing the finer movements of the right hand. A slight numbness likewise was complained of occasionally in the right thumb, thus indicating involvement of the right upper extremity. Within the next few months numbness in the right lower extremity, and pains in the calf muscles, were complained of, and I have been able to detect at times a slight but manifest weakness in the flexors of the toes of the right side; no sensory and no electrical changes.

In May, 1908, my patient submitted to the operation for removal of the cervical ribs. The operation was performed by Dr. Alfred S. Taylor, of New York. We found the relations of the rib on the left side and the plexus roots as had been determined by palpation (Fig. 8). The relation of the cervical ribs to the first normal rib had been shown by the *x*-rays. The subclavian artery crossed the first rib, anterior and below the end of the cervical rib, the roots of the plexus lying over the supernumerary rib. The eighth cervical and first dorsal roots crossed the distal end and were undergoing the most tension and pressure. This coincided with the clinical symptoms. The relation of the structures was about the same on both sides. The upper roots were so situated as to be but little compressed. The roots appeared normal to the naked eye. A few fibers of the scalenus muscle were inserted into the upper surface of the extra

rib on both sides. The subclavian artery crossed the first rib anterior to and below the end of the cervical rib; the roots of the brachial plexus were lying over the cervical rib. The cervical ribs on both sides articulated with the bony process attached to the upper surface

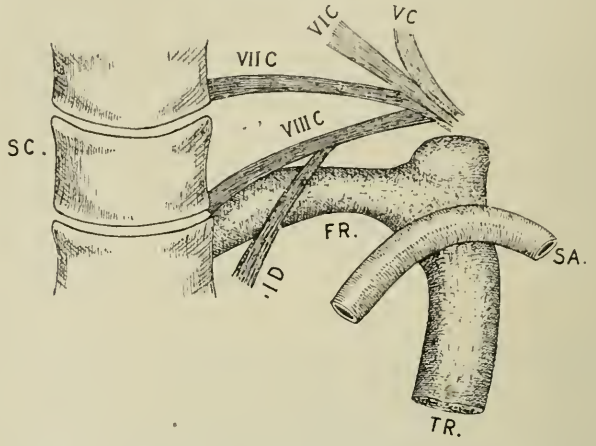

Fig. 8.—Diagram showing the anatomical relationship of the false or cervical rib (*FR*, to the subclavian artery (*SA*); first or normal rib (*TR*); spinal column (*SC*); fifth, sixth), seventh, eighth cervical and first dorsal nerve roots (*VC*, *VIC*, *VIIC*, *VIIIC*, *ID*).

of the first rib. There was a strong fibrous capsule between the two, but very little motion. Fig. 9 shows the size and shape of one of the false ribs after removal. Immediately after the operation there was paralysis of almost the entire brachial plexus distribution,

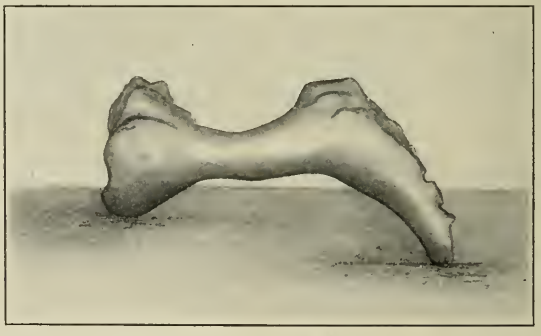

Fig. 9.—One of the false ribs after removal (natural size).

both sensory and motor, affecting particularly the left side. In the course of a week, motion and sensation began to return, and there has been slow but constant improvement. At present, over one year after the operation, there is still very considerable atrophy and still very slight sensory changes along the lower forearm on

the inner side. Fig. 10 shows a radiogram taken after the removal of the cervical ribs; it should be compared with Fig. 7. The details of the technique of the operation will be published later by Dr. Taylor.

Cervical rib as a factor in the estimation of obscure nervous symptoms is entitled to greater attention than it has received. In those cases in which these ribs are discovered, the patient complaining of nervous disturbances, those diseases of the nervous system should be borne in mind which are known to develop upon the basis of an abnormal predisposition, a neuropathic diathesis. At the same time, the hereditary and family relations must be taken more seriously into consideration. Cervical rib was observed in two

FIG. 10.—Radiogram of the author's case, made after surgical removal of the false ribs. (The radiogram was made by Dr. L. G. Cole, New York.)

sisters, by Israel.[5] In all the cases of cervical rib which are brought to the physician's attention, the entire nervous system should be examined. The condition is often combined with other nervous diseases—especially syringomyelia—which are not due to the cervical rib, but arise on the common soil of the same neuropathic diathesis (Oppenheim[6]). Hysteria and hypochondria have been observed in patients having a cervical rib, and Oppenheim mentions two cases presenting symptoms of gliosis, in whom a direct compression of the recurrent nerve had been assumed on the basis of the

[5] Berl. klin. Woch., 1901, 1189.

[6] Lehrb. d. Nervenkrankht., 1905, vol. i; Berl. klin. Woch., 1901, No. 47.

recurrent paralysis. Sensory disturbances which are not limited to the region supplied by the brachial plexus point to the presence of other nervous affections. The coincidence of nervous diseases, arising upon the basis of congenital anomalies of development, assumed by Oppenheim, is of especial interest in patients having cervical rib.

Anatomy and Embryology. Cervical ribs correspond to the processus costarius of mammals, and originate through the failure of fusion between the processus costarius and lateralis, with continued growth of the former (Aron[7]). Their location is at the anterior aspect of the transverse process of the sixth and seventh cervical vertebræ, which possesses an independent bony centre. (The transverse processes of the cervical vertebræ consist of an anterior and a posterior segment, between which lies the foramen transversalium. The anterior shank of this transverse process corresponds to a costal rudiment, and has accordingly been designated as processus costarius.) If the processus costarius of the seventh cervical vertebra is abnormally well developed, the result is a cervical rib. As a rule, in these cases, a supernumerary rib of unequal development is found on either side, one side presenting a short stump, the other a more or less well developed rib.

At a very early period of fœtal life the "Anlage" for the ribs is found along the entire vertebral column, from the highest to the lowest segment. The development of true ribs is confined to the thoracic region, whereas the "Anlage" in the cervical and lumbar vertebræ undergoes retrogression into short rudiments, which may still be recognized by the peculiar configuration of the transverse processes. In certain rare cases, accessory ribs have been observed in the lumbar region, but this anomaly is without clinical importance. There is no case on record in which a supernumerary rib had developed in both lumbar and cervical regions in the same individual. Genuine cervical ribs articulate almost invariably with the seventh cervical vertebra, and are composed, as a rule, of a head, neck, and tubercle. The course of ossification presumably resembles that of normal ribs, in which the centres make their appearance in the head and tubercle about the age of fifteen years, fusion with the rest of the rib taking place about eight years later. In the majority of the cases the anterior extremity of the cervical rib is curved off downward at a right angle, the bone becoming continuous with the first rib.

Cervical ribs discovered accidentally at autopsies, or in living individuals, are by no means rare; Pilling,[8] in 1894, was enabled to compile 139 authentic cases. Women seem to be somewhat more subject to them than men; Ranzi[9] found 17 cases in women against

[7] Berl. klin. Woch., 1892, p. 826

[8] Inaugural Dissertation, Rostock, 1894.

[9] Wien. klin. Woch., 1903, No. 10..

12 in men, and Bernhardt's[10] observations were to the same effect. Among the 42 patients operated upon, collected by Keen,[11] there were 31 females and only 11 males. The fact that cervical ribs are, as a rule, bilateral (67 per cent.) has been established by the *x*-rays; one is always better developed than the other, and usually at a higher level, as is evident in my own case. A complete cervical rib, extending from the spinal column to the sternum, is very rare. There are two cases on record in which two cervical ribs were found on the left side, and likewise but two cases in which symptoms were well developed on both sides. My case makes the third. The anomaly was most frequently discovered in patients past twenty years of age, and some patients had actually passed the half-century mark (Rutkowski,[12] Keen's case). In rare cases the symptoms date from childhood (my case).

Gruber's classification has been so frequently repeated that the more recent grouping suggested by Blanchard[13] is given here, after Kammerer:[14] First group, in which there is a complete supernumerary rib attached to the sternum; only one autopsy recorded. Second group, in which the cartilage of the supernumerary rib is united with the cartilaginous end of the first dorsal rib; also a rare occurrence, but more common than the first variety. Third group, in which the two extremities of the rib are developed as bony structures, but the intermediate portion is represented by a band of fibrous tissue. Fourth group, in which the two extremities of the rib are developed, but not united by a fibrous band. The anterior extremity may be of bone or cartilage, and it may be attached to the sternum or to the cartilage of the first rib. The posterior extremity is practically always attached to the seventh cervical vertebra, and its anterior end is either free or united to the first rib by an articulation or by bony tissue. Fifth group, in which the supernumerary rib is represented only by a segment attached to the vertebra and there is no indication of an anterior extremity.

The clinical symptoms of cervical rib are more readily understood upon the basis of a similar classification, being without exception referable to pressure upon important adjacent structures, namely, the roots of the brachial plexus and the subclavian artery. As a rule, both the nervous and vascular phenomena develop gradually, up to several years, but the onset may be abrupt, probably determined by traumatism.

Nervous Disturbances. Cervical rib must be admitted as an etiological factor in local neuralgias of the arm and hand. In a

[10] Berl. klin. Woch., 1901, p. 1189.

[11] The Symptomatology, Diagnosis, and Surgical Treatment of Cervical Ribs, Amer. Jour. Med. Sci., 1907, cxxxiii.

[12] Zur Diagnostik der Halsrippen, Ztschr. f. klin. Med., 1906, 60.

[13] La revue scientifique, 1885, i, 724.

[14] Cervical Ribs, Annals of Surgery 1901, 34.

general way, the symptoms caused by interference with the brachial plexus, which originate not so much as the result of pressure, but rather of stretching and pulling of the plexus above the supernumerary bone, are not in proportion to the size of the rib. Small cervical ribs may cause considerable trouble, and large ribs may be relatively harmless, since the nerves pass across the first portion of the cervical rib, and the further development of the bone is accordingly a matter of indifference for the plexus (Seiffer[15]). The disturbances caused by compression of the brachial plexus are rather sensory than motor. The first of all nervous symptoms is pain, radiating down the arm, sometimes as far as the hand (my case). After a period of paresthesia, hyperesthesia, or hyperalgesia, there follows a diminution of sensation. In regard to the sensory loss in cases of cervical rib, analgesia is said, as a rule, to be more pronounced than tactile anesthesia (see Fig. 1), suggestive of a root lesion, as first pointed out by Sherrington. Ataxia may be present, but complete paralysis has not been reported.

The typical sensory change of cervical rib consists in the loss in the distribution of the eighth cervical and first dorsal root, more particularly the latter, which may be exclusively involved. It is this nerve which supplies the fibers to the nerve of Wrisberg, or lesser internal cutaneous nerve, the smallest of the branches of the brachial plexus. It will be remembered that the nerve of Wrisberg is distributed to the integument of the inner side of the arm, the back part of the lower third as far as the elbow; and that it connects with the posterior branch of the internal cutaneous nerve. The ulnar nerve distribution is likewise involved.

Owing to involvement of the ulnar nerve fibers, the inner side of the forearm is likewise usually hypesthetic. The exclusive involvement of this area alone should direct attention to the possibility of cervical rib. The involvement of the fibers from the eighth cervical and first dorsal branches, resulting in hypesthesia along the ulnar surface of the forearm, is well explained by Howell. The first dorsal route, as it becomes part of the brachial plexus, passes almost directly upward on the neck of the first thoracic rib; it thus easily comes in contact with the false supernumerary rib, curving downward and forward. As Howell has observed, the longer ribs cause less pressure than the shorter ones. The first dorsal route lies on the upper surface of the neck and body of the first rib, and in lateral movements of the neck and head may be compressed against the seventh cervical rib, though when the head is held in a straight position the nerve may not be in contact with the cervical rib.

Neuromuscular Disturbances. The symptomatology of cervical rib may closely resemble that of a lesion of the first dorsal branch of the brachial plexus. A connection between wasting of

[15] Monats. f. Psych. u. Neurol., vol. xvi, Heft. 4.

the intrinsic muscles of the hand and the presence of cervical ribs was first pointed out by Thorburn[16] in 1904. Two cases of atrophy of the intrinsic muscles of the hands, reported by him, are shown by the *x*-rays to be associated with the presence of cervical ribs. Lewis Jones,[17] upon the basis of *x*-ray examinations of all his accessible former patients with this type of muscular atrophy, agrees with the above assumption of cervical rib playing an important part in the production of atrophy of the muscles of the hand. "Well-marked cervical ribs were found to exist in 10 out of 14 cases examined." Moreover, an extra rib was present on both sides in eight patients who presented symptoms on both sides; whereas there was a cervical rib on the affected side alone in two patients with wasting of one hand. As pointed out by Keen, the muscles involved point to the lower cords of the brachial plexus, and not to the upper. According to him, the wasting is especially marked in the thenar and hypothenar muscles, but the interossei and forearm muscles may be affected, and there may be loss of power in the muscles of the shoulder and the chest. As to the cause of the muscular atrophy, it seems to be more rational to seek this in the compression of the nerves rather than in the diminished blood supply. This view seems to be borne out by the results of surgical interference, and is substantiated by my own observation.

Vascular Disturbances. Circulatory symptoms may be entirely or nearly absent, even in well-marked cases of cervical rib, as in the cases reported by Fischer[18] and myself—for the reason that the subclavian artery passes in the normal way over the first rib. There is generally a well-marked pulsation in the neck, but this is not invariably the case. Aneurysms of the subclavian artery, in the portion lying between the rib and the clavicle in the supraclavicular space, and thrombosis in the arteries of the upper extremity have been observed as the result of pressure from a cervical rib. Grave nutritive changes are of uncommon occurrence, but endarteritis obliterans and gangrene of the finger tips have been reported as a result of arrest of circulation. In milder cases the temperature of the arm on the side of the rib is lower, and the hand has a tendency to become pale and bloodless on trifling exertion. The subclavian vein does not suffer as does the artery, since its anatomical position gives it abundant space.

Local Evidences. A pathognomonic local sign of cervical rib does not exist. A more or less vertical osseous protuberance above the middle of the clavicle, in the lateral cervical region, especially when combined with superficial pulsation of the subclavian artery, and pressure symptoms in the brachial plexus, may be regarded as

[16] The Seventh Cervical Rib and its Effects upon the Brachial Plexus, Medico-Chir. Trans., 1905, 88, p. 109.

[17] Cervical Ribs and Atrophy of the Hand, Quart. Jour. Med., 1908, i, 2.

[18] Deut. Ztschr. f. Chir., 1891-92, xxxiii.

a presumptive, but hardly as a positive sign. A guarded diagnosis of cervical rib under these circumstances should be confirmed by means of the *x*-ray plate.

Scoliosis is almost invariably associated with cervical rib. Their relationship as to cause and effect is in dispute. I believe that the scoliosis is not a simple mechanical effect of the cervical rib. A careful study of the anatomical features of the cases reported in the literature does not support the view that the scoliosis results from the mechanical displacement. Among 400 cases of scoliosis, Helbing demonstrated by *x*-rays that 2 per cent. were accompanied by cervical rib. This author describes two characteristic features of this form of scoliosis, high position of a very rigid scoliosis, and turning or mal-position of the head with asymmetry of the face skeleton and resulting difference in the contour of the neck and shoulder on the two sides.

Levi[19] in his report of a case of bilateral cervical rib, in a girl having multiple sclerosis, warns us against a premature diagnosis of cervical rib on the basis of the *x*-ray picture. The transverse process of the seventh cervical vertebra is so prominent in *x*-ray exhibits that in two other cases the diagnosis of cervical rib of the first degree was rendered by experienced *x*-ray specialists. One of these was a case of syringomyelia, the other of plexus neuritis, and the diagnosis of cervical rib was proved to be erroneous by comparison with a series of radiograms of normal cases. In this connection it is of interest to note Jones' comment on a camera photograph of a bony specimen in the Museum of the Royal College of Surgeons (see Hinds Howell's paper[20]), which has cervical ribs on both sides, but the one on the left side is short and irregular in shape, and lies in close relationship to the first true rib. "Such a rib photographed by *x*-rays in the living subject would probably give an impression on the plate of an overgrown transverse process of irregular outline, an appearance which is sometimes seen in *x*-ray photographs of the cervical region."

Association with Nervous Diseases (Degenerative Type). There are several cases on record of syringomyelia (Borchardt,[21] Oppenheim, Marburg[22]), one of multiple sclerosis (Levi), and one of cervicobulbar palsy (Spiller-Gittings[23]), in individuals having cervical ribs. One of Murphy's[24] patients had suffered for thirteen years from an enlargement of the thyroid gland. In cases of this kind it is permissible to assume the presence of some defect of the central nervous system from the embryonic period, the cervical rib repre-

[19] Neurol. Centralbl., November 1, 1894.
[20] Symptoms Produced by Seventh Cervical Ribs, Lancet, June 22, 1907, i.
[21] Symptomatologie u. Therapie d. Halsrippen, Berl. klin. Woch., 1901, p. 1265.
[22] Syringomyelie und Halsrippe, Wien. klin. Rundschau, 1906, xiii.
[23] New York Med. Jour., October 5, 1908.
[24] The Clinical Significance of Cervical Ribs, Surg., Gyn., and Obs., October, 1906.

senting merely another congenital anomaly. Precisely as in syringomyelia, other changes have been repeatedly met with which had to be interpreted as congenital anomalies of development. Cervical ribs may be associated with a symptom complex which cannot be referred to compression of the plexus, but must be interpreted as manifestations of an hysterical or hysteroneurasthenic type. Oppenheim believes that when this combination is more carefully sought for, numerous cases of cervical rib will probably be found in which the nervous symptoms, even when localized in the arm of the same side, cannot be referred to compression. Syringomyelia in these cases is not uncommonly introduced by a scoliosis, which causes the cervical rib to protrude, giving rise to traction upon the brachial plexus, with the concomitant symptoms.

Sequels of Cervical Rib in the Skeleton. Scoliosis has been frequently reported in association with cervical ribs, the convexity being, as a rule, toward the side of the extra rib, or toward the larger rib in bilateral cases. Four cases of cervical rib, observed by Garré,[25] all presented primary scoliosis in an unusual location, namely, at the transition of the cervical rib into the thoracic spinal column (see my case). Drehmann,[26] who made a special study of this type of scoliosis, arrived at the conclusion that the majority of his cases (10) of primary cervicodorsal scoliosis, limited to a few vertebræ, were due to supernumerary ribs. The motor paresis and gradual muscular atrophy observed in the scoliosis of cervical rib are ascribed by Hoffa to the constant pressure upon the brachial plexus. In view of the small number of clinical cases of cervical rib hitherto published, the relatively high percentage of coincident scoliosis is certainly noteworthy; but while there is an etiological connection between a special form of scoliosis and cervical rib, this does not imply that every cervical rib is necessarily followed by scoliosis. Hochenegg[27] calls attention to the fact that congenital scoliotic curvatures of the cervical vertebral column are very common additional findings in cases of cervical rib.

Treatment. In the various desultory writings upon the subject of supernumerary rib there is no approach to unanimity of opinion as to treatment. It is only too generally conceded that conservative measures are indicated when no symptoms are present or only those of moderate pressure on the plexus. Removal of the rib, according to some observers, remains as an emergency procedure when palliative measures have failed. It is true that cervical rib is frequently associated with central nervous disease, to which the symptoms may be largely referable. A recent writer on the subject, Hinds Howell, maintains that palliative measures be tried even in severe cases. In the case reported by Kammerer the atrophic

[25] Ztschr. f. orthop. Chir., 1903, ii.
[26] Allgem. med. Ctrztg., 1906, No. 2.
[27] Lehrb. d. spez. Chirurgie, 1907.

muscles of the left arm were in the same condition three and one-half months after the pressure on the plexus had been removed. Howell reports two cases and Furnrohra[28] a third, in which operation was followed by complete paralysis of the arm. The plexus not having been ostensibly damaged, it is claimed that the nerves reacted thus to surgical traumatism. It is possible that the report of these cases was given too soon after operation. One year at least should elapse before one may judge of what degree of repair to expect. Removal of the rib in Marbourg's case was followed by rapid progress of the trophic and sensory disturbances, with progressive symptoms of syringomyelia. This writer is opposed to operation in complicated cases. One of Israel's patients was operated upon for a severe brachial neuralgia without motor disturbance. The operation had no effect whatsoever upon the neuralgia, and a serratus paralysis was added when the patient was seen a few months after. Beck[29] is likewise of the opinion that the removal of a cervical rib which causes no disturbance is unwarranted. Rawling[30] says, on the basis of two cases operated upon by him and two others operated on in St. Bartholomew's Hospital, that complete removal is usually a matter of the greatest difficulty, due to the small area of operative field and the numerous muscular attachments which the rib receives. In removing these, many small but troublesome vessels are divided. Complete removal, with ablation of the projecting part of the bone only, may be followed by a temporary relief of the symptoms, which return, however, as the result of new bone formation at the free extremity of the resected rib. My own observation does not substantiate this statement. In Beck's case the symptoms returned with undiminished vehemence three months later, and another operation became necessary. Jones mentions two instances illustrating a return of pain and muscular weakness and due to new bone formation. It is therefore imperative that the removal be complete, that is, that the periosteum be removed together with the rib.

It is doubtless true that the removal of a cervical rib requires an experienced and dexterous operator. The very marked advance, however, in modern surgical technique, especially in what might be called neurological surgery, leads us to a greater degree of boldness in handling surgically those cases of cervical rib in which serious disturbances on the part of the nervous or vascular system manifest themselves. The achievements of later-day surgery in resection of nerves, especially parts of the brachial plexus, warrant a more radical procedure and assurance of better results than have hitherto attended operations. In view of the rapidity with which compression symptoms of cervical rib sometimes develop, it may be

[28] Quoted by Hinds Howell.
[29] Jour. Amer. Med. Assoc., June 17, 1905.
[30] Ibid.

questioned whether the removal of these ribs be advised when their presence becomes clinically demonstrable.

The salient points in the case which I have presented might be summed up as follows: (1) Onset of symptoms at an unusually early age (seven years). (2) Severity of symptoms, which were practically limited to the nervous system. (3) Hypesthesia in the region supplied by the inner cord of the brachial plexus (ulnar distribution). (4) Progressive atrophy of the small muscles of the hand, including the thenar and hypothenar eminences. (5) Cervico-dorsal scoliosis. (6) Stationary character of the symptoms referable to the cervical rib, in the absence of surgical intervention. (7) Demonstration of the exact location of the roots of the brachial plexus in their relation to the supernumerary rib on the left side; digital pressure upon each producing numbness and tingling in corresponding area of arm. (8) Diminution in size of the third normal rib on the left side, well shown in the radiogram. (9) Beginning symptoms due to pressure on the opposite side. (10) Surgical removal of both cervical ribs.

THE INTERPRETATION OF APHASIA.

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General Considerations. The novel and revolutionary views of Pierre Marie, the vigorous defence of the classic theories by Dejerine, the noteworthy revisions of the subject by Moutier and by von Monakow, and the brilliant discussion before the Neurological Society of Paris have aroused a keen interest in aphasia, and have given an especial zest to its study. Like others, I have studied anew the material at my disposal, and have endeavored to lay aside, for the time being, previously acquired impressions and beliefs. The interpretation of aphasia is most important, as this affects vitally our conceptions of cerebral function in its entirety. That which follows presents in brief the point of view at which I have arrived. The cases used in illustration are, of course, cited merely in outline.

Whatever view we may be disposed to take with regard to the nature of aphasia, whether we accept the older or classic view, that aphasia consists of two primarily and essentially different elements, sensory and motor, or whether we hold with Marie that aphasia is a unity the essential characteristic of which is an impairment of the faculty of speech comprehension, it will best serve the purposes of

[1] Read at the International Medical Congress, Buda-Pest, September 1, 1909.

an analytical study if we begin with general considerations. Clearly our first task is to consider the feature, if any, common to all forms of aphasia. That such a feature exists is practically admitted by all writers. If we begin with an analysis of so-called motor or Broca's aphasia, we find, of course, first, a loss or impairment of the power of speaking, but also in addition an impairment of the power of comprehending speech, both spoken and written. I need only cite the well-known position of such stanch exponents of the classic view as Dejerine as to the existence of alexia, and of Thomas and Roux as to the failure of the comprehension of spoken speech.

Dejerine tells us that in true cortical motor aphasia or Broca's aphasia, mental reading is always affected, that the patient reads but without comprehending what he reads, and that the sight of a word does not evoke a corresponding idea in the patient's mind. As regards the comprehension of spoken speech, Dejerine tells us also that the functioning of the auditory images is not absolutely perfect; that if the examiner speaks a little rapidly to the patients, the latter often fail to grasp completely or at once the exact sense of what is said; that the same thing occurs when the sentence spoken is a little long. These statements are in keeping with general experience. Thomas and Roux have likewise shown, by their method of syllabic analysis, that the auditory interpretation of spoken speech is impaired in motor aphasics. Their method is briefly as follows: The patient being shown an object, there are pronounced before him several syllables, among which are found, it may be, the first, the last, or the intermediate syllable of the name of the object. The first syllable is quite often recognized by the aphasic, but the last or the intermediate syllables are never recognized. Failure or impairment of recognition of spoken words is thus absolutely demonstrated. Failure or impairment of recognition of speech, spoken and written, can only result in failure of speech comprehension.

That in so-called sensory or Wernicke's aphasia we have likewise an impairment of auditory and visual interpretations goes, of course, without saying, and it, therefore, results as a matter of simple logic that Broca's and Wernicke's aphasia have one symptom in common, namely, an impairment of speech comprehension, and it would further appear that such differences as may be noted are differences in degree and not in kind. From these preliminary considerations, therefore, it results that the subject which must of necessity first claim our attention is that of impairment of speech comprehension.

When we pause to consider the nature of speech comprehension or speech recognition, the theory of verbal images, auditory and visual, at once suggests itself, and yet no second thought is necessary to realize how unsatisfactory this theory is. That it explains nothing, but only obscures the problem, soon becomes apparent. Its hopeless inadequacy and insurmountable difficulties have already been pointed out by others, and I will not take time to consider them at length.

Suffice it to say that the theory of images means merely that every object seen or heard is in some way duplicated or imaged in the brain, and that these images are stored up in definite centres. If there be images of things heard and seen, there must also be images of impressions received through all of the other senses. Again, to make the application of the theory complete, we must in the case of speech also add motor images—whatever that may mean. Finally, we must suppose that there are special areas in which these images are combined, associated, or otherwise dealt with, in order to make thinking possible. No adequate conception can be formed of such images and such processes, and in truth the theory deals not so much with ideas as with words.

It is perfectly legitimate to attempt another explanation of the function of language, and although such an explanation must of necessity be largely speculative, it will be seen that it is, at least, possible to couch an explanation in physiological terms. Clearly the evolution of speech recognition or speech understanding must have been preceded by the evolution of a sense of simple sound recognition; that is, the first step must have consisted in the formation in the mind of primitive man or of his progenitors of an association of the sounds of nature with the objects or phenomena giving rise to these sounds, just as man has learned to associate the tactual qualities of the objects of nature with the objects themselves. The evolution of the faculty of sound recognition is, in a sense, therefore, analogous to the evolution of the faculty of stereognosis. In stereognosis there is an association of the tactual qualities, in sound recognition an association of acoustic qualities.

In other words, to man's knowledge of the external world, made up of the perception of tactual qualities, there is added, among other things, a knowledge of the external world made up of its qualities of sound. The primitive recognition of natural sounds, that is, the association of the sounds of nature with the phenomena giving rise to them, is a faculty which man doubtless shares with other animals. It must of necessity, in addition, form the basic substratum upon which in him the more complex function of language has been built. Certainly it seems legitimate to assume that language recognition and language comprehension have been evolved from this faculty of the primitive recognition of sounds. In ordinary sound recognition there is the immediate association of the sound with the corresponding object in nature. In the sound recognition of speech there is the recognition of definite sounds associated not directly with objects in nature, but with the conceptions of these objects. The steps by means of which spoken speech was evolved will probably never be known and will forever remain a matter of speculation. It is fair to assume, however—at least, it does no violence to any laws of probability—that the first attempts at spoken speech consisted of efforts on the part of man to reproduce sounds similar to those heard by him

in nature. It would seem that in some such way as this notions and ideas were in the beginning communicated to his fellows, no doubt vaguely and imperfectly at first. Based upon such rudimentary beginnings, a progressive evolution of spoken speech and speech comprehension is readily conceivable.

Association. Having formed this elementary conception of the speech function, and having divested our minds entirely of the theory of speech images, let us turn our attention to disturbances of speech comprehension. In doing so, we must, of course, keep separate the differences obtaining between the failure of simple sound recognition and the failure of speech recognition. Looking upon the term stereognosis as a convenient model, simple sound recognition can properly be termed acougnosis and speech recognition logognosis. Acouagnosis and logoagnosis would mean the loss or impairment of these functions respectively, and would be analogous to stereoagnosis. The study of the speech function is in reality a study of word-sound associations, just as the study of stereognosis is the study of tactual associations. The failure of a word to call to mind the qualities of an object, and to combine these qualities into a conception of the object, must obviously depend upon a failure of association; further, this association involves, as we will see presently, much more than mere sound association. For the present, however, it may be broadly stated that the failure of the recognition of articulate sounds as words or of the meanings of phrases or sentences, is dependent upon a defect of association, that is, a defect in the power of the combination of auditory impressions—of auditory impressions among themselves and also with impressions received through other channels.

That association is involved has, of course, been many times recognized by students of aphasia, but that it is the main or essential feature of aphasia has not been generally held. Approached from this point of view, the study of aphasics becomes exceedingly interesting. The peculiarities presented by the defects of association vary greatly in different cases, both in the degree in which the symptoms are present and to some extent as to the kind of association that is defective. In order to make my meaning clear, I will use as illustration briefly the following case, which belongs to one of the large group of aphasics always at our command for study in the nervous wards of the Philadelphia General Hospital:

W. E., aged thirty-three years, a male, right-handed, was admitted to the Philadelphia General Hospital October 30, 1905. He presents the symptoms of a typical, spastic, right-sided hemiplegia with aphasia. Spontaneous speech is limited to yes and no and to an imperfect pronunciation of his name. Agraphia and alexia are also present. In other words, he presents the clinical picture of a so-called motor or Broca's aphasia. Apparently, he is able to comprehend much of what is said to him, much of what is going on about him, and conducts himself properly in the ward and at the table.

However, when his faculty of comprehending spoken language is tested in detail, as by the method of Marie, it is found that he is able to comprehend simple instructions only, such as, "Place your hand upon your head," "Place the pencil upon the paper;" at most he can carry out consecutively only two such instructions. He also fails to carry out an instruction correctly when it embraces, instead of two objects, three or more. When asked to count, he says, "One, two, three," but is unable to proceed any farther. Although he cannot read, he is asked to copy the written word "boy;" this he is unable to do. The word is then placed before him in large printed characters; he makes a drawing of the letters, but does so very imperfectly. Asked to repeat monosyllabic words, such as boy, girl, cat, man, milk, etc., he is able to say them, but with very imperfect enunciation; there is a marked dysarthria. A number of objects having been placed before him, he is unable to name any of them spontaneously. However, if he is told to point out an object, the name being plainly pronounced by me, such as spoon, knife, fork, cup, pencil, etc., he does so correctly. Now, a given object having been selected, say a tumbler, he is asked, "Is the tumbler rough?" He nods his head in the affirmative and says, "Yes," although the glass is entirely smooth. Asked now whether the tumbler is smooth, he again answers in the affirmative. Questioned a third time whether it is rough, he again answers in the affirmative. An attempt to communicate to him the facts as to this physical quality of the tumbler fails. He is similarly unable to distinguish between such qualifying words as "round" and "square." Errors were made alike if the objects were merely placed before him and if he held them in his hand. It is to be noted that a knowledge of the qualities such as I have mentioned are acquired by the sense of touch, but are normally associated in the mind with the spoken name of the object; they are part of the conception which the spoken name calls forth. Asked, Is the tumbler full of water? He says, "No," which answer is correct. The tumbler being held before him a moment later, he is asked to repeat its name. He now utters the word "water," and when the attempt is made to correct him and he is told that the name is tumbler or glass, he shakes his head in the negative and says, "No."

A number of pieces of colored chalk, blue, white, yellow, and red, were now added to the objects on the table. Asked to pick up the blue chalk and place it on a piece of paper before him, he picked up the yellow chalk. This test was varied and repeated very many times; and the percentage of errors was so large as to lead to the inference that he did not recognize the qualifying words signifying color. It was also noticed that if the instruction at any time embodied three factors, he usually failed in the proper sequence of the things he did; that is, he would become confused and make errors. As far as it was possible to determine, he was not color blind.

He was next tested as to his ability to distinguish prepositions and

phrases indicating relations of space. Thus, a tumbler having been placed in his hand, he was asked to hold the tumbler *above* the table, to hold it *below* the table, to hold it in *front* or to the *side* of the table. Here the percentage of failures was so large as to justify the inference that he did not recognize these words or phrases at all. A similar result was experienced in a test involving the difference between such prepositions as *in* and *on*.

Further, it was easily demonstrated that his recognition of verbs was also very limited. He recognized "put," "is," "hold," "pick up," but he could not make any differentiations of tense. Thus, when he was tested as to "is" and "was," he failed, and in the few instances in which he succeeded his answers were evidently the result of pure guess-work. For instance, a pencil having been laid upon a piece of paper before him, he was asked, "*Is* the pencil upon the paper?" and again, the pencil being removed, "*Was* the pencil upon the paper?" This test, simple in character, was repeated many times; his answers were given "yes" or "no" at random, and there could be no doubt as to his failure to distinguish between these elementary differences of tense.

Tests with other aphasics yielded similar although of necessity varying results, the results being somewhat in proportion to the impairment of the general comprehension, as instanced by the failure of the patient to carry out simple or serial instructions. When the patient was able to carry out complex instructions or a number of serial instructions consecutively, it was usually found that he preserved some knowledge of words and phrases indicating physical qualities, and also relations of time and space; that is, there seemed to be a proportionate recognition of adjectives, prepositions, qualifying phrases, and the ability to make at least simple distinctions of tense. This is well illustrated by the following case:

L. J., aged forty-nine years. Here there is likewise a Broca's aphasia. The loss of motor speech is so complete that the patient cannot say even "yes" or give his name; he says "do" for "no;" when urged to speak, he says "dindo," which seems to be his stock expression.

Question. "What is your name?"

Answer unintelligible.

Q. "Can you say any words at all?"

A. Shakes his head in the negative. Is emotional and weeps.

Q. "Say 'yes.'"

A. Fails.

Q. "Say 'no.'"

A. "Do."

Q. "Say 'all right.'"

A. "Aw aw da."

Q. "Stand up, take your chair, and bring it toward me."

Complies perfectly with the instruction.

Q. "Strike on the floor three times with your cane, then sit down and place your cane on your knee."

Complies perfectly.

Q. "Rise from your chair, take three steps toward me, go back, turn your chair so that it faces the wall, and then sit down."

Complies perfectly.

Three pieces of paper of unequal sizes having been placed on the table, he is asked to take the largest piece, to crumple it and throw it on the floor, to take the smallest piece and hand it to the nurse, and to take the medium-sized piece and to hand it to me. He complies with the instruction except that he not only crumples the piece of paper that he throws on the floor, but also the pieces that he hands to the nurse and myself. When the tests are made still more complex, he invariably fails.

A group of objects having been placed upon the table before him, he correctly indicates the objects as they are named by me. He also recognizes words indicating the physical qualities, such as smooth and rough as applied to a glass, and such physical qualities as color. He also recognizes such words and phrases as indicate space relations, that is, "above," "below," "in front of," "to the side of," etc.

He can recognize his own name when written before him. He recognizes the word "hand" when this is shown to him in either written or printed characters. This is also true of other simple monosyllabic words indicating objects. However, if a sentence is written, such as "Raise your left hand," he cannot construe it and fails to comply with the instruction. In other words, this patient, whose sound recognition of speech has suffered relatively moderately, but who has a very marked loss of motor speech, has associated with this loss of motor speech a very pronounced alexia, an alexia, too, which is especially evident the moment he attempts to combine even a limited number of words into a phrase or sentence. He is also unable to tell the time.

The peculiar inability of the aphasic to combine words into phrases and sentences is shown in a number of ways. Thus, W. E. is able to say after me each of the following words *separately:*

"The," "pencil," "is," "on," "the," "paper." While he is able to pronounce these words with sufficient clearness to be readily understood, he is utterly unable to *combine* them into the sentence "The pencil is on the paper." The most that he can do after repeated urging is to say, imperfectly, "pencil paper."

The following case shows the same thing to be true in the instance of written words:

J. McN., aged fifty-seven years, presents a marked right-sided hemiplegia with contractures and aphasia. His speech is limited to "yes." On rare occasions, when asked, "How are you?" he replies, "All right." He recognizes his spoken name, for he turns around when addressed. Asked whether his name is John, he says, "Yes." Asked

whether it is William, he first shakes his head in the negative, but afterward appears to be confused and in doubt. He is unable to comply with simple instructions, such as, "Raise your left hand." Asked to put out his tongue or to shut his eyes, he fails to comply. He will, however, repeat various gestures made before him in pantomime, but his repetition of the gestures is imperfect; his gestures are, of course, made with the left hand. He feeds himself and appears to recognize such utensils as a spoon and cup, and to use them properly. However, when an open-faced watch is placed in his hand with the face down, he makes no attempt to turn the watch over so that he can see its face. When the face is turned up for him, he looks at the watch, smiles, but gives no sign of recognition. When he is approached from behind and the watch—a loud-ticking one—is held close to his ear, although not touching him, he again fails to give the least sign of recognition. He is not deaf, as is readily demonstrated when his name is pronounced in an ordinary voice, as he immediately turns. He appears to recognize the various articles of his clothing, and assists the nurse when he is being dressed. He has complete agraphia. An attempt to write leads merely to the forming of unintelligible markings upon the paper. He is, however, able to read aloud single words, such as "Ireland," "Lawrence," "Jordan," "leaf," "house," "head," "hand." These words, to our surprise, he pronounces, and although the pronunciation is dysarthric, they can readily be understood. If, however, a simple instruction be written before him, such as, "Put your hand on your head," he fails to comply, just as he does when the instruction is given to him verbally. In other words, he has no power to combine the individual words which he is able to read. He cannot grasp a phrase, either spoken or written, so as to enable him to comply or perform an instruction, no matter how simple.

Reënforcement. Every one who has studied aphasics has noted that the symptoms presented by an individual case of aphasia not infrequently vary. This fact may be observed at different examinations or may be noted in the progress of one and the same examination. While the degree of variation is usually not great, both the fact of its existence and the character of the change are important as throwing an additional light on the nature of aphasia. The variation may be due to several causes: (1) A patient subjected to an examination, even if not very prolonged, may sooner or later present signs of fatigue, that is, the answers or compliance with tests, at first successful, may, as the examination proceeds, fail; (2) it may be noted in repeated examinations that the patient responds more and more readily; in other words, that the examinations are themselves re-training or reëducating the patient. There is, however, a third cause of variation of symptoms much more interesting and much more important than either of these and the phenomena of which can best be explained by a theory of reënforcement.

Every one is surprised now and then by the emission of words and phrases by the aphasic spontaneously when previous examinations had failed to elicit anything more than perhaps "yes" or "no" or some simple expletives. That such phrases are heard under the stimulation of unusual excitement is well known. For instance, one of my patients, whose single utterance is "yes," when I insisted upon his saying the word "no," failed as at previous examinations. Finally, upon my insisting repeatedly and emphatically, his face flushed and he suddenly shouted, "I can't say no," at the same time shaking his head. While I have studied this man a great many times, this is the only sentence that I have ever heard him utter. It is fair to assume that in this instance the general cerebral activity had been forcibly aroused or stimulated, and that this had had the effect of reënforcing the impaired speech function; that is, inhibition had been momentarily overcome by a powerful reënforcing wave.

The curious instance of the singing aphasics can only be met by a similar explanation. For instance, I had under my care some years ago a Broca aphasic, whose speech was limited to "yes," "no," and a few brief expletives. He could, however, sing—among other things—Auld Lang Syne, carrying the air well and enunciating each word clearly. No other explanation, it would seem to me, than that of reënforcement overcoming inhibition can apply in such a case.

Reënforcement in aphasia can be illustrated in a number of other ways. For instance, one of my aphasics who at one examination was able to count as far as three, at a subsequent examination, after he had been stimulated by a number of tests, counted as far as sixteen. Here, again, we have doubtless to apply a principle of reënforcement. Again, another of my aphasics counts as far as twenty. He does so apparently without effort, but becomes confused when twenty or twenty-one is reached. Further, he enunciates the names of the various numbers quite correctly during the act of counting. If, now, I allow a moment or two to elapse, and then ask him to repeat after me one of the intermediate numbers, say thirteen, *separately*, he fails. He makes a sound, it is true, but it is quite unintelligible. Tested with other separate numbers, the result is the same. It would appear that in this case the uttering of the number in serial sequence is, after the start is made, largely a matter of reënforcement. Counting is taught in childhood in such a way that a serial association is early formed in the mind—a serial association basic and strong. In our patient, the count having been started, each number evokes its neighbor in the series; each reënforces the one that follows and necessarily along a pathway of time-worn association. Taken out of its sequence, a number is either not enunciated at all or the enuciation is dysarthric. In our patient it requires the reënforcement resulting from the formation of the previous numbers to make the emission of the given number possible.

The existence of more complex associations and reënforcements can be illustrated in still other ways.

C. A., aged fifty-six years, who early in May, 1908, suffered from a paralysis of the entire right side, can answer "yes" and "no," give his name imperfectly, and can utter a limited number of other words. His enunciation is markedly dysarthric. His name is Christopher A.

Q. "What is your name?"

A. "Christian."

Q. "Is your name not Christopher?"

A. "Yes, Christopher."

Q. "Have you no other name?"

A. "No." (After an interval) "Almus" (incorrect).

Repeated efforts to have him pronounce his name in full and correctly fail.

Q. "What is your age?"

A. "Thirty-eight." (As already noted, his age is fifty-six.)

Q. "What is your business?"

A. After a long delay he answers "Carpenter."

Q. "Where did you work last?"

Answer unintelligible.

Q. "Are you comfortable?"

A. "Yes."

Q. "Do you want anything?'

A. "No."

Q. "How long have you been in the hospital?"

A. "Couldn't tell."

Q. "Can you count on your fingers?"

A. "Yes."

Q. "Count your age on your fingers."

A. Now counts aloud as far as 29, jumps to 40, and then goes back to 21.

He is unable to say the word pencil, although he recognizes the pencil and correctly points at it. When, however, he takes hold of the pencil, handling it correctly, he says, "pencil." The same thing is noted in the case of a key ring. He looks at it, is unable to say the word, but after handling the object for a time says "ring." It would seem as though we have here to do with both stereognostic and visual reënforcement. The visual recognition and naming, by an aphasic, of an object is in a sense the result of a visual reënforcement, and in a similar sense this must be true of its tactual recognition. When the words pencil keys, and ring are said before him, he correctly points out the objects. Other tests were made with this patient with regard to reënforcement by taste and smell. Although not very successful, they were very suggestive. In the case of syrup, for instance, which was poured out before him, but which he would not or could not name, he at once called "molasses" as soon as he tasted it. In a test made with vinegar, concerning which he would say nothing from

merely looking at it or smelling it, he finally called "cider" when he tasted it. He is unable to read even his own name in either printed or written characters. He cannot write or copy. When urged to write, he makes with his left hand the letter "C" backward, as in mirror writing. Asked to write twenty, he puts down 01. He will repeat imperfectly a few simple words, but he cannot combine them into sentences. Once only he repeated after me the sentence, "I am well."

In this instance there appeared to be an unmistakable reënforcement of the word-forming power or of word emission by the perception of tactual qualities in two instances, and by the perception of those of taste in a third. Responses were also at times obtained by urging and insisting upon an answer, although here readiness of fatigue interfered with the results.

The following case, which presents a marked loss of speech comprehension, but no impairment of emissive speech, is interesting because of the failure of both tactual and visual reënforcement to enable the patient to name objects. It is not improbable that the failure was related to the intensity of the loss of speech comprehension.

M. J. O'B., aged forty years, had been admitted to the nervous wards of the Philadelphia General Hospital, with an unsatisfactory history of an injury to the back of the head some months before and the subsequent development of convulsive attacks and "loss of memory." When questioned, a difficulty of speech comprehension at once becomes apparent, although no difficulty of emissive speech, no difficulty of enunciation, no anarthria, can be detected.

Briefly tested at first by a series of instructions, according to the method of Marie, it is found that he frequently fails to carry out simple instructions, such as picking up an object from the table. He invariably fails when the instructions embody two consecutive acts. Thus, asked to pick up a piece of paper from the table, he may comply, but if asked to pick up the paper and throw it into the waste basket, to crumple it in his hand, or to mark it with a pencil, he invariably fails. The same result ensues if the number of the objects with which he has to deal is increased. Among other things, he was questioned as follows:

Q. "What is your name?"

A. Answers correctly.

Q. "Where were you born?"

Looks about him for some time, and finally answers, "Thirty."

When the question is repeated in different ways, he finally answers, "Don't understand."

When the question is now modified, "Were you born in America or Ireland?" he answers, "I was born in Camden."

Q. "Why can you answer as to your place of birth at one time and not at another?"

A. "Sometimes I can and sometimes I can't."

Q. "Are your father and mother living?"

A. "My mother is, my father dead."

Q. "Where was your father born?"

The patient again assumes a look as though he did not comprehend, points back of himself, and finally says, "You can say, I can't."

When the word Ireland is suggested, he nods his head in the affirmative. He is then able to repeat the word Ireland immediately after it has been said before him, and the enunciation is clear and distinct.

Q. "What is your business?"

A. "Buy barrels."

Q. "What do you do with them?"

A. "Work for my brother."

Q. "What did you do with them?"

A. "Sold them."

Q. "Have you any relatives?"

A. "Yes."

Q. "Who are they?"

A. "A man."

Q. "What is he?"

A. "He makes those." (Rises and points to the spigot of the water cooler.)

Q. "What is this man to you?"

A. Again points to the spigot of the water cooler.

Q. "Is he your father?"

A. "Yes."

Q. "Your father is dead. Is he your brother?"

A. "Yes, brother."

He then himself asks the question, "Do you mean Frank?" and then answers the question himself, saying "Yes, brother."

Q. "Are you sitting or standing at present?"

A. No answer.

Q. "Am I sitting or standing at present?"

A. "Yes sir."

Q. "What day is this?"

A. "Thursday" (correct).

Q. "Is it day or night?"

A. "Day."

Q. "Is it morning or afternoon?"

A. "Morning" (correct).

Q. "What is this?" (indicating a chair).

A. "I know, but can't say."

He is now asked to touch it, to lift it, and to sit upon it, but is still unable to give the name.

Q. Asked again, "What is it?"

A. "I can't say."

Q. "What is it for?"

A. "To sit on."

Q. "Point to a table in this room." (The room contains two tables and a desk.)

A. "There isn't any."

Q. "Is there a desk?"

Points correctly at the desk.

Q. "Is this a table?" (indicating a chair).

A. "No."

Q. "Is it a desk?"

A. "No." (Patient again points to the desk.)

Q. "Is it a broom?"

A. "No."

Q. "What is a broom for?"

A. "Anything that is dirty."

Q. "Is this a chair?" (chair indicated).

A. "No."

Q. "What is this?" (indicating a knife).

A. "Can't say."

Q. "What is it for?"

A. "To cut with."

Q. When the knife is placed in his hand, he is equally unable to name it.

Q. "Is it a spoon?"

A. "Can't say."

Q. "What is this?" (handing him a watch). He looks at it, feels it, and places it to his ear, but cannot say what it is. Finally, he says, "I know what it is, but can't say it."

He can repeat readily the names of objects when they are laid before him, but his ability to call the names to mind spontaneously is greatly impaired. When the word "pencil" is placed before him in printed characters, he replies, "Cannot say it." Asked then to point out the object for which the printed word stands, he fails to do so, though there are several pencils on the table.

He correctly indicates, as already stated, his hand, his head, indicates correctly a chair, but fails to indicate a clock, although a large clock is almost directly in front of him. He is able to write his name, but cannot write sentences spontaneously or write at dictation. He copies by drawing the letters imperfectly of words placed before him.

He can read monosyllabic words, such as hand and head, when these are in large written or printed characters. He fails, however, to understand this written sentence: "Put your hand on your head." Cannot construe the sentence and fails to comply with the instruction. When he is told verbally to raise his hand and place it upon his head, he complies, but only after much delay and after repetition of the instruction. He fails in very simple problems of arithmetic, although he forms the figures very well.

In this case, which would, of course, be included in the classical division of the sensory aphasias and in which anomia would be considered as the leading feature, we have of special interest: (1) The occasional breaking through of answers or even spontaneous statements when avenues of reënforcement are opened up by the excitement and stimulation of the examination. (2) Contrary to what we observed in the case C. A., we note failure of tactual reënforcement to bring about the formation and emission of the name when the visual reënforcement fails, and (3) the very marked inability to execute serial instructions. Both of the latter features are doubtless related directly to the degree of the impairment of the speech comprehension.

We are justified from clinical evidence alone in the inference that the area of the cortex concerned in speech recognition is normally in close relation with other areas concerned in the recognition of the physical qualities of the external world. That this relation is intimate in the case of the visual and tactual functions goes without saying; that it also exists in the functions of taste and smell seems equally incontrovertible. The loss of the ability to associate properly, for instance, articulate sounds with tactual qualities, must lie in a break, structural or functional, in the relations between Wernicke's region, on the one hand, and the region of tactual recognition or stereognosis on the other. The same must be true in the case of visual recognition and of recognition by the other senses. It would appear from these considerations that the function of speech comprehension must be very general in its character, one in which very extensive regions of the brain are concerned; and this fact must be borne in mind in estimating the effects of a limited lesion. Here the dangers and difficulties of interpretation are great. The symptoms present may in part owe their existence to disturbances in the relations between the area involved and quite distant regions of the brain.

The phenomena of reënforcement seem to prove that some, at least, of these disturbances of relation are functional; if the break were always structural, reënforcement could at no time occur. Many of the symptoms, therefore, noted in aphasics must be due to inhibition, and the theory of the diaschisis advanced by von Monakow shows very clearly how this change or splitting off of function, or, better still, *suspension* of function, may occur. On no other basis than that of the overcoming of inhibition are we able to explain the various phenomena of reënforcement. In order that reënforcement may occur, a pathway cannot be altogether closed, but need be only so much influenced by a lesion as to impede or suspend for the time being association. Clearly it is association which forms the chief problem in the study of aphasia.

Emissive Speech. In some of the illustrations I have used, reënforcement has of necessity been considered in relation to emissive or spoken speech. When we turn our attention to this faculty, we find that clinical studies reveal additional facts of exceeding

interest. For comparison I selected: (1) A typical case of pseudobulbar palsy; (2) a case which appeared at first sight to be a pure motor or subcortical aphasia; and (3) a typical case of Broca's aphasia. The views of Marie in regard to anarthria urgently suggested such a comparison.

In outline the case of pseudobulbar palsy is briefly as follows:

T. R., aged thirty-five years, suffered a year before his admission to the Philadelphia Hospital from an apoplectiform seizure, which left him with a mild left hemiplegia, from which he rapidly recovered. Three months later he had a second stroke, this time affecting the right side, but also transient in character. After the second stroke it was noticed that his speech was very indistinct. His general condition improved decidedly, but the difficulty in the speech persisted.

When admitted, it was noted that his gait is somewhat shuffling, his legs slightly spastic. The knee-jerks are increased and there are an ankle clonus and Babinski reaction on the left side. The patient is unable to extend his lips or to protrude his tongue beyond the lips. He usually holds his mouth open; has difficulty in holding liquids in his mouth and in swallowing; cannot close the mouth firmly; cannot whistle. The tongue presents no atrophy, but the patient's power to direct it upward, downward, or to either side is greatly impaired. Examination of the larynx fails to reveal positive findings; at one time an abductor weakness, at another an adductor weakness, appears to be present.

It is important also to record that the patient is constantly either smiling or laughing; automatic laughter is a marked feature of his case. To some extent this laughter is reflected in the general tone of the patient, who in reply to questions says that he is happy and not depressed.

When his speech is investigated, it is found that he is able imperfectly to form vowels and still more imperfectly consonants; indeed, the ability to form consonants is so feeble that it can hardly be said to exist. Repeated examinations, however, have made me more or less familiar with his peculiarities, and I am able to interpret much of what he says or tries to say, imperfect as this is.

Of course, there is here no question of aphasia, properly speaking, no failure of speech comprehension. He understands everything that is said to him, he reads, writes, and tells time correctly. He carries out verbal serial instructions correctly as long as these embody only three factors. If they embody four factors, he fails. If the serial instructions are written instead of verbal, he again complies correctly; he again fails, however, when the number of factors is four, unless he stops in course repeatedly to read his instructions. It is also noted that while he is able to do simple sums in arithmetic, he fails as soon as the additions or subtractions become at all complicated. At one time, also, as do aphasics so frequently, he began an addition at the left hand column; possibly embarrassment and con-

fusion played some role in this gross error, which he did not himself perceive; the test was made at the blackboard, and in the presence of a class of students. However, his marked arithmetical incompetence is rather remarkable, inasmuch as he was by profession a draughtsman, and, if he is to be believed, well educated. The question arises, of course, whether in lesions so gross as those of pseudobulbar palsy there is not necessarily some general mental impairment. The fact, however, whatever its explanation, is exceedingly interesting. It becomes especially so when we turn our attention to the next case, the one which, at first sight, seems to be one of pure or subcortical motor aphasia.

R. J., aged sixty-three years, male, right-handed, suffered six weeks before admission to the hospital with a right-sided hemiplegia. This hemiplegia was slight and of short duration. At the time of his admission to the hospital his physical examination was negative, save that the grip of the right hand was not quite as good as the left, and that there was some flattening of the right lower face. He could not retract the right angle of the mouth as well as the left. He could not whistle. He could protrude the tongue well and move it in all directions. He suffered, however, from a marked impairment of speech, and was at once classified as a "motor aphasic."

Examined in detail, it is found that he presents a high degree of anarthria. This is so great as strikingly to resemble the anarthria of the pseudobulbar case just cited. He gives his name as Josup Josun, which is intended for Robert Johnson; pronounces "yes" at times correctly, at times as "gess," and "no" frequently as "do." Enunciation is very indistinct and difficult to follow; often final syllables are not pronounced at all. He counts aloud, but with marked dysarthria: bon (1), tno (2), tee (3), our (4), fiz (5), chick (6), cheven (7), eat (8), etc. He recognizes objects and, making allowance for his high degree of anarthria, names them correctly. He can repeat after me short sentences, but with, of course, marked anarthria. When the sentence contains two phrases, the repetition is very incomplete. When the sentence contains three phrases, each embodying a separate idea, he always fails; what he says becomes completely unintelligible.

The patient is on duty as cook for the night watchmen; the head nurse states that in the mornings he asks for his supplies in one word, or in separate words, never in phrases or sentences. Frequently, instead of attempting to communicate his wants by word of mouth, he will write them; *e. g.*, "Kindly give me some sugar. Dining-room men used mine." Again, "The men in the dining room stopped me taking potatoes for the night men. They grumble when they don't get potatoes. They cook them for supper and breakfast." It should be added that he discharges his duties as cook well. He can also write at dictation simple words. In writing sentences, he usually begins properly, but soon makes mistakes. Tested verbally as to carrying out serial instructions, he complies promptly and readily.

In this respect he does not differ in the least from the pseudobulbar case just cited. If, however—and this is the important point in his case next to the anarthria—if he be handed a series of *written* instructions, he carries them out imperfectly, and even with the written instructions in his hand he sometimes fails altogether. In other words, he betrays an alexia—an alexia which consists in the inability to *comprehend* properly the written instructions. When he attempts to read aloud, only a few words here and there can be understood.

Here, then, is a case the anarthria of which closely resembles that of pseudobulbar palsy, and which is, notwithstanding, associated with an alexia present in a degree; but for this symptom the case would fit exactly into the group of subcortical or pure motor aphasias. Does he not, however, even in this respect resemble the case of pseudobulbar palsy just cited? For, in the latter, let us recall there was also a distinct difficulty in the interpretation of written instructions. The case of R. J. suggests to our minds the query, Does *pure* motor aphasia really exist in nature? Personally, I have never met with it; is it not always pseudobulbar palsy?

The patient suffering from pseudobulbar palsy, we will recall, was constantly laughing; indeed, he presents a typical instance of the automatic laughter not infrequently met with in pseudobulbar palsy. In our third patient we have an instance of a motor aphasic who, like the pseudobulbar case, is constantly laughing. Briefly, W. W., aged fifty-seven years, right-handed, right-sided hemiplegic, has a pronounced anarthria, his speech being limited to "yes" and "do" (for "no"); he understands much that is said to him, executing serial instructions fairly well; he suffers, however, from a pronounced alexia. While he is able to read an occasional isolated word, he fails utterly to construe a sentence and fails, therefore, in executing written instructions. Other than this, he presents no peculiarities save that he laughs, laughs automatically, laughs just as much as does the patient with pseudobulbar palsy. I have occasionally met aphasics who laughed readily, laughed a great deal, but I have never met one who laughs quite as much as W. W. On the other hand, as is well known, there are aphasics who weep readily—who are readily provoked to weeping during an examination. Of course, it may be claimed that laughter and weeping in pseudobulbar palsy and laughter and weeping in aphasia are merely analogous facts and bear no clinical or pathological relations to each other. To me, however, the facts are most suggestive, both clinically and pathologically, especially when we take into consideration, on the one hand, the known involvement of the lenticula in pseudobulbar palsy, and the involvement of the lenticula as claimed in the anarthria of Marie and found to exist among others in one of my own (published) cases. The pathology of automatic laughter and weeping is, of course, not known, that is, if it have a pathology apart from that of pseudobulbar

palsy; that we meet with it at times in Broca's aphasia is not surprising if some relation between the two affections be admitted.

It might be claimed that our case of pseudobulbar palsy is in reality a case of aphasia; that this is shown by the fact that the first stroke, which was left-sided, was not followed by the speech disturbance, but that the latter ensued only upon the second or right-sided stroke. However, the fact that both strokes were slight and transient in character, as they so often are in pseudobulbar palsy, so as to leave little subsequent physical evidence, and the further fact that the patient presents distinct weakness of the lips, that he is unable to hold liquids in his mouth, has difficulty in swallowing, cannot close his mouth firmly, cannot whistle, and, although the tongue presents no atrophy, that he cannot protrude it beyond the lips and cannot move it well up or down or to either side, prove conclusively that the case is one of pseudobulbar palsy and not aphasia, and yet some of the clinical findings are, as already pointed out, suggestive of aphasia.

The Significance of Alexia. To my knowledge no attempt has ever been made adequately to explain the intimate association of the loss of motor speech and alexia. It is in my experience a striking fact that in by far the larger number of cases of Broca's aphasia the loss of the power to read exceeds—and usually decidedly—the loss of the power to comprehend spoken speech. If the loss of motor speech depends upon lesion of the third frontal convolution, why this remarkable association with alexia? Is it not a more reasonable explanation to assume that the lesion, whatever its situation, is one which interferes with speech comprehension—the one symptom common to all aphasics—and that the form of speech comprehension which of necessity suffers most is the comprehension of *written* speech? Comprehension of spoken speech is a faculty as old as the race, comprehension of written speech at most an acquisition of a few generations. Which faculty would we expect, therefore, to suffer most? Is it not natural that the infinitely older and basic faculty of the comprehension of spoken speech should frequently suffer less, or even little, while the power to comprehend written speech is literally swept away?

It is quite evident that in the study of aphasia general conceptions of cerebral function must ever be kept in mind, lest special reasoning lead us too far astray. It is for this reason that general procedures, such as the serial instructions of Marie, often prove so valuable. The serial instructions are a test, (1) of the general faculty of speech comprehension; (2) of the presence of apractic elements; (3) of the general intellectual integrity. Apraxia, when present in aphasia, means necessarily a lesion of great extent or a lesion involving widely diffused associations. General intellectual impairment, of course, argues for a still more extensive diffusion of the morbid condition.

The application of the theory of the diaschisis of von Monakow enables us to explain many of the vagaries of the lesions of aphasia.

How greatly the lesions vary, both in location and size, is well known; this is especially apparent in the revisions of von Monakow and of Moutier. In a measure the theory of the diaschisis enables us to reconcile these variations, and it also enables us to explain the suspension of function with subsequent recovery. It cannot, however, enable us to upbuild a function once destroyed. Thus, given a lesion of the third frontal convolution, with loss of the emissive speech function and subsequent recovery of this function, diaschisis cannot be invoked to explain the recovery of that function *as the function of the part destroyed*, namely, of the third frontal convolution. To maintain that the neighboring cortical areas have assumed the function, or that the function has been restored because the individual happened to be ambidextrous, is virtually begging the question. The obvious objections to such explanations have already been pointed out by others. However, whether the third frontal is or is not concerned in aphasia is really not an important matter. If it is, the clinical and pathological evidence should be forthcoming. All will agree, I think, that this evidence is at the present day most meagre. It is certainly significant that permanent speech disturbances do not follow lesions limited to the third frontal convolution. It is quite safe to assert that thus far not a single unequivocal case, a case free from criticism, has appeared in the literature. As Marie has pointed out, and later Pick and von Monakow, permanent disturbances of speech associated with lesion of the third frontal convolution are only present in cases in which the lesion is deeply penetrating and extensive in its distribution. Negative cases, that is, non-involvement of the third frontal convolution, on the other hand, are not at all infrequent. I have myself only recently acquired two such specimens; a third I placed on record a few years ago.

In the foregoing paragraphs I have purposely refrained from the use of terms implying special theories, such as word blindness, word deafness, transcortical, subcortical, or conduction aphasia, or paraphasia. I have thought it wiser to lay stress upon general facts such as in my judgment lend clearness to our conceptions of the speech function and react indirectly upon our conceptions of cerebral activities in general.

SCHOOL LIFE AND ITS RELATION TO THE CHILD'S DEVELOPMENT.[1]

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I suppose it is generally known that a great wave of interest has been passing over this country during the last two or three years. This wave is a movement in favor of so changing the laws of the different States that the early life of human beings should be more protected than it now is under the State laws which govern matters pertaining to child labor. A great deal of discussion has also been carried on regarding the local government of the public schools. The object, in both instances, has been to investigate whether the laws which have existed for many years are the best that can be made, and whether we, in grading our public schools, are really carrying out ideas which from a modern point of view are considered best. There is no doubt, in regard to the former proposition, that the laws governing child labor are not only fundamentally wrong from their lack of wisdom, but that even if they were right, they are so constituted that they cannot be properly enforced.

While carrying on some scientific work in connection with the development of early life, by means of the Röntgen-rays, I arrived at certain conclusions which proved to me that those who are in charge of the welfare of children are ignorant, in many ways, regarding certain facts connected with the physical condition of children. As a corollary to this, it was easy to show that many of the rules made in the past for the good of children, are with our present knowledge manifestly far from beneficial, and, in fact, are founded on ignorance of the latest scientific modern investigations. Ignorance in itself is not a crime, but when we who, as developed adults, are supposed to protect by our knowledge an earlier stage of development which is manifestly unfit to judge for and care for itself, ignorance becomes a crime if we wilfully do not make ourselves cognizant of what science is teaching us. I shall, therefore, try to point out some plain truths, such as, from a medical point of view, have come to my notice in connection with the rearing of young children, the fitting of these children to become good citizens in the future, and their being prepared, both physically and mentally, to judge of and adjudicate the great questions which must come before them in matters of legislation for their country. An educated mind without a sound body is as unfitted

[1] The Sunday Free Lecture, delivered at the Harvard Medical School, Boston, March 14, 1909.

to deal vigorously and wisely with the questions of the day, as is a good foot for walking when imprisoned in a tight boot.

Physical suffering perverts good judgment wherever it appears and whenever it is allowed to continue. It is manifestly the case at present that the young of our country are not protected by certain laws and certain rules, which are supposed to protect them during the formative period of their lives, when proper and wise protection means prevention from possible future and lasting injury to both brain and body. I shall not, here, present the reasons why I am so firmly impressed that the various laws and investigations in connection with child labor have been founded on a wrong basis, although carried on by those who are enthusiastically honest in the reforms which they are endeavoring to make. I shall merely state that the results of my investigations point toward the fundamental principle that a child's physical life should be the first factor in the problem of arranging and moulding this life which we as adults have to solve.

The question, therefore, immediately comes up as to whether there is any known means by which we can readily determine, in a large number of cases and in a limited time, what the child's physical condition as represented by its development really is. We know that we cannot judge of these conditions by height, or by weight, or by the eruption of the teeth. All these sources of evidence which for so many years we have considered to be reliable as indices to aid us in our work have one by one proved to be falacious. The tall, undeveloped child, the heavy child with weak underlying muscles and bones, and the frequent variations as the result of modern life and parentage which occur in the appearance of the teeth, are not manifestations on which we can build up a system for grading children. A much more accurate system is needed to determine when they shall be allowed to undertake certain degrees of manual labor and certain strains in athletic sports, and to give out the mental energy required for the different grades of study during their school life. It is this latter class of cases to which I wish to direct attention.

The guide to the grading of children in school, from time immemorial, has been based in great measure on the number of years since they were born; that is, chronologic age has held a position in, and has exercised an influence over, child life for so many years that it has become one of the traditions of the past, and traditions, as you well know, are difficult to set aside. My investigations with the Röntgen-rays show very conclusively that because a number of children are born in the same year it does not follow that these children, as years go on, have developed equally; on the contrary, after a few years some may be far ahead of the development of the average, and some below that average. We also know that the question of development which indicates strength and the power

of resistance to external influences is so closely connected with a child's mental and physical condition that we can no longer, with our present knowledge, keep this element in the background. If twenty boys, or twenty girls, all of whom were ten years old chronologically were pitted ten against ten, whether in football or in basket ball, a very evident discrepancy in strength, resisting power, and in endurance would become at once manifest. This difference in the individual development of these children can easily be made manifest by a Röntgen picture of the underlying framework of their bodies, which represents the basis on which their individual strength should be judged. Quite a number of these twenty children can, in this way, be shown to be perhaps a year or a year and a half in advance of what they would average when judged by their equal chronologic age; some of them would be found to be a year or a year and a half below this standard average of development. In this case, therefore, we should likely have ten of the children whose development is only that of the average chronologic age of eight and a half pitted against ten who had the average of ten and a half years of age. It is through these discrepancies that this subject has been brought to my notice most vividly, when I have seen, as often happens, children with an insufficient anatomical development brought to consult me for anemic and weakened conditions often verging on nervous prostration, which conditions have been evidently produced by the endeavor to keep these children abreast, in both school and athletics, of children of an equal age.

In the rearing of our children, therefore, we should not be influenced entirely by their chronologic age, but should study their individual development, and grade them in divisions of perhaps A, B, C, D. In this way we shall prevent a child, in its daily life, from having its tissues, whether of brain or body, overwhelmed by unnecessarily advanced surroundings.

A possible index by which we can intelligently determine what the surroundings of a special child shall be, can, so far as my studies have carried me, be learned from an investigation of the changes in the development of the joints from birth to adolescence. In one or two seconds, without the slightest danger to the child, a Röntgen picture can be taken of an especial joint.

In the course of my investigations of some hundreds of cases, in which I have studied the joints in normally developed children, it has been shown that it is not necessary, in most cases, to take a picture of all the joints. It has also been shown that in healthy individuals, during the formative period of early life, the bones of the wrist in a very large number of cases are so comparatively uniform in their development in comparision with the other joints, that they can be taken as an index of the development of the special child. It is of great significance that this anatomical index will be

of marked value in the determination of the questions connected with child labor if we remember how children, so far as labor is concerned, are graded, mainly on a chronologic basis. The law may say that a child shall not enter a mill until it is twelve years old. The parents wish to have that child, for pecuniary reasons, enter a mill; they therefore swear falsely to the child's age. The child, again, may be tall for its age, and, although it is only eleven years old, may look as if it were twelve. Large numbers of false birth certificates also exist in this country, so that a chronologic law can easily be avoided. This is proved by the number of children who, though manifestly unfitted for the work which is given to them in the mills, are found in the mills and are honestly supposed to be within the requirements of the law, although they not only may not really be of the chronologic age required by the law, but even if they are of that age, may not be in any way fitted to perform the work allotted to them.

This same reason can be applied to the grading of children in the schools. Their age, in most cases, at least, is honestly given, but it is readily seen that if they are graded chronologically, the same conditions, both physically and mentally, which are good and proper for some of these children, are manifestly bad for others. This same reasoning holds good in regard to the question of kindergarten. Much more definite decisions should be made on the time when an individual child should begin with its kindergarten, or should be advanced to the school grades, than is now the case. It is an argument based on false conclusions to say that a bright and precocious child should be advanced in its school grade until it meets with brains which are capable of safely undertaking mental study beyond that met with in the grade of the chronologically younger children, where the bright child is at first classed. These older brains, being more developed, are not harmed by the physical demands which become strains to the bright brain of the younger child. Precocious children should, from this reasoning, not be advanced to a grade in the school which represents a higher type of physical development, but should be employed in mastering the numerous school occupations of the present day which can be acquired out of doors in connection with physical exercises. These bright brains should not be allowed to be advanced to a school grade of higher physical development until their physical development corresponds to their mental power.

This rule of making the development of a child correspond to its mental condition works in both ways. If a child has a bright, in one sense precocious brain, beyond its chronologic age, but at the same time has an anatomical development corresponding to the development of its brain, it can then be advanced into a higher grade chronologically without harm.

There is no doubt that the principle of grading children by divisions of development determined by some anatomical index, which can be obtained by means of the Röntgen rays, will be the grading of the future. We should consider, also, that the question of exactness plays a very important role when we are determining what this developmental index should be. The Röntgen-rays in their picture tell the truth, and by means of this truth we have a powerful weapon with which we can oppose the deliberate falsehoods of parents, guardians, officials, and of those who, for commercial reasons and through excessive greed, wish to obtain labor at the low cost at which it can be bought when children are employed.

Of equal importance with the labor problem, in its direct baneful influence on the child's life, is the unwise and almost criminal desire of parents, guardians, and school teachers, to advance the child in school as fast as its brain, as a piece of machinery for acquiring knowledge, will allow, without considering that any piece of machinery, no matter how good, may be overstrained and rendered unfit for use when its function is pressed beyond what it is intended to carry. Overtaxed physical conditions may result in weakened brain power. A bright brain is not necessarily strong in its functions. Overtaxed mental capacity may result in physical weakness. It is, therefore, only a common sense view of the subject, and one which I hope every one will try to understand and try to carry out in his or her children's lives, namely, to insist that the grading of children in school shall be based upon an equality of physical and cerebral strength. This simply means equality in development. This, again, simply means a determination by means of an anatomical index of what the special development is. This, however, does not mean that a highly developed physical condition, with a corresponding mental condition, should interfere with the line of action which I have just laid down. The dull brains with a highly developed physical condition can be left to take care of themselves. The brain in this case is taking its rest, will not exert itself, and will rarely be harmed. These children can be allowed to fall back in their classes no matter what their physical development is, and educators can place them where they wish.

A great deal of harm comes to children through their parents and guardians supposing that, because their neighbors' children are allowed to take up, according to their chronologic age, certain traditional or modern amusements or studies, according to the community in which they live, their children also are equal to standing the same more or less necessary strain. For some children the continual round of dancing school, music lessons, children's theaters, and other outcomes of civilization involving exaggerated physical and mental activities, are clearly pernicious, unless under very careful supervision, a supervision which should be adapted to the needs and capabilities of the especial child.

FIG. 1.—The wrist of a girl, aged six months, in which only two small bones are seen to have appeared in the cartilage of the wrist.

FIG. 2.—The wrist of a girl, aged two and three-fourths years, in which there are three carpal bones—thus representing a higher degree of development than is seen in Fig. 1.

To those who have conscientiously studied children, not merely in their diseases, but as they are seen in their homes, in their nurseries, in their play, and in the many phases of the routine of the various out-of-school occupations, in fact, in all the many means of wear and tear which are brought to bear on the modern child, it is an easy matter rightly to understand the bad results which are so often attributed to the school, and often attributed unfairly. It is the combination of all which is required of children out of school, as well as in school, which brings about the last drop in the bucket. If we could begin all over again, and grade our children on the basis of an equal mental and physical development, half of the disturbances of early life which come under the notice of the physician would cease to be in evidence.

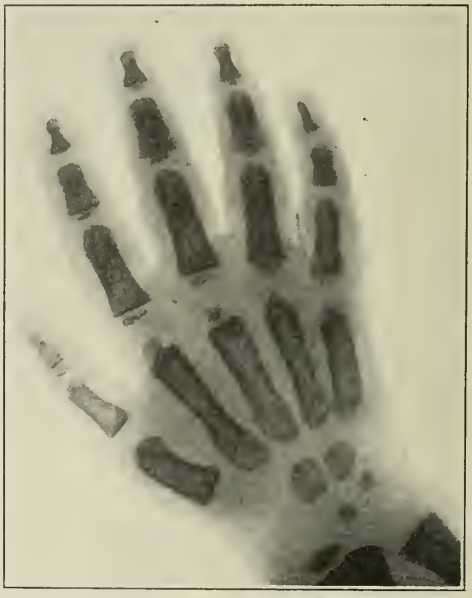

FIG. 3.—The wrist of a girl, also aged two and three-fourths years, showing with its four carpal bones a still higher degree of development than does Fig. 2.

The tendency of what our educators call the most advanced education is to harm the well-being of individuals who must eventually become citizens and govern other human beings. Numerous instances can be quoted as to how misguided are the ideas of certain educators, even though they be prominent as educators. We can hardly believe that a body of so-called educators, meeting together to arrange their schedule for the teaching of children through graded years, should gravely debate among themselves what studies each of them would prefer to teach in such and such a year, rather than at what stage of development, both in mind and

in body, it is best for the child to begin such studies; yet this has lately happened.

The parents are often much more at fault than are the teachers. The parents, as a rule, wish to see their children advanced as rapidly as possible, and this phenomenon, which may be called greed of mind, corresponds to what in child labor we have called greed for money. The school teachers over and over again protest against the child's being forced to do work beyond its physical capacity,

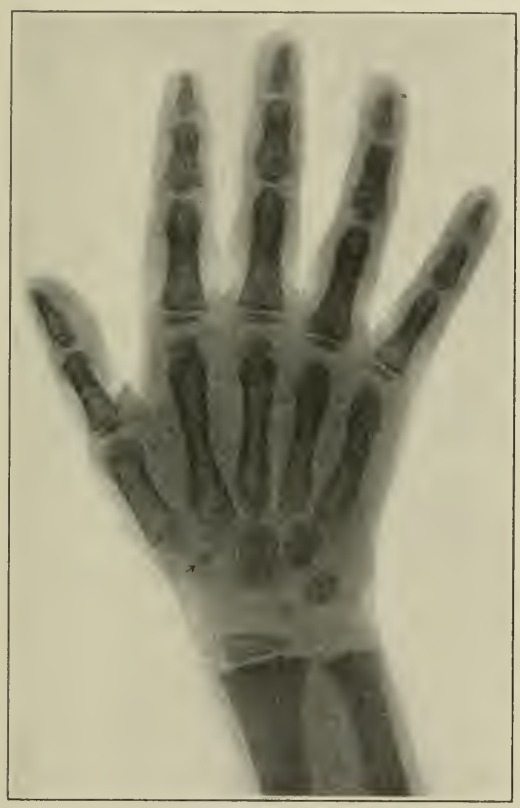

FIG. 4.—The wrist of a girl, aged three and a half years, with an even greater degree of development, as represented by five carpal bones.

but rarely can induce the parents to take a more common sense point of view until the child's physician tells them that if this principle is carried out much longer the child may become a cripple for life.

It is very evident, from what I have said, that the school life of a child has a very strong and important influence in relation to its development. I ask, therefore, those who are interested in the welfare of children to prevent this unjustifiable grading

of children in their school life. Overtaxing the functions of the brain will result in mental fatigue, and this, in its turn, will affect the entire nervous organization of the child and retard its physical development. This lowering of a child's nervous force and interfering with its physical development renders the child more vulnerable to disease. so that in order to reduce the high mortality which

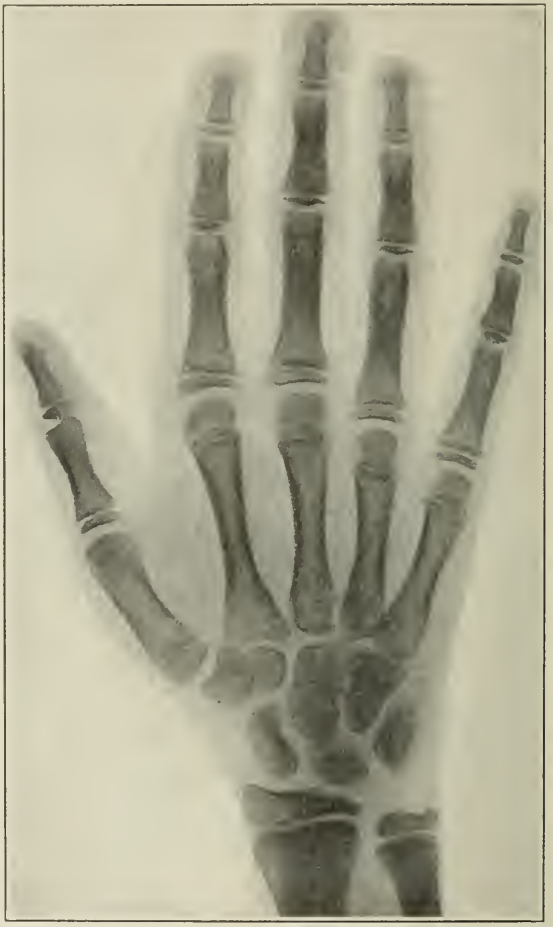

FIG. 5.—The development and massing of the bones of the wrist in a girl, aged eight and one-fourth years.

occurs in children, we should see to it that by dealing with this developmental idea the child's power of resistance is increased, and, becoming in this way less vulnerable to disease, it will not only have its life preserved where otherwise it might be lost, but in its future life will enjoy a vigorous and healthful existence.

The remedy for the evils which at present surround the school

life of young human beings is that the professed educators, the school teachers, the parents, the guardians, and those physicians who have devoted themselves to the study of early life should act in accord, each giving to the other the especial ideas which have come from the results of their experience and from the study in certain lines in which they have become proficient. As a prerequisite, however, for all changes which will in the future be brought to bear in connection with early life, it should be recognized that

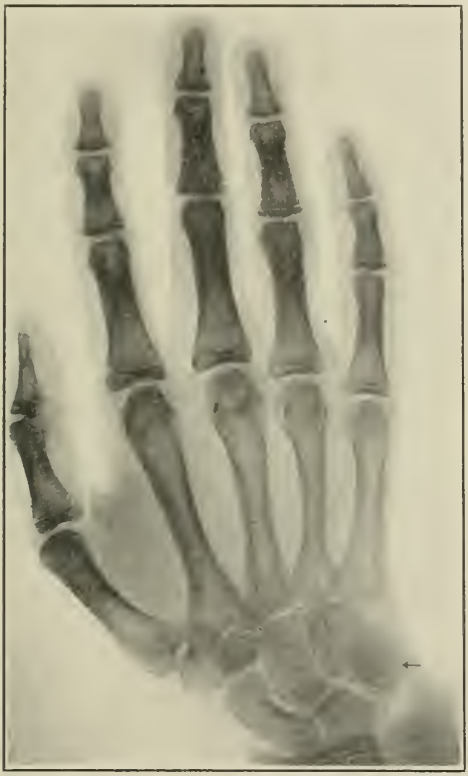

FIG. 6.—The greatly advanced development in number, size, and massing of the bones of the wrist in a boy, aged thirteen and one-half years.

chronologic age should be done away with as a basis on which to grade children in schools, in sports, and in the various degrees of work in which they are employed. In fact, chronologic age should be retained only for matters pertaining to the legal profession and in questions of inheritance.

In order that one may understand what I mean by an anatomical index, I append pictures (Figs. 1 to 6), which, in their progression from cartilage to bone, represent different degrees of strength,

owing to the greater strength of bone over cartilage. Starting from an almost entire lack of bone in the first year of life, one will notice that these bones increase in number and in size until finally the almost fully developed wrist is attained at about the age of fourteen to fifteen years. This assumes that the quality of the bone tissue is also normal. This also necessitates a qualitative study in the future of bone tissue as well as bone size.

It is thus seen that there is a complete series of stages of development of the wrist from infancy to adolescence, showing in this way different stages of qualitative and quantitative strength, each of which should be equal to the endurance called for by certain kinds of mental fatigue as well as manual labor.

COMPRESSION OF THE PULMONARY VEINS, THE PRESSURE FACTOR IN THE ETIOLOGY OF CARDIAC HYDROTHORAX.[1]

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The hydrothorax that sometimes accompanies heart disease has been recognized for many years. Why it occurs in some cases and not in others is not clear, for it is certain, as both Flint and Da Costa[2] long since remarked, that the phenomenon bears no constant relation to the extent of the cardiac disease. "Not only are these lesions (cardiac) marked in cases in which dropsy has not occurred, but dropsy occurs in other cases in which the lesions are comparatively slight" (Flint[3]).

In seeking an adequate explanation for the occurrence of these effusions we have concerned ourselves chiefly with the effects, the mechanical effects, arising from pressure exerted by the dilated heart. We recognize fully that before an effusion can occur the presence of factors other than pressure may be necessary, for

[1] Read at a meeting of the Pathological Society of Philadelphia, May 27, 1909.

[2] Medical Diagnosis, eighth edition, p. 422.

[3] Diseases of the Heart, second edition, 1870, p. 37.

example, toxic, vasomotor, bacterial, or metabolic influences, or a hydremic condition of the blood. Krehl[4] states in this connection: "Certain patients suffering from heart disease in the state of broken compensation show a watery condition of the blood, both the specific gravity and the proportion of proteids in the blood being diminished (Grawitz). A weakness of the right ventricle is especially apt to give rise to such a thing in the blood. . . . This hydremia occurs in a comparatively small proportion of all cases of broken compensation; but where it does occur it is quite usual for it to disappear with any improvement in the circulation."

Any one of those agents, however, which affect the composition of the blood will fail to account for the fact that these effusions have a marked preference for one or the other side of the chest, usually the right. That pressure alone will cause the transudate is improbable, but it is certainly a factor of the greatest importance. Another point is that the pressure must be from dilatation, as it has long been recognized that hypertrophy of the heart by itself is incapable of producing an effusion, and that dilatation, with or without valvular lesions, is an essential feature.

Recognizing that dilatation of the heart was necessary, most observers contented themselves with the explanation that a transudation resulted from ven us stasis, either alone or in association with impoverishment of the blood. In recent years, however, the azygos-vein theory has been much in favor, largely as a result of papers published by Steele[5] and Stengel.[6] Their explanation of these effusions, and particularly of the right-sided preponderance, is that the dilated right heart by extension upward exerts pressure on the root of the right lung and thus indirectly pinches the azygos major vein as it curves over the root of the right lung to enter the superior vena cava. Baccelli,[7] in 1863, first called attention to this possibility, although his explanation differed somewhat from that of Steele and Stengel. His idea was that the enlarged heart, by dragging the superior vena cava downward, carried with it the vena azygos major, thus drawing it tightly around the root of the lung and causing it to be compressed.

What has commended this theory to many is the fact that the vena azygos major receives as tributaries the intercostal veins of the greater portion of the right chest, vessels which drain the major portion of the right costal pleura. The natural assumption has been that with compression of this vein in its upper part there would result a venous stasis on the right side which would lead to a transudation. The theory has gained additional strength from

[4] Clinical Pathology, American edition, 1905, p. 176.

[5] University Medical Magazine, 1897, Journal of the American Medical Association, October 1, 1904.

[6] University of Pennsylvania Medical Bulletin, 1901.

[7] Quoted by Steele, Jour. Amer. Med. Assoc., October 1, 1904.

the fact that these effusions are so frequently right-sided, or, when bilateral, greater on the right side. When the condition was bilateral the smaller, left-sided effusion was explained on the basis that the vena azygos minor, which drains less of the left chest than does the major of the right, and empties into the latter at the eighth or ninth thoracic vertebra, would become affected only secondarily and to a slighter extent.

Every concrete theory hitherto advanced to explain the condition has been based on the assumption that the fluid is derived from the parietal pleura; and, of the vessels of this part of the membrane, the azygos veins have been held by most writers to be the source of the fluid. To this explanation five objections can be raised: (1) Only about two-thirds of the parietal membrane is drained by the azygos veins; (2) the collateral anastomoses of the azygos veins are so free and so numerous that, in the event of pressure, competent by-paths would soon be established and carry away any excess of fluid in the azygos radicles; (3) the vena azygos minor, emptying into the major, is subject to the same influences as the latter, and therefore the effusion should always be bilateral; (4) it is anatomically impossible for the heart, either directly or indirectly, to exert pressure upon the azygos major vein; (5) it does not explain purely left-sided effusions.

1. First, as to the azygos veins draining only about two-thirds of the parietal pleura. Like all serous membranes, the pleura consists of two portions, a parietal and a visceral, and the circulation in the two differs greatly, each receiving and discharging blood in vessels which belong to the structures with which the membrane is in contact. An example of this principle is afforded by the pericardium, of which the vessels of the parietal layer are derived from the thoracic aorta and the internal mammary, while those of the visceral layer or epicardium are branches of the coronaries. The pleura has a similar arrangement, but for the present the vessels of only the parietal layer will be considered. The arteries are derived from the superior and aortic intercostals, the internal mammary, the mediastinal, the œsophageal, the bronchial, and the phrenic, and the return circulation is through corresponding veins.

The most important thing to have clear in this connection is the vessels into which these veins drain. The intercostals vary somewhat at their vertebral ends, but the average arrangement is for the first intercostal on both right and left sides to empty into the innominate, while the second, third, and fourth on the right side form a trunk which opens into the vena azygos major, and the remainder on this side open separately into the same vessel. On the left side the second, third, and some times the fourth form a trunk, the superior intercostal vein, which opens above into the innominate and is connected with the left upper azygos vein. The

intercostals from the fifth to the eighth on the left side form the left upper azygos and open into the azygos major or minor, while the remaining intercostals open individually into the azygos minor. At their sternal ends the upper nine or ten intercostals of both sides open freely into the internal mammary, or its musculophrenic tributary, the lower two or three having no such termination.

Of the remaining veins of the parietal pleura, the internal mammaries empty into the innominate, the mediastinal into the same vessel, the œsophageal into the azygos and also into the inferior thyroid and the gastric, the bronchial into the azygos, the inferior phrenic into the inferior vena cava, and the superior phrenic into the left innominate. The cervical pleura drains into the innominate, the posterolateral part of the right costal pleura into the azygos major, the upper third of the posterolateral part of the left costal pleura into the innominate above and the azygos minor below, and the lower two-thirds into the azygos minor. The anterior part of the costal pleura empties its blood into the internal mammaries, the mediastinal pleura into vessels which eventually reach the innominate and the portal, and the diaphragmatic pleura into veins which are tributary to the innominate and the inferior vena cava.

Analysis of this summary shows that the venous drainage of the parietal pleura is into widely separated vessels, and consists of veins which open into the azygos major and minor, the innominate, the inferior thyroid, the gastric, and the inferior vena cava. The deduction to be drawn from this is that while the azygos veins are of importance in carrying away the blood from the parietal pleura, only a portion of the membrane is drained by this system, perhaps not more than two-thirds.

2. Second, as to the numerous and wide anastomoses of the azygos veins. In order to render clear this portion of the discussion, it is perhaps advisable to give a brief resume of the tributaries and anastomoses of the azygos veins. The tributaries of the azygos major are the lumbar, the right intercostals with the exception of the first, the œsophageal, the posterior mediastinal, the venous plexus around the thoracic aorta, the posterior pericardiac, the bronchial, and the azygos minor. In connection with these vessels it is important to know how extensive and how practicable are their peripheral anastomoses. The azygos major begins just below the diaphragm, and at this point anastomoses with the right ascending lumbar vein, which connects it with the four right lumbar veins and, through the lowest of these, with the iliolumbar or the common iliac vein. The azygos minor has the same connections on the left side, and by these anastomoses a ready side track into the inferior vena cava is established.

As regards the intercostals, "In the middle portion of their course the upper six or seven veins give off branches, the costo-axillary

veins, which ascend toward the axilla and open into either the long thoracic or the thoracico-epigastric vein and so into the axillary, and, as it approaches the vertebral column behind, each vein receives a dorsal branch which accompanies the spinal branch of the intercostal artery and returns the blood from the skin and muscles of the back and also from the spinal column and its contents, this drainage being by means of a spinal branch which connects with the intervertebral veins."[8] "Laterally, at each intervertebral foramen the internal (spinal) plexuses send branches out from the spinal canal along the nerve trunks, and by means of these intervertebral veins, which have the form of plexuses at their origin and receive communicating branches from the external vertebral plexuses and from the veins of the spinal cord, the internal plexuses pour their blood into the vertebral, intercostal, lumbar, and lateral sacral veins, the connection with the intercostals being through their rami spinales."[9] In addition to these anastomoses the upper nine or ten intercostals open anteriorly into the musculophrenic or internal mammary veins. It is evident, therefore, that obstruction to the flow of blood through the intercostals would result in the blood seeking and finding other courses, which are many and wide—the axillary, the vertebral, the lumbar and the lateral sacral.

The œsophageal veins anastomose with the inferior thyroid veins above and the gastric below. There are thus afforded paths into the innominate and the superior vena cava by way of the inferior thyroids and into the portal by way of the gastric. The posterior mediastinal veins open into the left innominate, and the destination of the plexus around the aorta is probably into the azygos and œsophageal veins. The posterior pericardiac veins anastomose freely with the anterior pericardiac, which open into the internal mammary, and through these a path is established into the left innominate or into the superior vena cava. The bronchial veins anatomose in the lung with some of the radicles of the pulmonary vein and thereby afford a wide open channel for any dammed back blood. The vena azygos minor (vena hemiazygos) begins in the left upper abdomen and enters the thorax through the left crus of the diaphragm. Thence it ascends at the left side of the thoracic aorta and at the level of the eighth or ninth thoracic vertebra crosses to the left beneath the thoracic duct and thoracic aorta and empties into the vena azygos major. The tributaries of this vessel are the ascending lumbar vein, the left lower four or five intercostals, the œsophageal, the posterior mediastinal, and occasionally the left upper azygos. The peripheral anastomoses of these vessels are, with the exception of the left upper azygos, the same as have been described in connection with the azygos major.

[8] Piersol's Human Anatomy, p. 896.

[9] Ibid., p. 898.

The left upper azygos vein (vena azygos minor superior, or accessory hemiazygos) begins at the second intercostal space on the left side and descends to the left of the middle line. At about the eighth thoracic vertebra it crosses to the right posterior to the aorta and œsophagus, to empty into the azygos major. Sometimes it empties into the vena azygos minor as the latter curves toward the right, and even when it empties into the azygos major it is frequently connected directly with the azygos minor. Its tributaries are the upper seven or eight intercostal veins, the left posterior bronchial veins, and a twig which connects it with the left innominate. The potentialities of these have been indicated in the description of the azygos major.

It would seem from this summary that the possibilities of a wide and ready collateral circulation through many by-paths, some leading into the superior and some into the inferior vena cava, are so great that it is almost unthinkable that any degree of obstruction could be sufficient to cause leakage into the pleural sac. In this connection it should be remembered that the pleura and its vessels are constantly subjected to a suction or negative pressure equivalent to 6 mm. of mercury, and must, therefore, be stronger and better equipped to resist pressure than any others in the body. Were this not so the pleural cavities of everyone presenting favorable blood conditions would fill with fluid until the pressure in the sac would be equal to the pressure within the lung.

In connection with the above resume of the azygos anastomoses it is interesting to note that Broadbent,[10] in referring to the pressure of aneurysms on the venous trunks, states that "there may be pressure on the azygos vein which passes up behind the root of the lung to open into the superior vena cava just before it enters the pericardium. This does not appear to give rise to any characteristic physical signs or symptoms, probably on account of the free anastomosis with the lumbar veins, which enables the blood to return without difficulty via the iliac veins and the inferior vena cava." This anastomosis mentioned by Broadbent is obviously but one of many.

3. Third, as to the vena azygos minor being subject to the same influences as the major. It will be remembered that the former vein, after receiving the left upper azygos, the lumbar, and the lower intercostal veins, crosses the vertebral column and empties into the azygos major. Being a tributary to the latter and the largest branch of the azygos tree, it must of necessity be subjected to the same pressure influences. Granting that obstruction at the azygos arch is responsible for these right-sided transudates, it is difficult to understand why the effusion should ever be limited to one side, especially the right, since stasis would probably first be manifest at the

[10] Heart Disease, fourth edition, p. 445.

greatest distance from the point of pressure, that is, in the radicles of the vena azygos minor.

4. Fourth, as to the possibility of the heart exerting pressure upon the azygos major vein. In order to discuss this intelligently, a review of the course and relations of the vein is essential. The vena azygos major (vena azygos) begins in the right upper abdomen and passes into the thorax either through the aortic opening in the diaphragm or between the median and intermediate portions of the right crus. It ascends in the posterior mediastinum to the right of the median line, having behind it the right intercostal arteries and the bodies of the thoracic vertebræ. To its left lie the aorta, the œsophagus, and the thoracic duct. On reaching the level of the seventh thoracic vertebra it inclines slightly to the right, and at the level of the fifth or fourth bends forward and passes between the mesial surface of the right lung and the right aspect of the trachea at or just above the obtuse angle (155 degrees) at which the right bronchus leaves the trachea. Either at or posterior to the plane of the anterior surface of the trachea it enters the superior vena cava just above the point at which the pericardium leaves the latter vessel. A number of measurements taken in the dissecting room of the University of Pennsylvania showed this point of entrance to be about 6 cm. above the entrance of the superior cava into the right auricle. The terminal portion of the vessel, as it arches over the right bronchus, is called the "azygos arch." Valves, usually to the number of four, are present in the vessel, but they are frequently unpaired and usually incompetent. In 22 per cent. the valves are absent.[11]

To justify the statement that the heart cannot press on the azygos major vein it is necessary to review briefly the anatomical relations at the roots of the lungs. These roots are made up of the bronchus, the pulmonary and bronchial vessels, nerves, lymphatics, and areolar tissue. The superior vena cava descends anterior to the root of the right lung to terminate in the upper portion of the right auricle, and this upper part of the auricle lies in an anterior relation to the root, an important fact, of which use will be made later. After leaving the vertebral column to course forward the azygos major passes in an arciform manner over the right pulmonary root to empty into the superior vena cava about 6 cm. above the termination of the latter in the right auricle. A dilated right heart, to exert any pressure on the azygos arch, would necessarily displace upward and backward the pulmonary veins, the bronchus, the pulmonary arteries, and all the minor elements in the lung root. To realize that this is an impossibility one has but to study the actual preparation made in a body in which the viscera have been hardened in situ, and to take into consideration not only the intrinsic

[11] Gruber, quoted by McMurrich in Piersol's Human Anatomy, p. 983.

strength and resistance of these structures, but also their firmness of attachment. The ligamentum latum pulmonis alone, a firm reflection of the pleura connecting the lung root with the diaphragm, would be a potent factor in restraining any upward excursion of the bronchus and its associated structures. Study of the accom-

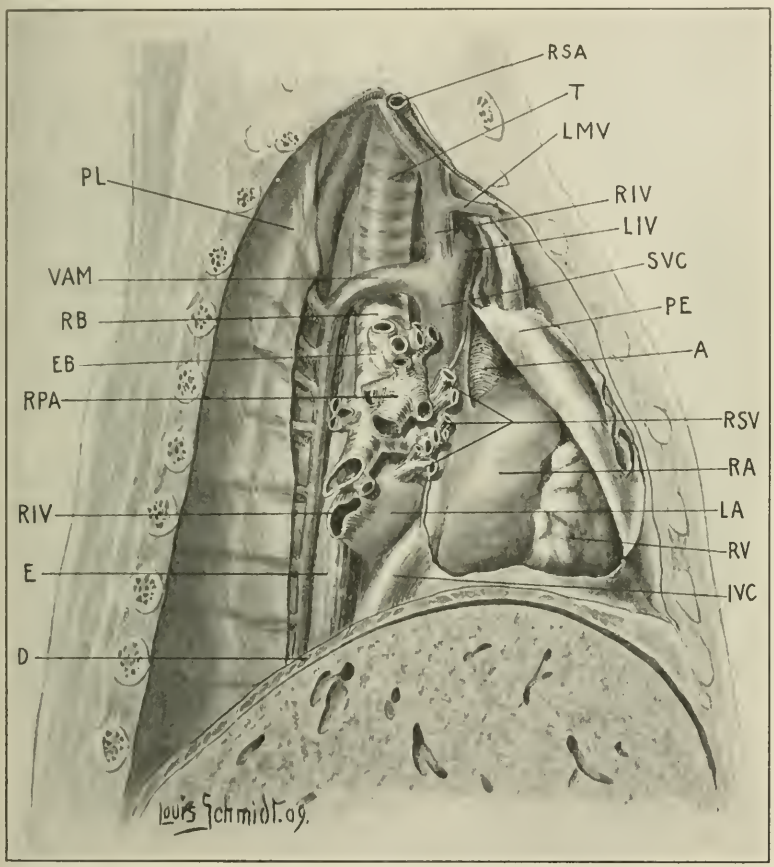

FIG. 1.—View from the right of the mediastinal contents and the root of the right lung. *D*, diaphragm; *E*, œsophagus; *RIV*, right inferior pulmonary vein; *RPA*, right pulmonary artery; *EB*, eparterial bronchus; *VAM*, vena azygos major; *PL*, pleura; *RSA*, right subclavian artery; *T*, trachea; *LMV* (should be *RMV* in the illustration), right internal mammary vein; *RIV*, right innominate vein; *LIV*, left innominate vein; *SVC*, superior vena cava; *PE*, pericardium; *A*, aorta; *RSV*, right superior pulmonary vein; *RA*, right auricle; *LA*, left auricle; *RV*, right ventricle; *IVC*, inferior vena cava.

panying figures (Figs. 1 and 2) should convince one that the pressure claimed to be possible is out of the question. Theoretically there might be some direct pressure exerted by the heart upon the ascending part of the azygos, but cross-sections of the thorax (Fig. 3) show this to be impossible. The vein is so completely and

well protected by the œsophagus and the aorta that such a contingency need not be considered.

Incidentally it might be stated that autopsy reports in which the assertion is made that the azygos major vein is dilated are, as a

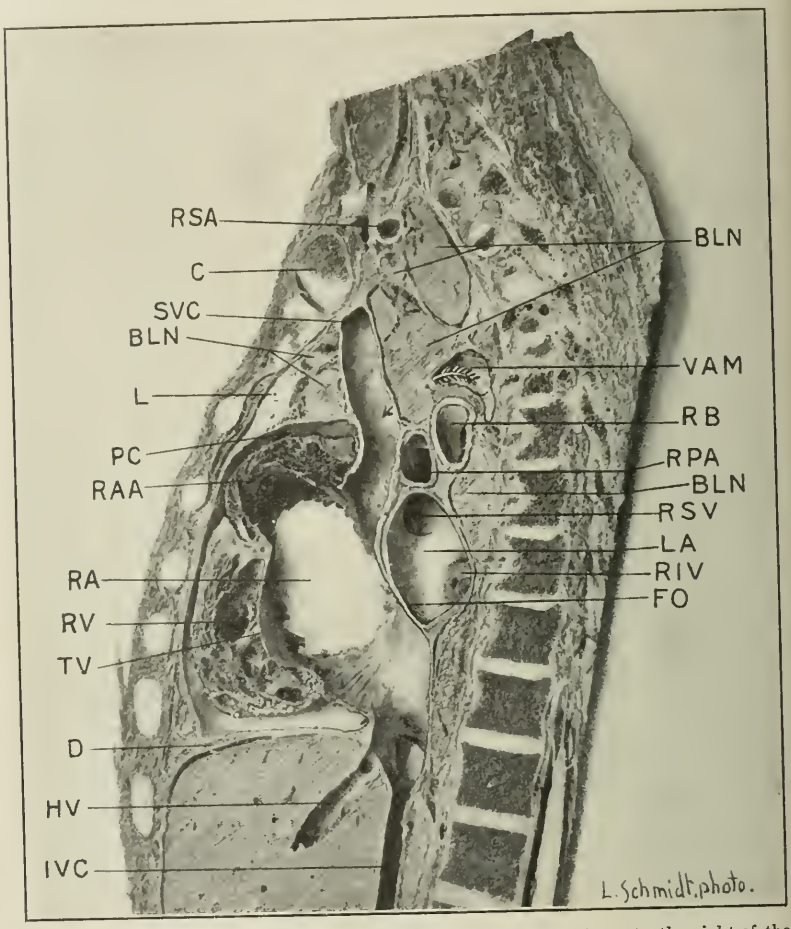

Fig. 2.—Anteroposterior section through the thorax in a plane 2 cm. to the right of the sternum. *IVC*, inferior vena cava; *HV*, hepatic vein; *D*, diaphragm; *TV*, tricuspid valve leaflet; *RV*, right ventricle; *RA*, rignt auricle; *RAA*, right auricular appendix; *PC*, pericardial chamber; *L*, lung; *BLN*, bronchial lymph nodes; *SVC*, superior vena cava; *C*, clavicle; *RSA*, right subclavian artery; *VAM*, vena azygos major; *RB*, right bronchus; *RPA*, right pulmonary artery; *RSV*, right superior pulmonary vein; *LA*, left auricle; *RIV*, right inferior pulmonary vein: *FO*, fossa ovalis.

rule, untrustworthy. One writer (Steele[12]) states that he found this vein dilated "to the size of a goose quill." When one realizes that the normal diameter of this vessel is about 1 cm., it can readily

[12] Jour. Amer. Med. Assoc., October 1, 1904.

be seen that such reports are untrustworthy. The average impression is that the azygos major is about the size of a radial artery, and many have been the expressions of amazement when our preparations have been shown to friends and the fact demonstrated that this vessel has at its upper end a normal caliber equal to that of a carotid or even a subclavian artery.

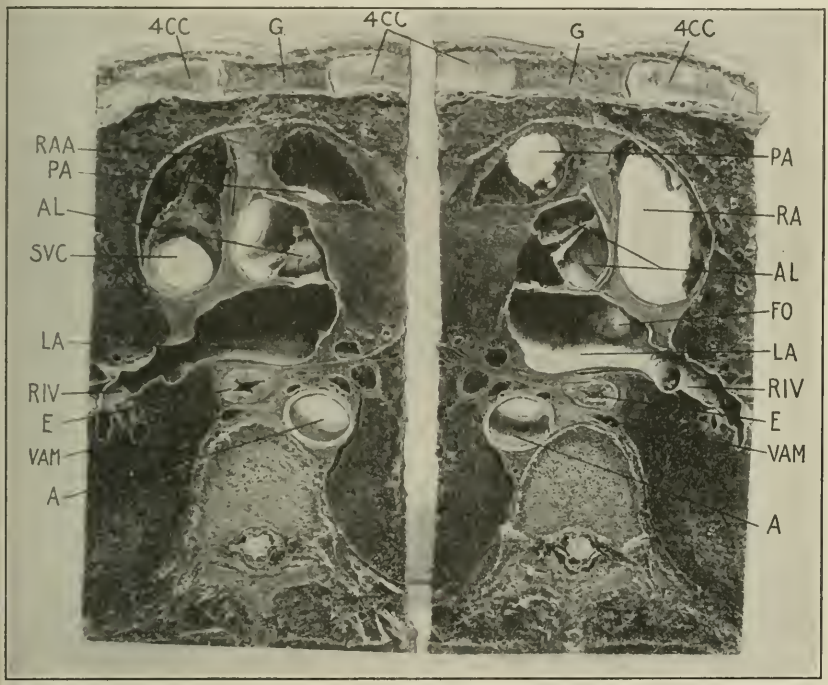

FIG. 3.—Lower (on the left) and upper (on the right) views of a transverse section through the mediastinal portion of the thorax at the level of the fourth costal cartilage. *A*, aorta; *VAM*, vena azygos major; *E*, œsophagus; *RIV*, right inferior pulmonary vein; *LA*, left auricle; *SVC*, superior vena cava; *AL*, aortic leaflets; *PA*, pulmonary artery; *RAA*, right auricular appendix; *4CC*, fourth costal cartilage; *G*, gladiolus; *RA*, right auricle; *FO*, fossa ovalis.

5. Fifth, as to the inability satisfactorily to explain why the effusion is, in a not inconsiderable number of cases, entirely confined to or much greater on the left side. Thus, of 108 cases recorded by Steele and Lord,[13] no less than 30 of them (27.7 per cent.) were left-sided, either entirely or to a great extent. Steele,[14] in his second paper, advanced the theory that this might be explained by the dilatation of the heart being greater on the left side than on the right. "In 11 cases of effusion confined to the left pleura, or greater on that side, the left heart, and especially the left ventricle,

[13] Osler's Modern Medicine, vol. iii.

[14] Journal of the American Medical Association, October 1, 1904.

was especially mentioned as enlarged in 9 cases, that is, in four-fifths of the cases of left-sided effusion." He admits, however, that it is difficult to explain these cases on this theory, as there is no large venous trunk analogous to the vena azygos major to be compressed. He also mentions Rosenbach's explanation, namely, that the pressure exerted by the enlarged left ventricle on the left lower lobe produces atelectasis. As a result of long-continued congestion produced by collapse of the lung there arises a low grade inflammation of the pleura, and finally, an effusion forms in the pleural sac which is partly transudate and partly inflammatory. Steele's comment on this is that his own "list of cases gives nothing to confirm or deny the theory of Rosenbach. I can only call attention to the fact that left-sided cardiac dilatation seems to bear some relation to left-sided pleural effusions."

Authors' Explanation. The explanation which we wish to offer to account for these effusions is as follows: The fluid comes not from the parietal, but from the visceral pleura, and the outpouring is caused, so far as the pressure factor is concerned, by dilated portions of the heart pressing on and partly occluding the pulmonary veins.

Two sets of bloodvessels enter the lungs, the bronchial arteries and the pulmonary arteries. The former are nutritional in function and are derived from the aorta or from the first aortic intercostal. They enter the hilum of the lung and follow the posterior surface of the bronchi, some of their terminal branches reaching and supplying the pleura. The pulmonary arteries, in addition to carrying venous blood to the lungs, send twigs to the pleura. In this membrane there is a capillary anastomosis between the terminal branches of the bronchial and pulmonary arteries, on the one hand, and the venous radicles which are tributary, not to the bronchial, but to the pulmonary veins, on the other. This has been shown by Miller,[15] who says: "The capillary network into which the bronchial artery breaks up in the pleura gives rise to radicles which join the pulmonary vein." He also states: "I have failed to demonstrate by the use of granular injection masses any anastomosis between the bronchial and pulmonary arteries. On the other hand, by using injection masses which flow freely I have injected both sets of vessels (pulmonary artery and vein), but only by a backward flow (from the bronchial artery) of the injecting mass through the capillary network." It is evident, therefore, that the venous blood from the visceral pleura is poured into the pulmonary veins, and, as a corollary, that any obstruction to the flow through this vein, if of sufficient power and duration and if accompanied by whatever condition of the blood essential to transudation, would produce leakage through the visceral pleura into the pleural sac.

[15] American Journal of Anatomy, vii, 404, 405.

Points in favor of our theory are that it explains equally well right-sided, left-sided, and bilateral collections of fluid, and also accounts for certain intrapulmonary conditions found clinically and postmortem in association with hydrothorax. Starting with the assumption that the transudate comes from the visceral pleura as a result of pressure on the pulmonary veins, a study of the anatomical relations between the heart and the roots of the lungs illumines the discrepancies which have long been noted. Anatomically the relations and pressure possibilities differ widely on the two sides.

The Anatomy of Right-sided Effusions. These are due to pressure exerted by a dilated right auricle upon the right pulmonary veins and upon the right end of the left auricle. That a dilated right auricle does press upon the right pulmonary veins is shown to be not only possible but inevitable by a study of the mutual relations of the right auricle and the right pulmonary root. It is shown with special clarity if the thoracic contents are hardened in position before the chest is opened. Studies under these conditions were made in three ways, by anteroposterior sections, a valuable method of studying thoracic relations which has been too much neglected, by transverse sections, and by lateral dissections of the mediastinum after removal of the lungs. The following facts were definitely brought out: Of the four chambers of the heart, three appear on the anterior surface and one on the posterior. The anterior cavities are, from right to left, the right auricle (the appendix mainly), the right ventricle, and the left ventricle, and all are plainly visible on anterior inspection. The main cavity or atrium of the left auricle is entirely a posterior structure (Figs. 2, 3, and 4), being invisible from the front, and it comes in relation anteriorly with the three other chambers as well as with the first portion of the aortic arch. Posteriorly it meets firm resistance and is separated from the bodies of the thoracic vertebræ by the contents of the posterior mediastinum, particularly the descending aorta and the œsophagus (Fig. 3). Above it is bounded throughout its entire length by the right pulmonary artery (Figs. 2 and 4), above which is the aortic arch. In front of its right-hand end, into which the right pulmonary veins open, are the right auricle and a little of the superior vena cava (Fig. 2), and in the lower portion of the interauricular wall is the thin fossa ovalis (Figs. 2 and 3), remaining from the foramen ovale.

If the right auricle were to become dilated it could expand forward but slightly, owing to the presence of the chest wall; it could not advance to the left on account of the right ventricle being there; below are the diaphragm and liver; above are the superior vena cava and an overlapping wedge of lung tissue. The path of least resistance is to the right, upward and backward, and in so expanding there would be inevitable compression of the structures which lie

behind the right portion of the auricle. These are the right end of the left auricle and the root of the right lung (Figs. 1, 2, and 3). Of the component elements of the latter the most anterior are the pulmonary veins and behind these are the bronchi. The lower portion of the right end of the left auricle extends somewhat below the bronchus, and is bounded posteriorly by the œsophagus and anteriorly by the right auricle. In case, therefore, the latter expands, the two parts of the circulatory system which cannot escape compression are the right pulmonary veins and the right end of the left auricle, compression of the latter being facilitated by the thin fossa ovalis between the two auricles. The result of such compression would be stagnation in the right pulmonary veins and consequent leakage, on the outside, from the visceral pleura, and on the inside, into the lungs, the latter a point to be taken up later.

The Anatomy of Left-sided Effusions. The left auricle is unfortunately named, except in that the name indicates its physiological position. It is the posterior auricle, forming as it does the back surface of the heart, being the only portion of the heart to touch the posterior body wall and lying behind the three other cavities of the heart. It is a slightly flattened, transversely placed cylinder with tributaries entering at both ends (Figs. 2 and 4). From its left upper anterior portion arises its auricular appendix (Fig. 4), which passes forward and curls around the left anterolateral aspect of the root of the pulmonary artery (Fig. 5). Posteriorly the appendix rests against the left upper pulmonary vein and anteriorly it comes close to the chest wall, from which it is separated only by pericardium and pleura and a small thin wedge of lung. Above, outside the pericardium, is the pulmonary artery, below is the left ventricle, and the only direction in which free expansion is possible is toward the left (Fig. 5). The left auricle is so hedged in on all sides that dilatation would first affect the appendix, which must act as a safety valve. The main cavity, or atrium, abuts firmly against the aorta, the œsophagus, and the vertebræ posteriorly (Fig. 3), and marked expansion in this direction would cause dysphagia from pressure on the œsophagus, a symptom not complained of in these conditions. In front are the other chambers of the heart, so placed that enlargement anteriorly would be prevented by the impinging of the anterior cavities against the chest wall. Upward bulging would be blocked by the right pulmonary artery and the aortic arch, and below is the diaphragm, braced and supported by the liver. The natural expansile relief to overfilling would take place first to the left, and this would necessarily mean distention of the auricular appendix.

Occupying a position posterior and to the left of the appendix, separated from it only by the pericardium and the pleura, is the left upper pulmonary vein (Fig. 5). Behind, to the left, and in immediate contact with this vessel is the left bronchus (Fig. 5).

Distention of the left appendix would necessarily squeeze the left upper pulmonary vein between the appendix and the bronchus and be a factor in causing transudation. The left lower pulmonary vein is in its extrapericardiac portion out of reach of the appen-

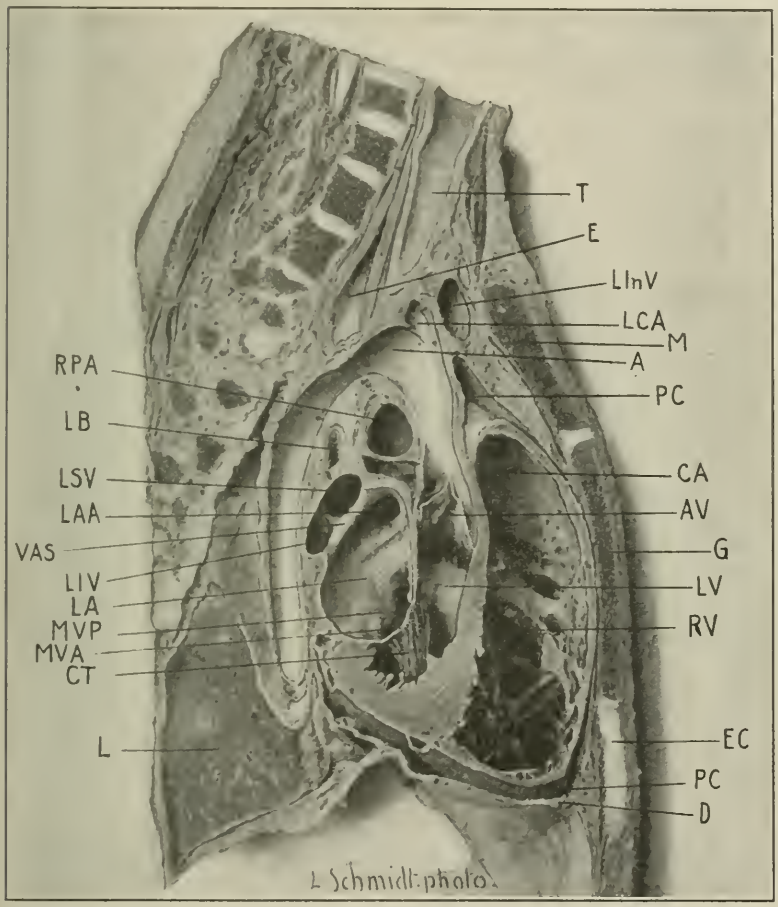

FIG. 4.—Median anteroposterior section through the thorax. *L*, lung; *CT*, chordæ tendineæ; *MVA*, mitral valve, anterior leaflet; *MVP*, mitral valve, posterior leaflet; *LA*, left auricle; *LIV*, left inferior pulmonary vein; *VAS*, veno-appendicular septum; *LAA*, left auricular appendix; *LSV*, left superior pulmonary vein; *LB*, left bronchus; *RPA*, right pulmonary artery; *T*, trachea; *E*, œsophagus; *LInV*, left innominate vein; *LCA*, left common carotid artery; *M*, manubrium; *A*, aorta; *PC*, pericardial chamber; *CA*, conus arteriosus; *AV*, aortic valve leaflets; *G*, gladiolus; *LV*, left ventricle; *RV*, right ventricle; *EC*, ensiform cartilage; *D*, diaphragm.

dix, but it is so placed that dilatation of the left ventricle would involve the vein in compression between the ventricle and the bronchus (Fig. 5). In addition, there is in the left end of the left auricle, a structure which may be a factor in left-sided effusions.

This is the almost vertical partition, which might be called the veno-appendicular septum, between the opening into the auricle of the appendix anteriorly and the left pulmonary veins posteriorly (Fig. 4). A dilatation of the appendix which would thrust this

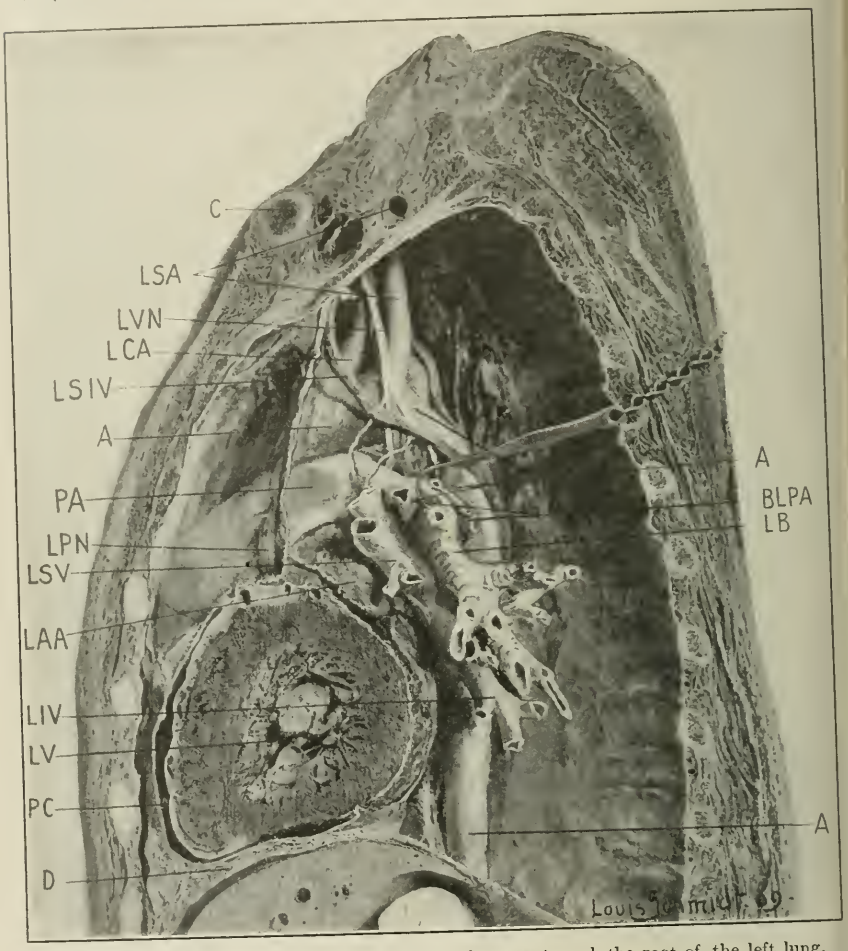

Fig. 5.—View from the left of the mediastinal contents and the root of the left lung. (Retouched photograph.) *D*, diaphragm; *PC*, pericardial chamber; *LV*, left ventricle; *LIV*, left inferior pulmonary vein; *LAA*, left auricular appendix; *LSV*, left superior pulmonary vein; *LPN*, left phrenic nerve; *PA*, pulmonary artery; *A*, aorta; *LSIV*, left superior intercostal vein; *LCA*, left common carotid artery; *LVN*, left vagus nerve; *LSA*, left subclavian artery; *C*, clavicle; *A*, aorta; *BLPA*, branch of left pulmonary artery; *LB*, left bronchus.

septum backward would seriously hinder the flow of blood from the left pulmonary veins into the left auricle and be a factor in causing transudation from their radicles.

Greater frequency on the right side is due to the fact that dilata-

tion of the right auricle is more common and more easy than a similar condition of the left side, and such dilatation is the only factor needed to cause damming back in the right pulmonary veins. On the left side, in order to include both upper and lower veins, there is needed dilatation of the left auricular appendix and of the left ventricle, with possibly a retrodisplacement of the vertical septum mentioned above, three factors as against one on the right side.

As far as we know, but one writer has come any where near the same conclusion as have we. West,[16] in considering the various factors leading to hydrothorax states that "It is not agreed from which set of vessels the effusion comes, but I think it must be from the pulmonary artery or vein; and though the obstruction leads rather to œdema of the lung than to pleuritic effusion, still the two are frequently associated together." Surely, if there is leakage from the pleura there should be a similar filtration into the lung tissue and air spaces, a condition which should cause changes in the breath sounds and the presence of moist rales. If the latter part of West's statement is correct, that is, that œdema of the lung and pleural effusion are frequently associated, it would seem to bear out our theory as to the factors at work. May it not be more than possible that the rales so frequently heard above an effusion and hitherto ascribed to compression of the lung, are due to intrapulmonary leakage from the same pressure on the pulmonary veins that is causing the presence of the fluid in the pleural sac?

Recently one of us had the opportunity of studying a case of failing compensation in the service of Dr. H. A. Hare, in the Jefferson Hospital. There were found signs of a small effusion in the right chest, and the lung above the fluid was the seat of small moist rales. The other lung was absolutely clear. The fact that such cases as this are not uncommon would tend to support the theory advanced in this paper.

A PRACTICAL HOSPITAL POLYGRAPH.

By Theodore B. Barringer, Jr., M.D.,
of New York.

(From the Department of Medicine of Cornell University Medical College, New York.)

A hospital polygraph, in order to be thoroughly practical, must be readily transportable from bed to bed, and should not be so elaborate and heavy in construction as to necessitate moving the patient to it.

[16] Diseases of the Organs of Respiration, ii, 757.

It also must have a capacity for several long records, and as simple a mechanism as is consistent with accuracy. None of the polygraphs

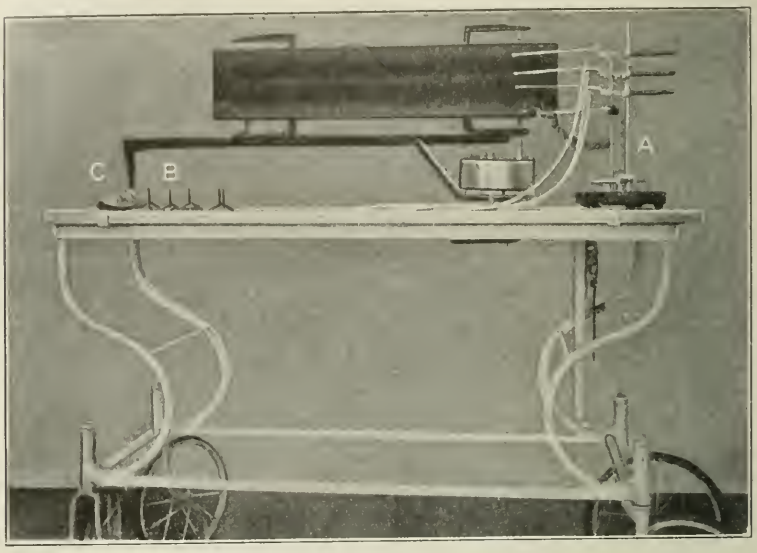

Fig. 1

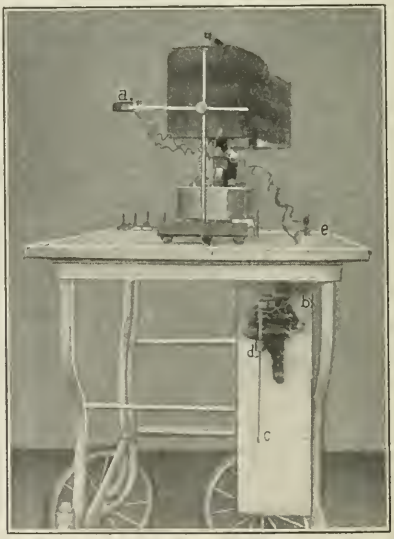

Fig. 2

I have seen has satisfied these requirements, and I have put together the apparatus illustrated in Figs. 1 and 2. There is nothing particularly original about this polygraph, for most of the parts are well

known. They have simply been collected so as to make an instrument possessing the desired requisites, and its successful use during the last year in the wards of Bellevue Hospital shows that this end has been attained. The carriage is made from an ordinary rubber-tired hospital stretcher, whose iron top has been replaced by a pine board, securely fastened to the stretcher frame. This carriage permits a rapid and easy moving of the polygraph from patient to patient, and saves much time. The drum is the long paper kymograph made by the Harvard Apparatus Co. The tambours are made with a very shallow cup and a screw adjustment, which governs the position of the writing points. They are quite sensitive, and embody the idea of Mr. J. L. Hoyt,[1] by whom they were made. In Fig. 1 they are shown, mounted on a heavy tangent stand (*a*), made for me by the Harvard Apparatus Co. The cups (or receivers), which receive pulsations from the jugular, apex beat, or liver, are of various shapes and sizes (Fig. 1, *b*). In transmitting the radial pulsations, I have used with fair success a Mackenzie transmission sphygmograph (Fig. 1, *c*). The time marker, shown in Fig. 2, consists of a signal magnet (*a*) actuated by an electric current, which is interrupted at regular intervals by the vibrating mechanism of a modified electric bell. I am indebted to Dr. H. B. Williams, of the Department of Physiology, for the idea and for its practical application.

The electric bell, with the bell removed and the clapper cut off, is shown at *b*. A rod (*c*) carrying an adjustable weight (*d*) is attached in place of the clapper.

Underneath the table, behind the vertical board to which the bell is attached, a dry cell is slung. At *e* is a switch. By moving the weight on the rod, the time unit can be varied. This one is set at one-tenth second, which I have found very satisfactory.

LOCOMOTOR ATAXIA AND PARALYSIS AGITANS IN THE SAME PATIENT.[1]

By Augustus A. Eshner, M.D.,
PROFESSOR OF CLINICAL MEDICINE IN THE PHILADELPHIA POLYCLINIC AND COLLEGE FOR GRADUATES IN MEDICINE.

It is generally admitted that locomotor ataxia is due to disease of the posterior nerve roots and of the posterior columns of the spinal cord. Various lesions in the nervous system have been described in fatal cases of paralysis agitans, but there is no common agreement in this connection, while, on the other hand, evidence

[1] Of the Department of Physiology of the College of Physicians and Surgeons.

[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 11 and 12, 1909.

has been presented in favor of the view that the symptoms of the disorder are attributable to changes in the muscles themselves. The one disease is, in the vast majority of cases, caused by antecedent syphilis, while of the other, it may be definitely stated that, although the ultimate etiological factors are unknown, syphilis certainly is not to be included among them. Neither have the two affections anything in common symptomatologically. In fact, they may be looked upon somewhat as clinical antitheses, the one being characterized by a condition of muscular hypotonia, the other by a condition of muscular hypertonia. Their associated occurrence in a given case, therefore, can be viewed only as a mere coincidence. Inasmuch as both diseases are not altogether common, it is not surprising that such association should be infrequent. To the small number of cases of this kind recorded in the literature, I am, through the courtesy of Dr. John K. Mitchell, permitted to add another instance.

A married white man, aged seventy years, occupied as a farmer, applied at the Orthopedic Hospital and Infirmary for Nervous Diseases, October 24, 1904, complaining that for more than a year he had been suffering from attacks of shooting pain in the left lower extremity, with numbness of the left thigh and a sense of weakness, and also impairment of vision. Sexual desire had been absent for two years. The functions of the bladder and the rectum were preserved. There was a sense of tingling on the forehead, but no vertigo. The gait was somewhat awkward and ataxic. Station was rather good with the eyes open, but poor when the eyes were closed. Coördination was preserved in the upper extremities, but there was some tremor of the hands. Sleep was poor. The appetite was fairly good. The bowels were constipated. The radial arteries were tortuous and felt atheromatous. The knee-jerks were absent and the elbow-jerks were diminished. The pupils were small, reacting in accommodation, but not to light. There was no diplopia, and the ocular movements were well performed. The visual fields were concentrically contracted from advanced optic atrophy. The field for green was entirely abolished. The changes were more advanced in the left eye than in the right. There was a large hernia in each inguinal region. The action of the heart was rhythmic, its sounds clear. The patient denied all history of venereal infection and of any other disease as well. He had been exposed a good deal to cold, but he had suffered no traumatism and from no form of intoxication, although he indulged moderately in the use of tobacco and of alcohol.

Under treatment with moderate doses of potassium iodide improvement in the subjective symptoms took place, but various paresthesias, such as a feeling of swelling and drawing about the mouth, itching of the eyes and nose, numbness in the lower extremities, about the hips, on the back and chest, and the genitalia, a sense of crushing

of the breast, of weight and pressure on the chest, of stiffness of the lower jaws, a feeling of looseness of the teeth, stinging pain in the face, made their appearance, and the tremor became rather more marked. There was considerable lacrymation, which was only partially diminished by dilatation of the lacrymal ducts and the use of collyria. Headache became a rather marked and constant symptom.

In the summer of 1905 the man was in the hospital for ten weeks, where, in addition to other treatment, he was given a course of coördination exercises, and he improved in many directions. Later many of the previous symptoms returned, and they gradually grew worse. In April, 1906, it was noted that the patient could walk well, maintaining his equilibrium except in rising suddenly from the sitting posture. Vision grew worse and appeared to be obscured by a mist. There was complaint of pain in various parts of the body, with itching in some and numbness in other situations. In May the gait was awkward and ataxic, station unsteady and swaying. It was now particularly noted that, on standing with the eyes closed, the right hand became involved in tremor not apparent on voluntary movement. The gait gradually grew more ataxic and staggering, and the condition of the patient became worse in other respects. The countenance was observed to be fixed, and there was also some tremor of the head. For the first time the presence of paralysis agitans was definitely recognized. There was persistent complaint of numbness of the buttocks, perineum and adjacent thighs, and girdle sense developed. Dryness of the mouth and stiffness of the jaws also caused annoyance. Discomfort was occasioned by the sense of a foreign body in the throat that would not come up or go down. In May, 1907, there was much difficulty in walking, on account of swaying and staggering, and the feet were moved clumsily. In June there was twitching of the face and jerking during sleep. In August, station was extremely unsteady and tremor in the right upper extremity was marked.

The patient presented himself at the hospital for the last time on December 30, 1907. Subsequently I learned, on inquiry, that he had died within an hour on September 3, 1908, in an apoplectic attack, in all probability due to rupture of a rigid cerebral vessel. His eyesight had grown gradually worse, but the pricking and stinging sensations in the face were absent during the last six months of life. His daughter, when spoken to about the tremor, said that to her knowledge there had been some shaking of the hands at times for many years.

We thus have a case of locomotor ataxia in an elderly man, exposed in his occupation to the elements and without a history of syphilis, in the course of which symptoms of paralysis agitans appeared and went on to classic development. As already stated,

there appears to be no reason to believe that the association was other than accidental.

The first reference to the association of locomotor ataxia and paralysis agitans that I have been able to find is contained in a graduation thesis by Anton Heimann[2] on paralysis agitans. Herein is contained the report of a case occurring in a man, aged fifty-two years, with symptoms of locomotor ataxia, who presented tremor of the lower lip and of the right upper extremity on excitement; and of a second case occurring in a man, aged sixty-six years, with some of the symptoms of both locomotor ataxia and paralysis agitans. S. Placzek[3] reports a case of locomotor ataxia in a man, aged fifty-two years, in the course of which symptoms of paralysis agitans developed. Weil[4] reports the case of a woman presenting symptoms of both locomotor ataxia and paralysis agitans. At a meeting of the Aerztlicher Verein zu Hamburg, Hess[5] exhibited a woman, aged sixty years, presenting symptoms of both diseases.

Under the title of tromoparalysis tabioformis, J. U. A. Wertheim-Salomonson[6] reports a similar association in a man, aged fifty-six years, who at one time had suffered from a transitory left hemiparesis and at another time from loss of consciousness without subsequent palsy. There was some weakness of both extremities on the right side, and memory and intelligence were enfeebled. The disorder was looked upon not as a simple combination of diseases, but as a distinct and separate affection, possibly due to posterior column disease of unusual pathogenesis—perivascular insular sclerosis. Seiffer[7] reports again the cases previously placed on record by Placzek and Weil. J. H. W. Rhein[8] reports the case of a man, aged fifty-seven years, presenting, in addition to symptoms of locomotor ataxia, fine rhythmic tremor of the hands "resembling in a striking manner that seen in Parkinson's disease." The tremor was considered an unusual symptom of locomotor ataxia rather than an actual manifestation of paralysis agitans. G. Kodderman[9] reports the case of a woman, aged sixty-two years, presenting symptoms of both locomotor ataxia and paralysis agitans. F. Penzoldt and R. Stintzing[10] state that temor, and even a well-marked clinical picture of paralysis agitans, occurs in cases of locomotor ataxia, and he refers to a case of such association. Z. Byehowski[11] reports the case of a woman, aged sixty-five years, presenting the muscular rigidity or hypertonia of paralysis agitans in the upper extremities and the face, in conjunction with the

[2] Berlin, 1888.
[3] Deut. med. Woch., July 7, 1892, p. 632.
[4] Neurolog. Centralbl., 1898, xvii, 713.
[5] Ibid., 1900, xix, 581.
[6] Ibid., 1900, xix, 741.
[7] Ibid., 1900, xix, 1119.
[8] Jour. Amer. Med. Assoc., December 27, 1902.
[9] Inaugural Dissertation, Jena, 1903.
[10] Handbuch der Therapie innerer Krankheiten, 1903, v: Handbuch der Therapie der Erkrankungen des Nervensystems, p. 698.
[11] Neurolog. Centralbl., 1904, xxiii, 786.

muscular relaxation or hypotonia of locomotor ataxia in the lower extremities, together with other symptoms of both diseases. A sister that had died was said to have presented a similar symptom complex. L. Bruns[12] reports the case of a man, aged sixty-four years, exhibiting a combination of symptoms of both locomotor ataxia and paralysis agitans. H. Eichhorst[13] reports a fatal case of locomotor ataxia in a man in whom the tremor of paralysis agitans developed a short while before death. Postmortem examination disclosed the anatomical lesions of locomotor ataxia.

Conclusions. From the foregoing facts and considerations it may be concluded that locomotor ataxia and paralysis agitans have little or nothing in common, etiologically, anatomico-pathologically, or symptomatologically. Their associated occurrence in a given case, therefore, must be looked upon as a mere coincidence. While by no means rare, neither disease is common, and their association is correspondingly infrequent. Only a small number of cases have been placed on record in which both diseases were present in the same patient.

A CASE OF CEREBRAL TUMOR PRESENTING A VERY UNUSUAL CLINICAL COURSE.[1]

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The following case seems to be one of unusual interest from the very peculiar, almost rhythmical, course that it pursued. Mrs. S., aged forty-six years, a married woman, and the mother of three healthy children, was first seen in May, 1906, complaining of severe headaches and constipation. The headaches had occurred during most of her adult life, and were nearly always associated with the menstrual period. While they were on she was scarcely able to speak, and often was sick at the stomach. Except for these headaches and the chronic constipation, she was and always had been well and full of healthy activity.

Her father died at an advanced age of cerebral hemorrhage; her mother at seventy years of a protracted diarrhœa. One brother

[12] Neurolog. Centralbl., 1904, xxiii, 978.

[13] Pathologie und Therapie der Nervenkrankheiten, Berlin and Vienna, 1907, p. 340.

[1] Read at a meeting of the Association of American Physicians, Washington, D. C., May 11 and 12, 1909

died of angina pectoris, and was found to have marked sclerosis of the coronary and cerebral arteries.

At the first consultation, in May, 1906 (about two years before her death), she was a healthy looking woman, and, as stated above, only complained of headaches and of chronic constipation. She was given a palliative and a cascara mixture to take regularly.

On June 6, 1906, it was noted that she looked "run down," spoke in a scanning manner, and wrote with difficulty, often leaving out words. The headaches were less severe. She was sent away for a long change. In October she was "looking fairly well," and had had no headache for a month. Bowels were regular with the cascara mixture. On November 6, for some days she had had occasional numbness and tingling in the left arm and hand. Right grip was sixty, and left fifty. There was no real numbness. The knee-jerks were normal. On December 10 it was noted that the patient had slight headache and was feeling dull, had difficulty in finding the right words, and "often says and writes wrong ones." Bowels were regular with medicine, and all other functions seemed to be normal. There had been no menstruation since last May. On May 15, 1907, the following note was made: "She is feeling depressed and easily worried, and has frequent headaches. Her weight keeps up. The hemoglobin registers 80 per cent. There has been no menstruation since last May."

She went away for a long change, and on returning the following note was made: "September 20, 1907.—Back from a long holiday, and looks fairly well, but faded. Hemoglobin, 80 per cent. If her bowels do not move she has severe headaches. Still has some hesitation in speech, and tends to use wrong words. Scarcely ever reads, and seldom writes letters, and if she does, may write wrong words. Sleeps well, and digestion is normal. The knee-jerks are equal and sluggish."

In October, 1907, she "had a dizzy turn. Had been sitting in the sun, and then while walking toward the house she sank down, but did not become unconscious." A few days later she had a similar turn and fell and hurt her head. Dr. McPhedran saw her with me, and we sent her into a private hospital for complete rest and observation. For about a month after entering here she seemed about the same, frequently complaining of headache and suffering from severe constipation, which drugs seemed almost powerless to relieve. Upon November 3, 1907, she seemed drowsy, although bright and cheerful when roused. The next day there was some diarrhœa—a most uncommon symptom for her—and later in the day she vomited. The next day the following note was made: "She vomited once during the night. No more diarrhœa. Since this morning she seems drowsy and now (4 P.M.), can scarcely be roused. Seems as if intoxicated, and if addressed smiles feebly, but will not put out her tongue or turn in bed. Resists arms being

moved. There is retention of urine, and this, on being drawn off with a catheter, shows no abnormality. There is no squint and the pupils are normal." Late that evening Dr. J. M. MacCallum examined the eyes and reported that they were practically normal, the veins being perhaps a little full, and slight haziness of the left fundus. By now she was absolutely comatose, with loud stertorous breathing, and fixed pupils, and it seemed as if she must die before morning. She was evidently suffering from acute cerebral compression of some sort. Next day, however, she was no worse. A lumbar puncture was done, and the fluid seemed under some increased pressure. The cerebrospinal fluid showed no chemical or physical abnormality upon most careful examination. No choline was present.

November 7. "She is rather brighter, and feels tickling of the soles of the feet. Pupils are unequal, the left being large and the right small. They do not react to light. Marked ankle clonus present in right foot. Both knee-jerks are exaggerated. A spastic athetoic condition is present in the whole body, but especially on the left side."

November 8: "She was markedly better, being quite intelligent, and recognizes people. Answers questions quickly and says that her tea 'is hot and too sweet.' The spastic condition of the muscles is gone, and the plantar reflexes are flexor in type. Pupils are equal and react normally to light. She still requires the catheter."

On December 9 it was noted that "she is quite bright and speaks better than she has done for months. No headache." She steadily improved, and on December 13 remarked that "it was a pleasure to eat and to sleep." There was no headache. Bowels were moving naturally, which they had not done for years. Urination was normal and the reflexes were also normal.

December 24, 1907. "Doing well, but there is some want of coördination in the movements of the right hand and arm. The grip is strong, but when she feeds herself she is apt to spill things, and therefore tends to use chiefly the left hand. Is easily tired."

In January she had a similar attack of what one might call cerebral compression, in which she became very dull and difficult to rouse. The right side of the body was distinctly weak, and the right pupil was larger than the left. There was slight ankle clonus on the right side and marked on the left. She had some vomiting. She got over this attack more easily than the first one, and on February 12 returned home apparently as well as when she entered the hospital three months before.

On February 15 it was noted that "she seemed well last night and slept well until 4 A.M., then became restless and soon vomited her evening meal unchanged. Vomited twice more during the morning, became drowsy, and by 3 P.M. was quite unconscious, lying on the back with head turned toward the left and the limbs

somewhat spastic. Face pale, temperature normal, and pulse sixty. Pupils small, equal, and react to light. Ankle clonus marked in *left* leg and slightly present in right. Planter reflex active and extensor in type on both sides." By 10 P.M. the pulse was 108, temperature 99.5°, and the systolic blood pressure was 210 mm. (it had frequently been measured between the attacks and never was found to be above 125 mm.). During the whole of next day she seemed to be at death's door with acute cerebral compression; but the next morning it was noted that the blood pressure had fallen to 120 mm., and by evening she was slightly conscious. The next day she was back to a condition normal for her between the attacks.

From then until March 14 she seemed to improve and was getting about the house; reflexes, pupils, etc., remained normal. The blood pressure on frequent measurements never rose above 130 mm. On March 14, 1907, she "woke at 5 A.M. feeling nauseated, and had some headache. Was dull all day, but the blood pressure did not rise above 120 mm., and by 10 P.M. she was feeling better and was sitting up in bed." Next day she seemed as usual, except that there was slight ankle clonus on the left side, and the planter reflexes were both extensor in type.

On April 8 this note occurs: "Was particularly well yesterday and walked downstairs and was speaking well. At midnight seemed restless, and then slept badly, and passed water involuntarily. This morning she seems dull, but there has been no vomiting. Is quite intelligent and answers questions clearly. There is some spasticity, best marked upon the right side. Ankle clonus and Babinski's sign well marked upon both sides." By night she was "much worse, and the spasticity almost amounts to convulsions, passing over the body in long waves. Systolic blood pressure 165 mm., but is hard to measure owing to the convulsive waves."

April 9, 9 A.M. "Had a bad night and is now quite unconscious. Spasticity very marked, but not in waves like yesterday. Pupils equal and small. Urine involuntary. Systolic blood pressure 150 mm."

From that time on the coma deepened, and the breathing got slower and slower, and she became livid. The blood pressure fell to 120 mm., but without the usual cerebral improvement. The pupils became very unequal, the left being large and the right pin-point, and later they became equally dilated. The breathing became slower and slower and then stopped altogether, the heart continuing to beat for several minutes after the last breath.

The report of the postmortem examination performed some sixteen hours after death by Professor J. J. Mackenzie is as follows:

On removing the clavarium the dura mater was found to be tense. The sinuses were filled with soft clot. On stripping the dura from the cerebrum it was found to be adherent over a large tumour mass which lay on and in the left cerebral hemisphere (Figs. 1 and 2). There were a few small flat white tumor nodules on the inner side of

the posterior portion of the dura over the left hemisphere and falx cerebri. There was a very large tumor situated between the tentorium and the occipital lobe on the left side; this was attached firmly to the tentorium and easily lifted away from the brain substance. There was considerable œdema of the pia-arachnoid; the vessels of

Fig. 1.—View of the tumor from above.

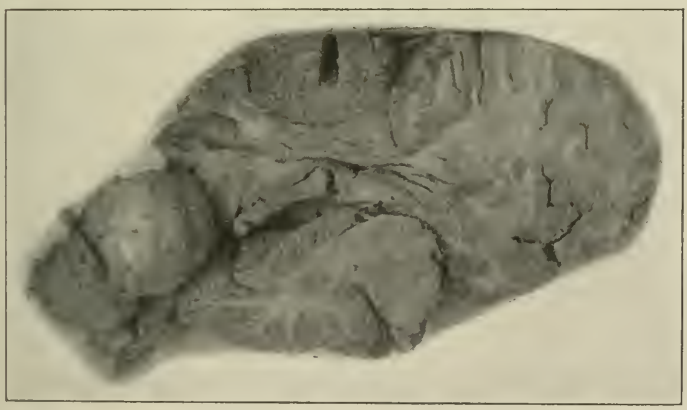

Fig. 2.—Section of the tumor.

the left side were markedly congested and the convolutions were flattened. There was some dilatation of the lateral ventricles.

The large tumor attached to the tentorium is roughly egg-shaped in outline and measures 6 cm. in its long axis. The larger end is 4.3 cm. in diameter; the smaller, 4.3 cm. in diameter. On section the tumor mass is very hard and fibrous, with a glistening cut surface. It lies upon and is attached to the tentorium on the left side, com-

pressing and pushing upward the occipital lobes, compressing especially the gyrus lingualis, gyrus cuneus, and a portion of the lateral occipital gyri. The smaller tumor masses are attached to the dura and the falx cerebri, the largest measuring 0.7 cm. in diameter, all of them quite flat, 1 to 2 mm. in thickness. They do not in any way compress the brain substance, are white in color, and of the same consistency as the large mass which is attached to the tentorium. The second large tumor, which lies upon the left hemisphere, is firmly attached to the dura. It does not infiltrate the brain, but compresses the convolutions beneath it. It is loosely adherent to the pia-arachnoid, but can be dissected off. It measures 5 cm. in its anteroposterior diameter, 4 cm. lateral diameter, and is 2 cm. in thickness. It is quite soft and of a pinkish color, with here and there areas of a deeper hemorrhagic tint, and showing throughout its substance yellow areas of fatty degeneration. In front it lies upon the gyrus centralis posterior, compressing it and pushing it forward and to the left. It also compresses most of the lobulus parietalis superior, and the lobulus parietalis inferior, which appears as a narrow band between the tumor and the marginal gyrus. The inner margin of the tumor is 2.2 cm. from the longitudinal fissure. On section the gray and white matter of the brain beneath the tumor, although distorted by the compression, do not show any gross evidence of degeneration.

Histology. The large hard tumor attached to the tentorium is exceedingly fibrous. Stained by the van Giessen method, it shows masses of red staining fibres. Between them are many cells with dark rod-like nuclei mixed with a small number of cells with large oval vesicular nuclei and faintly staining chromatin. In some areas these latter cells are present in larger numbers. The connective tissue fibers run in bundles in all directions. The vessels are well formed and are not numerous. Corpora amylacea are present but are not numerous. Mitotic figures are not seen. The soft tumor upon the surface of the parietal lobe is very cellular. The cells are somewhat smaller than the large cells of the other tumor. They are in the form of short spindles with oval vesicular nuclei. They are arranged in strands and bundles which are sometimes definitely related to the bloodvessels, and sometimes seem to run independently of them. There is very little fibre formation and the whole tumor is distinctly loose in texture. The vessels are numerous and thin walled. Mitoses are rare in this tumor also. Occasionally one finds multinucleated cells.

The histological characters of these tumors seem to point without doubt to their being endotheliomatic, arising from the dura. The large soft tumor is of the ordinary cellular type of endothelioma; the large hard tumor and the small metastatic nodules in the neighborhood would fall into the class of so-called fibro-endotheliomas. There seems no doubt that they are similar in origin, the large tumor

attached to the tentorium being probably the oldest, the soft tumor over the parietal region being the most recent. In connection with the clinical symptoms the loose texture of this latter nodule and the rich vascular supply is of great significance.

Resume. The patient for the greater part of her life had suffered from severe headaches, with some aphasia during the paroxysms. At the menopause the headaches rather lessened in acuteness, but the aphasia tended to last slightly, so that she would hesitate in speech and occasionally use the wrong words, both in speaking and writing. Next, she got an attack of what seemed like acute cerebral compression without localizing symptoms. This passed off, and she was as well as before for nearly ten weeks. Then the compression recurred, and again passed off, but recurred at intervals of a month, until the fifth proved fatal. Between the attacks she would seem nearly well, and on the day before the fatal one, spoke unusually well and walked down stairs.

The case during life seemed to us to be probably one of some sort of cyst, which periodically filled and after producing almost fatal compression emptied itself in some way. The postmortem findings were a surprise, and even yet it is difficult to explain the case. The spasticity from which she suffered during the acute attacks was nearly always most marked upon the left side, that is on the same side as the tumors were. This was probably due to cerebellar irritation from the pressure of the posterior tumor. The almost constant monthly recurrence of the acute compression is interesting. It suggests the possibility of the increased pressure being in some way connected with the menstrual function, although this had ceased over a year before. She always had had bad headaches at that time, and they continued after the flow had ceased, and the same cerebral congestion now occurring in a brain the seat of tumors had in some way produced the severe and finally fatal compression. It seems, then, that the case was one of almost symptomless brain tumor, complicated by periodic attacks of cerebral congestion, this congestion being a relic of the menstrual periods.

Vasomotor disturbances are very common at the menstrual epoch, and often persist after the menopause, as in a case recently come across in which a woman had suffered most of her life from epistaxis during the menstrual period, and after the menopause this bleeding persisted and was still occurring at the age of sixty.

The blood pressure always was very high during an acute attack. We always assumed that this was due to the cerebral compression, but Professor Adami suggested that the increased blood pressure might have been due to some unexplained cause, but very likely related to the menstrual period, and that, by producing great distention of the vascular tumors, might have been the cause of the acute cerebral compression. The anterior tumor being very vascular might well lend itself to such distention.

REVIEWS.

A System of Medicine. By Many Writers. Edited by Sir Clifford Allbutt, K.C.B., Regius Professor of Physic in the University of Cambridge; and Humphry Davy Rolleston, M.D., Senior Physician to St. George's Hospital, London. Vol. IV, Part I, pp. 764; Part II, pp. 566. London: Macmillan & Co., Ltd., 1908.

Volume IV of the second edition of Allbutt's *System of Medicine* is issued in two parts, of which Part I is devoted to diseases of the liver, gall-bladder, bile ducts, pancreas, ductless glands, and kidneys, and such other disorders as infantilism, obesity, adiposis dolorosa, and œdema, while Part II comprises a discussion of diseases of the nose, pharynx, larynx, and ear. Both parts contain much new matter, as well as revision and expansion of matter published in the first edition. Dr. Arthur Keith contributes new articles on the anatomy of the liver and on hepatoptosis. Dr. William Hunter contributes a new article on delayed chloroform poisoning, and has considerably revised his article on jaundice. He is now disposed to view all jaundice as obstructive, the one form resulting from obvious mechanical obstruction independent of changes in the blood or bile (simple obstructive jaundice), the other being dependent upon changes in the bile and blood, the actual cause of the obstruction being increased viscidity of bile consequent on intrahepatic catarrh (toxemic obstructive jaundice); this comprises the cases heretofore referred to as hematogenous jaundice. The facts in support of this undoubtedly correct contention are well marshalled, and are quite convincing; but perhaps they are not applicable to that unusual and ill-understood form of jaundice known as family jaundice. Another notable improvement is in the discussion of the cirrhoses of the liver. In the original edition, Dr. Hawkins described three types of cirrhosis—the alcoholic, the malarial, and the syphilitic (a very poor classification); in the new edition two types of cirrhosis are described—portal cirrhosis, by Dr. Hawkins, and biliary cirrhosis, by Dr. Morley Fletcher. Dr. Hawkins, in an excellent discussion of portal cirrhosis, points out the undoubted etiological significance of alcohol, and suggests that the action of alcohol is indirect rather than direct—favoring perhaps the activity of some other poison as yet undetermined; but he also points out the fact that cases of

this type of cirrhosis undoubtedly occur, in children especially, but also in adults, in which alcoholism plays no part. Portal cirrhosis then appears to be definitely toxic in nature, and alcohol, although the commonest, is by no means the only etiological factor. Mr. Mayo Robson contributes the articles on diseases of the gall-bladder and biliary ducts; they are such as one expects from a universally recognized authority. The valuable article on diseases of the pancreas, contributed to the first edition of the work by Dr. Fitz, has been replaced in the new edition by articles by Dr. W. C. Bosanquet and Dr. G. Newton Pitt. Dr. Bosanquet divides the causes of chronic pancreatitis into infective, toxic, and degenerative, and doubtfully adds a fourth, mechanical cause; he believes that the disorder is frequently due to the abuse of alcohol, and that it is often associated with fibrosis of the liver; major etiological significance apparently is not attributed to disease of the gall-bladder and biliary ducts. Mild grades of the disease (commonly encountered at the necropsy) are believed to run their course without noteworthy symptoms.

The discussion of diseases of the ductless glands has been much amplified, and now contains about all that is really known of the subject. Dr. Hector Mackenzie contributes the articles on cretinism and exophthalmic goitre; Dr. George R. Murray, those on myxœdema and lymphadenoma; Dr. E. F. Trevelyan, that on akromegaly; Dr. H. D. Rolleston, those on diseases of the adrenals and the spleen; and Dr. John Thomson, that on status lymphaticus. It is interesting to observe that in the treatment of myxœdema Dr. Murray prefers the liquid preparation of the thyroid, liquor thyroidei, which he directs should be freshly prepared once a fortnight. In the intractable headache of akromegaly Dr. Trevelyan suggests the wisdom of trephining.

Professor J. Rose Bradford contributes two articles on diseases of the kidney—the one on the general pathology of the renal functions, the other on nephritis, which replaces an article on the same subject by Dr. Dickinson in the first edition. Dr. Bradford's two articles are really excellent, and well repay careful perusal, particularly, perhaps, if one may choose from such general excellence, his discussion of so-called physiological albuminuria, uremia, the cardiovascular changes of nephritis, etc.; however, the real cause of the uremic manifestations and of the cardiovascular changes is still elusive. Professor A. Macalister contributes a short article on nephroptosis, and Mr. Henry Morris articles on the other diseases of the kidneys and the ureters.

Part II of Volume IV as now issued may be said to be a complete manual or text-book of diseases of the nose, pharynx, larynx, and ear. It is somewhat unusual to include in a *System of Medicine* such a thorough and complete discussion of subjects commonly relegated to the so-called specialties, but the excellence of the result amply

justifies the editors in their decision; and assuredly the volume will be read with interest and profit by many general practitioners as well as specialists.

Of the two parts, really two volumes, one may say that they contain a good deal of new matter as well as revision of older matter — all of which assuredly represents the statements and opinions of authorities well qualified to give expression thereto. The books are worthy members of their family, and may be heartily recommended. A. K.

Surgery, its Principles and Practice. By Various Authors. Edited by William Williams Keen, M.D., LL.D., Emeritus Professor of the Principles of Surgery and of Clinical Surgery, Jefferson Medical College, Philadelphia; and John Chalmers Da Costa, M.D., Professor of the Principles of Surgery and of Clinical Surgery, Jefferson Medical College, Philadelphia. Vol. IV; pp. 1194; 582 illustrations. Philadelphia and London: W. B. Saunders Co., 1908.

The second and third volumes of this system of Surgery were reviewed in these pages some time ago. The contents of Volume IV include: Hernia, by William B. Coley; surgery of the anus and rectum, by Robert Abbe; examination of the urine in relation to surgical measures, by David L. Edsall; surgery of the kidney, ureter, and suprarenal gland, by Joseph Ransohoff; surgery of the bladder, by Bransford Lewis; stone in the bladder, by Arthur Tracey Cabot; surgery of the prostate, by Hugh H. Young; of the penis and urethra, by Orville Horwitz; of the scrotum, testicle, spermatic cord, and seminal vesicles, by Arthur Dean Bevan; of the intestines, by Weller Van Hook and Allen B. Kanavel; surgery of the appendix vermiformis, by John B. Murphy; of the ear, by Edward B. Dench; of the eye, by George E. de Schweinitz; military surgery, by Surgeon-General Robert M. O'Reilly, U.S.A.; naval surgery, by Surgeon-General P. M. Rixey, U.S.N.; tropical surgery, by Major Walter D. McCaw, U.S.A.; and the influence of race, sex, and age in surgical affections, by William L. Rodman.

This curious collocation of articles, by which the eye and the ear are buried among the genitalia, and by which the appendix and intestines are separated from abdominal surgery by the surgery of the urinary tract, is probably due more to the dilatoriness of the authors of these misplaced articles than to any preconceived notion of the editors that the present arrangement is the best. Few of the articles in this volume are either systematic or scholarly; in many instances they appear to have been written hurriedly and with negligence, it seeming doubtful in a few cases whether the

writers have even taken the trouble to re-read their papers when once written, or when written for them even to peruse them in person. From this criticism, however, must be excepted the articles contributed by Edsall, Cabot, Young, Dench, de Schweinitz, O'Reilly, Rixey, McCaw, and Rodman, all of which give evidence of a conscientious endeavor to present the subjects treated in as comprehensive and thorough a manner as the limits assigned permit.

Coley opens the volume with a rambling and disconnected account of hernia; no student who has not studied the subject elsewhere need expect to profit by the present article unless he carefully revises and reëdits it so as to make it present some form of logical and orderly sequence. The illustrations are good as well as instructive. Abbe's article on the rectum and anus is not particularly noteworthy: we think his position in regard to the indications for iliac colotomy for imperforate rectum not sufficiently supported by facts; following Kelsey, he asserts the rarity, which is becoming recognized, of syphilitic strictures and ulcers of the rectum; no adequate account of the combined abdominal and perineal route for excision of rectal carcinoma is given. Edsall's article on the examination of the urine in surgery is decidely pessimistic, announcing that few tests give any more valuable information than is obtained by the routine examination for albumin, casts, etc. Of Cammidge's test he says: "The clinical results with it have been entirely unreliable in the hands of most skilful men. I believe that it has no clinical importance, but this is not yet fully settled."

Cabot gives an admirable clinical description of stone in the bladder, with interesting historical sketches of litholapaxy and lithotomy. He draws well-deserved attention to the former operation, which is too much neglected in this country. Young discusses other affections besides hypertrophy of the prostate, the most important of which is chronic prostatitis with its varying symptoms; he questions the role of inflammation in the production of hypertrophy, stating that it is rather a neoplastic change; perhaps to call it an adenomatoid condition would be safer.

Murphy's article on appendicitis appears to have been written several years ago, and though revised in some portions, in others is not brought up to date—notably in the account of the pathogenesis of the disease. Many of the illustrations are good, but those of the pathological lesions in the appendix are absolutely disgraceful. There is scarcely sufficient appreciation of the seriousness of the cases of those patients who have reached what Murphy calls the third stage of the disease, that is, the period immediately succeeding the subsidence of diffuse peritonitis. While a number of these patients may be assured of convalescence without immediate operation, yet in a large proportion there is only a lull before the storm of general peritonitis breaks again, owing to leakage through limiting adhesions. When patients, seen first while desperately

ill with diffuse peritonitis, have been brought successfully through to this stage by perseverance in the "Ochsner treatment" of abstinence, it is a very difficult thing to decide the most propitious time for operation; and it is to be regretted that so experienced a surgeon as Murphy evades the issue by passing over this question in silence.

In regard to military surgery, it is well pointed out by General O'Reilly that what the ordinary surgeon lacks is not so much a knowledge of how to treat the wounded in battle, but an understanding of the organization and administration of medical affairs in both war and peace; and he urges the National Guard surgeons to avail themselves more freely of the Army Medical School. Dr. Rixey's account of naval surgery is extremely interesting, describing as it does in detail the arrangements for treating the wounded during a battle and subsequently, and the construction and equipment of hospital ships.

The two most interesting subjects in tropical surgery discussed by Major McCaw are appendicostomy for amœbic dysentery, and the treatment of hepatic abscess. He does not recommend appendicostomy as a last resort, nor in patients whose recovery is improbable, on account of the extent of the lesions in the colon; but he urges it in those cases in which improvement under rectal irrigation occurs up to a certain point and then stops, showing that the disease is still in a curable stage, but that irrigation from below is inefficient. For the diagnosis of hepatic abscess he prefers exploratory laparotomy to the trocar. He describes only the abdominal and the subpleural thoracic routes for evacuating the abscess, but does not write as if he had much personal experience with operation; and his neglect to discuss the transpleural route is somewhat remarkable.

The present volume hardly maintains the standard set by those which preceded it; and it is to be hoped that the contributors to the final volume, the publication of which in the near future is announced, have made more successful efforts to expound intelligibly the principles of surgery and to further its art.

A. P. C. A.

Diseases of the Digestive Canal. By Paul Cohnheim, M.D., Specialist in Diseases of the Stomach and Intestines in Berlin. From the second German edition, edited and translated by Dudley Fulton, M.D. Philadelphia and London: J. B. Lippincott Co., 1909.

This is a refreshing book written obviously from material and information gathered in actual practice by the author himself. It is not burdened with references from the literature, but the author is

familiar with the important contributions to diseases of the digestive tract. The presentation is admirably clear. There is no hedging, nor can there be any doubt as to what the author means. Certain defects are apparent in the plan of the book. Of these, the most serious are: A persistent dogmatism, many statements being made in a positive manner for which no adequate proof is offered, and with which not all physicians working in this line would be likely to agree. Second, there is the omission of many things which it is to be presumed Dr. Cohnheim regards as useless, but which have authority to support them, and therefore should have been offered to the reader for consideration. A brief but admirable section upon anamnesis opens the book. The description of the physical examination cannot be regarded as complete so far as the methods described are concerned. The subject of auscultation which is one of the most important and in the hands of an expert reveals almost as much information about the abdomen as does the auscultation of the thorax, is discarded in three lines. The subject of the laboratory tests are given considerable space, and they are described sufficiently for the general practitioner. The diagnosis of chronic gastritis is apparently to be made provisionally in all chronic cases in which ulcer, carcinoma, or dilatation of the stomach can be excluded. There is a diagram of the evolution of the disease showing how in some cases, it leads first to hyperacid gastritis with subsequent subacid gastritis, and in others to subacid gastritis from the beginning. This corresponds more to the author's theory than to anything found in actual practice. The discussion of the diagnosis of ulcer is entirely inadequate. This may be judged from the statement that "epigastralgia occurring at a definite time after eating, is the most positive symptom of gastric ulcer." Percussion tenderness is entirely overlooked, and the value of occult blood minimized. Surgical treatment is restricted to perforations, persistent hemorrhage. and for the relief of complications. There is apparently some confusion between dilatation of the stomach and retention of the stomach contents, and although the diagnosis is not carefully described, it is said to be very easy. The subject of gastroptosis is rather cursorily treated. The treatment is largely limited, apparently, to mechanical means of support, and there is no discussion of the surgical treatment of the disease. The author is quite modern and gives a long paragraph on suggestive treatment for the condition called nervous dyspepsia. He speaks of this again in nervous or reflex vomiting. Nervous conditions can be recognized by the fact that actual pain is never a symptom. The sections on diseases of the intestines is one of the best brief descriptions of this subject that has as yet been made. As in the section on the stomach, the physical examination of the abdomen has been neglected. Typhlitis is resurrected, classified, and described. There are excellent chapters on diarrhœa and constipation. It seems doubtful if many physicians would be willing

to diagnosticate with assurance from the brief account of the symptoms and physical signs described by the author. The appendix contains one of those unfortunate tables designed for the use of the ill-equipped physician, who does not care to examine his patient thoroughly. There are a variety of diets which have the advantage of being quantified as well as indicating what foods are to be taken, and a sort of quiz compend at the end which has the extraordinary title of Clinical A, B, C of the Most Important Disturbances of the Digestive Tract.

The book is supplied with a number of prescriptions. One must remark the frequency with which belladonna or some of its preparations appears in them and for what a diversity of conditions this drug is used. It must also be noted with general regret that a very large number of proprietary preparations are recommended. There are a number of illustrations, many of them including portraits of the author. As these actually show him performing certain examinations, they may be regarded as of advantage. J. S.

A Text-book on Embryology. By Frederick Randolph Bailey, A.M., M.D., and Adam Marion Miller, A.M. New York: William Wood & Co., 1909.

Embryology as a science is so vast and so constantly being modified by new discoveries that a clear, concise, and up-to-date treatise on the subject is of great value, not only to the scientist, but the student as well. The forelying text-book brings the subject thoroughly up to date. As is general in such works, the book is divided into two parts—one on general development, and the other on organogenesis. In the former are presented the preliminary steps in development, including the structure of the cell and cell proliferation, oögenesis and spermatogenesis, maturation, fertilization, cleavage, formation of the germ layers, the fœtal membranes, and the development of the external form of the body. In dealing with these subjects the authors have taken great pains to avoid unnecessary elaboration, and present the subject matter in as succinct a form as possible. Special attention is paid to the various theories with regard to the processes involved and the physiological significance of the processes themselves. A very valuable feature of the work consists of the practical suggestions offered, not only in the matter of technique, but also how, when, and where to procure and prepare material for practical work. The second part of the work on organogenesis takes up the development of the various organ systems at length, somewhat too copiously, perhaps, for the needs of the average medical student, but with a clarity of diction and careful handling of the subject matter which holds the attention of the reader at all times. Students

of medicine and practitioners will greatly appreciate the paragraphs at the end of each chapter on the anomalies occurring in the different organs under discussion. This feature alone will recommend the work as particularly valuable as a text-book for medical men. The illustrations are numerous, well chosen, and well made, and contain a number of photogravures showing actual conditions. The last chapter of the work on teratogenesis materially aids in explaining the causes and method of development of those strange conditions so puzzling to the average practitioner of medicine.

H. H. C.

DIE WURMFORTSATZ-ENTZUNDUNG: EINE PATHOLOGISCH-HISTOLOGISCHE UND PATHOGENETISCHE STUDIE. (APPENDICITIS: A PATHOLOGICO-HISTOLOGICAL AND PATHOGENETIC STUDY). By L. ASCHOFF, Professor of General Pathology and Pathological Anatomy in Freiburg i. B., Germany. Pp. 114; 18 lithographic plates and 22 figures in the text. Jena: Gustav Fischer, 1908.

THIS monograph, the result of seven years' work, is based on a study of one thousand appendices removed at operation by the author's surgical colleagues, Rotter, Enderlen, Küttner, Kraske, and Krönig. Six hundred of these appendices have been studied with the utmost precision. The preparation of the volume was undertaken for two reasons: (1) Because so many papers and discussions on appendicitis show misinformation of the pathology and ignorance of the normal anatomy of the appendix; (2) because these later studies serve to confirm the conclusions published by Aschoff four years ago. In the limits allotted to this review it will be impossible to do more than very briefly outline the author's views, which differ in many points very materially from those usually taught.

Aschoff holds that all cases of appendicitis are similar, in that the same pathological stages occur in each. The first lesion ("Primärinfekt") in his experience has *never* been a catarrhal inflammation of the appendix: *always* he has found a localized inflammatory reaction in the depths of one of the crypts or recesses of the lumen of the appendix. This Primärinfekt, which is more often multiple than solitary, does not consist in an exfoliation of epithelium in the nature of an erosion or ulcer; it is a round-cell infiltration of neutrophiles under the epithelium of the crypt, and if the epithelium itself is altered at all, its place is taken by a plug of leukocytes and fibrin. The lymphatic follicles are not involved; but from the focus of primary infection the inflammatory reaction quickly spreads along the lymph channels to the serous coat, which may be widely inflamed before any extension in the mucosa occurs. This second or

phlegmonous stage may pass on into the formation of miliary intramural abscesses; these may perforate either into the lumen of the appendix or into the peritoneal cavity—miliary perforations. To this stage succeeds that of ulceration, which he has never seen unless preceded by the phlegmonous. The ulceration of the mucosa may result in perforation (macroscopic perforations), or in gangrene of the appendicular wall, owing to circulatory disturbances.

The pathogenesis of appendicitis consists in the localization of the primary focus in the distal end of the appendix, largely owing to stagnation of the contents of the appendix from the physiological kink in the middle third of the organ. Aschoff supports the enterogenous theory of infection, as opposed to the hematogenous: yet he declares that the inflammation does not extend as a catarrh from the cecum, since this and the proximal part of the appendix almost invariably are uninflamed. He thinks coproliths harmless in an appendix, and is inclined to regard them rather as the result than the cause of inflammation; but they may be a cause of relapsing appendicitis by favoring infection through mechanical trauma. They frequently are produced by stagnation of the appendicular contents between two strictures left by previous inflammation. Usually the severest inflammation is distal to the concretion, not over it; and perforation on its distal side is by no means rare. His bacteriological studies have shown the presence of diplococci (streptococci) as the primary infection; he never found Gram-negative bacilli (colon bacilli) in the Primärinfekt.

He denies the existence of a chronic appendicitis without previous acute stages similar in all respects to those described. The symptoms may have been insignificant, but the histological picture of such cases of chronic appendicitis convinces him that the pathological process was the same in all. Likewise, appendicitis obliterans is regarded as the result of ulcerative changes secondary to a phlegmonous stage, itself inaugurated by the primary infection of one or more crypts. Hemorrhages and the so-called hemorrhagic appendicitis he regards as artefacts due to operation.

We should have been satisfied if the pathologist had concluded his manuscript at this point. The exposition of his views as to the treatment of appendicitis impels one to apply the proverb, "*Ne sutor ultra crepidam.*" His argument for medical treatment (opium), by which he hopes to prevent general peritonitis by favoring the formation of an abscess in all cases in which recovery without perforation does not occur, appears to consist in urging the inclusion in the statistics of recoveries after medical treatment of all those cases which present so insignificant symptoms as never hitherto to have been considered frank attacks of appendicitis. The only *raison d'être* which he admits for operative removal of the appendix is the prevention of other attacks, which he regards as nearly inevitable.

A. P. C. A.

SELF-CONTROL AND HOW TO SECURE IT. By DR. PAUL DUBOIS. New York: Funk & Wagnall Co., 1909.

DR. PAUL DUBOIS, of Berne, has followed his work on the psychic treatment of nervous disorders with one entitled *Self-control and How to Secure It*. This at least is the not very satisfactory title which represents the French *L'Education de Soi Même*. The former book was an apotheosis of commonplace notions gathered together without originality, except in some points in which the originality was of no advantage. The present is worse than this. It has a superficial aspect of depth; it says a good deal about the worship of the True, the Beautiful, and the Good; but it does not amount to anything in the final analysis. It is vague, windy, and verbose, and the reviewer's only satisfaction in reading it he got from the page on which the following occurs. The author scolds the doctors and says they set up minor truths as dogmas and have created a code of doctrines which are nothing but superficial appearances of truth and, upon being put to the test, do not work in practice. "These hopes, often announced to the public, have developed that pre-occupation about health which is the disease of the present generation." After which he writes another hundred or two pages in which he promotes pre-occupation of the most unreasonable kind. J. K. M.

A TEXT-BOOK OF THE DISEASES OF THE EAR FOR STUDENTS AND PRACTITIONERS. By PROFESSOR DR. ADAM POLITZER, Imperial-Royal Professor of Aural Therapeutics in the University of Vienna. Translated and edited by MILTON J. BALLIN, Ph.B., M.D., Assistant Surgeon, of the New York Ophthalmic and Aural Institute, and CLARENCE L. HELLER, M.D. Fifth edition. 337 illustrations. Philadelphia and New York: Lea & Febiger, 1909.

IT is always a pleasure to review a new edition of a book when it really presents the most recent advances in the subject of which it treats. Professor Politzer's book has long been the standard work, not only in Germany, but throughout the world, on diseases of the ear. The present edition maintains the high standard of its predecessors, and is, if possible, even more satisfactory in its contents and arrangement. As an instance of the thorough and careful revision which this edition shows, we may cite the handling of the subject of labyrinthine suppuration, for nothing in the recent progress of otolgy has been more marked than the late advances in the study and treatment of diseases of the labyrinth. In the fourth edition, Professor Politzer separated the consideration of the anatomy,

physiology, and pathology of the sound perceiving apparatus from that of the sound conducting apparatus, placing the section dealing with this subject toward the end of the book. In this edition, the section dealing with the anatomy and physiology of the sound perceiving apparatus is placed immediately after that dealing with the consideration of the anatomy and physiology of the sound conducting apparatus, and the section which deals with labyrinthine suppuration is placed where it properly belongs, in juxtaposition, midway between the section dealing with suppurative diseases of the middle ear and the section dealing with intracranial complications of otitic origin. The recent studies of von Stein, Barany, Alexander, and others are given the fullest consideration, so that the reader is put in possession of all the facts bearing upon this most important topic. The above is but one instance of many in which the book shows revision and improvement. It is not merely a reprint with a few additions, but a thorough, careful, and complete revision and rewriting of it. Although, of course, many of the old illustrations are retained, there are a number of additional engravings in the present volume. It may be said of this work that there is not one page nor one illustration which could be spared from its contents. From cover to cover it contains nothing but what is essential to the science of otology. There is no padding of any kind, and he who possesses it may feel that it contains the most complete, authoritative, and recent exposition of aural science obtainable. Much credit is due to Drs. Ballin and Heller, the translators, who have shown their ability to render thoroughly, not only the text, but the spirit of the author's work. F. R. P.

New and Non-official Remedies, 1909. Pp. 167. Chicago: Press of the American Medical Association, 1909.

This is a small volume that contains a description of more than 200 new and non-official remedial preparations approved by the Council on Pharmacy and Chemistry of the American Medical Association prior to January 1, 1909. For the most part the preparations are discussed alphabetically, but a classification has been adopted that permits an easy comparison of remedies of similar origin and properties; mixtures are relegated to an appendix, and mention is made of a number of non-proprietary remedies that have not been admitted to the Pharmocopœia. The book dealing as it does, with the important physical and chemical properties of the newer remedies, their processes of preparation, their action, uses, and dosage, is a welcome addition to the Pharmocopœia, and should prove of interest and value to practitioners and pharmacists. A. K.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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Toxic Meteorism in Infectious Diseases.—Krönig and Klopstock (*Deut. Archiv f. klin. Med.*, 1909, xevi, 515) note the fact that in typhoid fever, pneumonia, puerperal sepsis, and erysipelas the presence of meteorism depends much less on the condition of the abdomen than on the degree of general infection. In the cases of pneumonia examined by them more than a half had this complication. It was much more common in the severe cases, and almost constant in alcoholics. Of the 27 fatal cases, 81.5 per cent. developed marked meteorism. It was less frequent in the young than in the old. They believe it to be dependent on something specific in the infection, as it is very rarely present in scarlet fever, diphtheria, miliary tuberculosis, or cholera. As to the cause of the tympanites, increased intestinal fermentation associated with high temperature, as well as more limited passage of gases, are factors, but the important thing is apparently a lowered intestinal tone. This lowering of the tone of the smooth muscle of the intestine is analogous to the lowering of vascular tone which is present in pneumonia and is probably due to cerebral toxemia. Experiments on dogs and guinea-pigs, in which the intestine was inflated, resulted in more rapid and superficial respiration, together with a rise in diastolic and systolic blood pressure—the latter not marked in the guinea-pigs. The respiration is affected by pressure and fixation of the diaphragm, while blood pressure change depends on the added demands of the rapid breathing, increased pressure in the abdomen, and obstruction to the return flow of blood from the lower half of the body. In fatal instances respiratory failure precedes cardiac failure.

Abdominal Symptoms at the Onset of Pneumonia.—Glaserfeld (*Berlin. med. Woch.*, 1909, xlvi, 1451) reports two cases of pneumonia in which the onset simulated appendicitis. While the local physical signs of pneumonia are usually evident in such cases in from two to four days, in one of the instances here reported they did not appear until the ninth day. The atypical onset is most frequent with involvement of the lower lobes, but may occur when any lobe is attacked. Both of these cases terminated in lung abscess, and in 21 similar cases, Bennecke found 13 which had complications. The differential diagnosis is often most difficult, but Glaserfeld lays especial stress on the following points: The facial expression in pneumonia is less anxious when the sensitive part of the abdomen is palpated than in appendicitis. There is no facies abdominalis. The tongue is usually moist, and only slightly coated. The respirations are rapid. The abdominal spasm is not so localized as in early appendicitis. The abdominal tenderness is superficial. Deep palpation in the appendicular region does not give rise to any marked degree of pain. A most careful examination of the chest is of the first importance.

The Leukocytes and Differential Count in Acute Abdominal Infection.—As a result of blood examinations made just before operation in 184 cases of acute appendicitis, and in a number of other acute abdominal conditions, Coons and Bratton (*New York Med. Jour.*, 1909, xc, 205) have devised a chart which forms a basis for the comparison of total leukocyte counts with the percentage of polynuclears, and which frequently throws light on both diagnosis and prognosis. They lay great emphasis on the polynuclear percentage as an indication of the presence and extent of sepsis. If the polynuclears are below 75 per cent. and a leukocytosis is present, one may look for an old, walled-off infection with perhaps an acute exacerbation. With the polynuclears ranging from 75 per cent. to 80 per cent., the blood evidence alone is rather indefinite. A polynuclear count of from 85 per cent. to 90 per cent. points to a severe infection, and in the presence of a leukocytosis indicates immediate operation. Especial stress is laid on a polynuclear count above 90 per cent. Twenty-five of the thirty-one cases with over 90 per cent. of polynuclears had some form of peritonitis. The worst prognosis was found in the cases of low leukocyte count with high polynuclear percentage. In these cases there was apparently little to be hoped for from operation.

The Intravenous Injection of Diphtheria Antitoxin.—Experimental evidence has shown that the amount of diphtheria antitoxin needed to neutralize a given amount of toxin depends largely on the time interval between the two injections. In order to obtain more rapid and effective action of diphtheria antitoxin, Schreiber (*Münch. med. Woch.*, 1909, lvi, 1591) has injected it intravenously instead of subcutaneously. Nineteen of twenty cases thus treated recovered. He used small doses of 2000 units at first, but later found that 6000 to 10,000 units produced no ill effects, even in children. With large doses the temperature fell more promptly than with the subcutaneous administration, and the general condition seemed to improve quicker. There was, however, no increase in the effect on the membrane itself.

The Opsonic Index in Typhoid-bacilli Carriers.—Gaehtgens (*Deut. med. Woch.*, 1909, xxxv, 1337) states that in a series of 386 cases of typhoid fever, 77, or 20 per cent., could be traced with great probability to chronic typhoid carriers. The role played by such individuals in the spread of typhoid fever makes it of the utmost importance to find a simple and accurate method for detecting them. The examination of the feces for the presence of the typhoid bacillus, which is as yet the only method employed, is difficult to carry out, and is unreliable. The organisms may be absent from the stools over long periods, so that repeated examinations are negative, and also there is always the chance that a suspected person may send a specimen of feces not his own, lest he be proved a carrier. Gaehtgens' investigations have been on the occurrence of antibodies in the blood of persons who have had typhoid fever. Previous research has shown that during the course of the disease there is a distinct rise in the opsonic index. The determination of the index on twelve persons who had had typhoid fever, but who were not carriers, showed that, except for a slight rise (1.4; 2.1) in 2 cases whose illness was only three to four months previous, the opsonic index was normal. Sixteen cases, however, who had been proved to be typhoid carriers, showed, with one exception, a distinctly high index, which averaged 2.8. The only instance in which the index was not high was in a man who had typhoid twenty-nine years before, and from whose stools the organisms were often absent for months at a time. The occurrence of agglutinins in the serum of typhoid carriers was also determined, with the result that while opsonins and agglutinins were frequently found increased in association, the agglutination reaction was negative in 25 per cent. of the cases, and was thus of much less value in the recognition of carriers than was the opsonic index.

The Measurement of the Blood Pressure.—In an effort to determine the value of the obliteration of the pulse as an index of systolic pressure, Leonard Hill (*Heart*, 1909, i, 73) found no evidence that the resistance (hypertonus) of the arterial wall to compression affects it. Relative softness or hardness of the arterial wall affects the conductance of the systolic wave, and modifies the reading, especially if the systolic wave is large. The arm and leg readings are the same in young men in the horizontal position, and differ in the standing or inverted posture by the hydrostatic pressure of the column of blood which separates the points of measurement. In vertical postures the leg pressure is very variable, while the arm pressure is kept about constant. The arm and leg readings differ after violent exercise and in aortic insufficiency because the big systolic waves are conducted better down the leg arteries, which are slightly thicker or more contracted and rigid. A marked difference between arm and leg readings was found in all cases of aortic regurgitation; in persons lying quietly in bed Hill considers such a difference as a sign of diagnostic importance. The two factors to be taken into account in reading systolic pressures are the actual maximal pressure of the cardiac output and the conductance of the pressure wave by the artery. The force of the wave is damped down in soft arteries, as sound waves are damped by velvet. Blood pressure readings should be taken under uniform conditions, with patients lying hori-

zontally, not emotionally excited, not after exercise, not after taking hot food, tea, coffee, or alcohol, and not after the hand and wrist have been warmed or chilled. The pressure should not be read when it is first raised, but after it has oscillated up and down several times, since this gives a more accurate index, especially in the leg arteries. The reappearance of the pulse gives the pressure more exactly than the disappearance.

The Examination of the Eye-grounds in the Differential Diagnosis between Pernicious Anemia and Severe Secondary Anemia.—Hesse (*Deutsch. med. Woch.*, 1909, xxxv, 1394) calls attention to the fact that in the diagnosis of pernicious anemia the examination of the retina is of great value, especially in those cases in which the blood picture is indefinite. In 47 out of 50 cases of pernicious anemia, retinal hemorrhages were found to be present; while in 51 cases of severe secondary anemia, in which the hemoglobin was below 50 per cent., and in 121 cases with a hemoglobin of 50 per cent. to 70 per cent., retinal hemorrhages were never found. In 64 instances of malignant tumor (43 of carcinoma and 29 of gastric carcinoma), in which there is especially liable to be confusion with pernicious anemia, he never saw retinal hemorrhages. Hesse believes not only that the presence of hemorrhages favors the pernicious type of anemia, and their continued absence a secondary anemia, but that they are of prognostic importance as well. In general the retinal hemorrhages are larger and more numerous in the severe cases, and the clearing up of the hemorrhages is one of the earliest indications of beginning improvement.

Nodal Bradycardia.—In the new English journal devoted to the circulation (*Heart*, 1909, i, 23) Mackenzie describes a hitherto unnoted type of bradycardia in which venous tracings show no wave due to the contraction of the auricle, and in which he believes the stimulus of the heart's contraction arises not, as normally, in the great veins, but in the auriculoventricular node, causing a simultaneous contraction of auricles and ventricles. In the four cases reported, the bradycardia was either temporary or permanent. The heart's rate averaged 30 to 40 per minute, and the rhythm was regular or irregular, with frequent long pauses. Two of the patients had attacks of syncope and convulsions, so that they simulated closely cases of heart-block (Adams-Stokes syndrome), but the venous tracings never showed evidence of disturbed conduction, nor was there any pulsation in the veins due to auricular systole. This type of bradycardia may arise suddenly in a heart previously showing a normal rhythm, or it may arise in a heart previously showing the nodal rhythm of the usual type, with the rate more frequent than normal.

Clinical Symptoms of Hypertrophy of the Left Ventricle.—Kurt (*Wien. klin. Woch.*, 1909, xxii, 1120) calls special attention to the significance of the heaving apex impulse as a sign of left ventricular hypertrophy. As evidence of moderate hypertrophy he believes the accentuation of the first sound over the left ventricle, just above the apex, to be of considerable importance. Normally, the first sound is less loud over the

left ventricle than over the conus of the right ventricle. He found this accentuation of the first sound to be present in cases of hypertrophy following exercise, pregnancy, old age, and associated with obesity, certain valvular lesions, early arteriosclerosis, and nephritis. It was not present in cases of orthostatic albuminuria. The weakening of the first sound, so frequently noticed with the heaving impulse of marked hypertrophy, he considers to be chiefly a matter of poor sound conduction.

The Rise of Blood Pressure in Nephritis.—Marcuse (*Berlin. klin. Woch.*, 1909, xlvi, 1352) bases his theory of the rise of blood pressure in nephritis on the investigations of Wiesel and Schur as to the presence of adrenalin in the blood of patients suffering from nephritis. He suggests that the renal inflammation causes an obstruction to the flow of blood in both kidneys, and that as a result of this there is an increased flow of blood in the inferior suprarenal artery, which is a collateral of the renal artery. The consequent hyperemia of the adrenals would lead to their hypertrophy, and with this would come an increase in the production of adrenalin which would cause a rise of blood pressure.

Effect of the Injection of Bile on the Circulation.—While it has been generally considered that the slowing of the pulse and the lowering of blood pressure in obstructive jaundice is caused by the retention of bile salts, King and Stewart (*Jour. Exper. Med.*, 1909, xi, 673) have shown, by the intravenous injection of whole bile and of its constituents into dogs, that these substances play a very unimportant part. The injection of pig's whole bile caused a preliminary rise in blood pressure, then a progressive fall, followed by a slowing of pulse rate. The bradycardia is due to heightened vagus tone, as it is relieved by cutting the vagi, or by the administration of atropine. The injection of an amount of sodium glycocholate somewhat in excess of that contained in a lethal dose of whole bile produced no circulatory effects. The injection of bile pigment (biliverdin), however, produced all the effects of the injection of whole bile, except for the initial rise of blood pressure, and the lethal dose of uncombined pigment corresponded almost exactly to the amount of pigment contained in a lethal dose of whole bile. Experiment also showed that when the bile pigment is in chemical combination with calcium or sodium, a comparatively non-toxic compound is formed. To study the role of calcium in the action of pigment further, obstructive jaundice was produced in dogs by tying the common bile duct. An analysis of the tissues then showed a definite increase in the calcium content of the blood, and a definite diminution of calcium in muscle, liver, and brain. They believe that the bile pigments circulating in the blood in obstructive jaundice absorb the available calcium from the organs and tissues, and by combination with it render themselves less toxic.

The Influence of the Thyroid on Carbohydrate Metabolism.—King's experiments (*Jour. Exper. Med.*, 1909, xi, 665) relate to the interaction of the thyroid and pancreas. Clinically, glycosuria is not uncommon in exophthalmic goitre, while in myxœdema it practically

never occurs. Gray and de Sautelle (*Jour. Exper. Med.*, 1909, xi, 659) have shown that glycosuria produced by the administration of ether is materially reduced by extirpation of the thyroid. In order to exclude as far as possible the action of organs other than those under investigation, and to make the conditions as simple as possible, King used Cohnheim's method of working with extracts of organs. His object was to find what influence the thyroid gland has on the action of muscle juice and pancreatic juice in breaking down dextrose. A series of experiments showed that the addition of thyroid to the mixture of muscle, pancreas, and dextrose retarded very definitely the destruction of dextrose. The same result was obtained when the thyroid was boiled—proving that the action was not due to a ferment, but to a thermostable body. Further work showed that iodothyrin, the active substance of the thyroid, acts exactly as does normal thyroid. His experiments show that the relation between the thyroid and the pancreas is antagonistic, in that the carbohydrate mechanism is rendered less efficient when the thyroid enters into the reaction, and they agree with the clinical evidence, which shows that the thyroid has a very definite control over carbohydrate metabolism.

SURGERY.

UNDER THE CHARGE OF

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Transplantation of Joints.—Lexer (*Archiv f. klin. Chir.*, 1909, xc, 263) believes that the transplantation of joints has a future, but that its perfection will come slowly. He offers some observations which he has made in connection with his experience. The incision should be in the form of a flap which should be so made that it will avoid as far as possible, falling over the transplanted part, so that there will be less opportunity for the escape of wound secretions at a suture and the formation of a fistula. In resections of the knee, the incision should begin on each side on a level with the upper border of the subcrural bursa and should extend downward to the tuberosity of the tibia, where the transverse portion of the incision is placed. The part should not be rendered bloodless for the removal of the diseased or injured joint preparatory to transplantation, since the least collection of blood would prevent the early adhesion of the soft tissues to the transplanted joint, which is necessary to quick healing. In fashioning the defect for the reception of the transplanted joint, the bone should be divided at right angles to the long axis. It may even be notched to

prevent rotation of the transplanted bones. In either case the parts should be fitted exactly together. The new joint itself should be as nearly intact as possible, and should be obtained from a simultaneously amputated limb. It should not be the seat of disease, injury, or tumor, so that the supply of proper material is scanty. Lexer believes that such joints remain sterile, as a rule, twenty-four hours after removal. He suggests that under the strictest antiseptic precautions, a joint may be taken from a dead body. The preparation of the part to be transplanted is very important. All ligaments, tendon attachments, fat, and muscles should be carefully separated from the periosteum, and the joint should be transferred immediately without contact with aseptic or antiseptic solutions. If any free periosteum remains attached to the bone ends of the defect, it should be made to cover the approximated cut ends of bone of the defect and transplanted joint. In one case a transplantation of a complete knee-joint, with its internal ligaments intact, at the end of two months, was successful, in that there was fair movement and no fistula. A month later, however, a fistula had developed. In fixing the transplanted piece in place, the use of all foreign bodies, such as nails, wire, or ivory pegs, should be avoided. If anything of the kind is necesssary, pegs of fresh bone should be employed. Plastic operations on the muscles are very important in the later function of the joint. The difficulties are considerable, however, on account of the frequent contraction and atrophy of the muscles or their destruction by disease. In the after-treatment, the first movements should be undertaken as soon as union is obtained between the bony ends. The end results can be judged only after a long time. Of most importance is the preservation of the cartilage.

Experimental Investigations on the Sensibility of the Abdominal Cavity.—Ritter (*Archiv f. klin. Chir.*, 1909, xc, 389) says that the question concerning the sensitiveness to pain of the abdominal cavity has been variously answered. He carried out a series of experiments on dogs and rabbits to determine this question. All the animals, especially the dogs, possessed a marked sensibility in all organs. To obtain positive results it was necessary to observe certain precautions. The use of local anesthetics, such as cocaine or its substitutes, as well as general anesthetics, was avoided, and, at the most, morphine was depended on. Parts poorly supplied with bloodvessels were the least painful. The bloodvessels themselves were the most painful. The best test of sensibility was by the ligature of bloodvessels. The pain caused by ligature was greater than that produced by irritation of the parietal peritoneum or pulling on the mesentery. Exposure of the intestine to the cold air or exposure to ordinary temperature for a long time causes the sensibility to diminish rapidly. The intestines are most sensitive in acute conditions. There have been observations made in men which tend to show that the abdominal cavity in them is sensitive. There are, possibly, certain differences between men and dogs. In men the ligation of vessels is especially painful. The lack of sensitiveness in the abdominal organs in men can not be due to the effect of infiltration anesthesia by cocaine, because this has a purely local effect. By the use of cocaine, however, the painful ligation of the vessels can

be prevented. The best explanation for any lack of sensation in the abdominal organs is to be found in the damage done to the fine sensory fibers in the abdominal cavity. These injuries have been demonstrated by various investigators, but only in animals. They can, however, be accepted for the same conditions in men.

An Operative Cure of a Hernia into the Fossa Duodenojejunalis of Treitz.—Heller (*Archiv f. klin. Chir.*, 1909, xc, 360) reports a case in which a man had been suffering for six months from a chronic intestinal obstruction, and had become very weak and miserable. Vomiting occurred every twelve to twenty-four hours, with a sudden ejection of 1 to 2 liters of a gall-stained watery fluid, but without bad odor. The central parts of the abdomen were distended, the flanks rather sunken. The central distention was in the form of a globular tumor about the size of a man's head, in the region of which peristaltic movements were visible and audible. The diagnosis of a probable tuberculous peritonitis was made, with a kinking of the upper part of the intestine. After a wide opening of the abdominal cavity had been made, there was visible a tumor which had somewhat the appearance of a thick-walled ovarian cyst. The large intestine could not be seen encircling the mass of small intestines. Upon raising the lower pole of the tumor, the lowest coils of the ileum could be seen slowly emerging from a funnel-shaped opening. They were adherent to the margins of the opening, and could not be drawn out. The hernial sac was then split in its whole extent in the median line, when the enormously distended coils pushed out. They were, however, adherent within and were separated with much difficulty. From the duodenojejunal flexure to the cecum the coils were separated from one another, centimeter by centimeter, in order to remove the numerous kinks in them. The stomach was seen to be very much distended, and to be continued with a wide open pylorus over into the duodenum, which was almost as large as the arm. The stomach and pylorus, as well as the enormously distended ileum, were especially adherent to the inner wall of the sac, and kinked. The remaining intestines were also dilated and hypertrophied. At the completion of the separation of the intestines, the patient was in collapse. Two days after operation he again became acutely collapsed. Upon a partial reopening of the abdominal wound, an acute dilatation of the stomach was detected. A Kader fistula was made into the enormously dilated ileum, from which escaped large quantities of gas, but almost no fluid. The patient improved. On the fourth day the fistula had closed. A half year after operation the patient had gained eighteen pounds. Digestion continues without disturbance.

Reduction of an Unreduced Dislocation of the Shoulder by Posterior Arthrotomy.—Madelung (*Archiv f. klin. Chir.*, 1909, xc, 1126) in resections of the head of the humerus, employs the Kocher method, which consists of a curved posterior incision with a chiselling off of the spine of the scapula. It gives a free exposure of the joint, the function of the deltoid and the other shoulder muscles remains good, and subluxation of the upper end of the humerus toward the coracoid process is prevented. Madelung employed it in a case of subcoracoid dislocation of the

right shoulder, unreduced seven weeks after the accident. The right arm and hand were apparently paralyzed, although there were no considerable disturbances of sensation. The elbow was rigid in a right-angle position. An attempt to reduce the dislocation under ether was unsuccessful. By the Kocher method the shoulder-joint was exposed and opened. The dislocation was easily and quickly reduced by the use of a strong resection hook, which was aided by a large lever with a spoon-shaped end. The acromion was fastened in position with sutures. Three days after operation passive movements and the use of electricity were begun. The wound was completely healed in seven days, but the symptoms of paralysis disappeared slowly. Ten weeks after operation the patient could elevate his arm to a level with the shoulder. Passive movements in the shoulder were completely free, and he could make a fist. Pronation and supination at the elbow were possible. Active extension, however, was only slight.

Ligation of the Veins in Portal Thrombosis from Appendicitis.—Wilms (*Zentralbl. f. Chir.*, 1909, xxxvi, 1041), in a severe case of appendicitis, ligated the veins passing from the cecum and appendix, with the result that the chills promptly ceased, and good healing followed. The course of the case was as follows: On May 14, a man, forty-two years old, was relieved of a large appendiceal abscess, which had been developing for ten days. He had had two chills at intervals of several days. On the second day after the incision had been made into the abscess, another chill occurred; on the third day three chills with temperatures of 41.1°, 40.8°, 40.3° C., and on the fourth day a chill with a temperature of 40.4°. Two hours later the ligation of the veins was performed, and was soon followed by a normal temperature and healing without further complications. All the cases of suppurative, portal thrombosis which Wilms had previously seen proved fatal, the great danger being development of liver metastases. Early operation for appendicitis has rendered them much less common than formerly. The resection of the cecum and lower end of the ileum may come into question after ligation of the veins. In performing this operation, Wilms passed his finger under the ascending colon, which, with its mesentery and that of the lowest portion of the small intestine, were separated from the posterior abdominal wall. After division of the anterior layer of peritoneum, the vessels came well into view. The small arteries were isolated so that they would not be included in the ligatures. All the veins were then ligated in two bundles. A tampon was placed on the cecum for drainage in case gangrene of the cecum occurred. Not a single chill followed the operation. The operation must be done before liver metastases develop.

The Operative Treatment of Tuberculosis of the Vas Deferens and of the Seminal Vesicles.—Cholzoff (*Ann. de mal. des org. gén.-urin.*, 1909, ii, 1121) says that these organs may be reached by one of three routes—the inguinopelvic, the perineal, and by temporary resection of the sacrum. Twelve cases are reported. When, after castration or epididymectomy, we are convinced that the tuberculous process in the seminal vesicles shows no signs of regression, but remains *in statu quo*

or is progressing, the diseased seminal vesicles should be removed, If the tuberculous process is frankly inflammatory from the beginning, and occurs simultaneously with the tuberculous process in the testicles or epididymis, the seminal vesicles should be removed at the same time as or soon after the castration or epididymectomy. If, in the operation of removing the seminal vesicles, the prostate is found to be tuberculous also, the diseased parts of this gland should be removed. When the tuberculosis involves the sexual and genital organs, the removal of the diseased parts of the genital apparatus is contra-indicated only when the tuberculosis of the urinary tract is too far advanced or there is involvement of both kidneys. If a simultaneous tuberculosis of the lungs or other organs is limited, and if it has not a marked effect on the general condition, operation is not contra-indicated. If the bad general condition depends upon the tuberculous lesions of the genital organs, we have a formidable reason for operating as soon as possible. The removal of the seminal vesicles can be performed by the inguino-iliac route in case of absolute necessity, when after orchidectomy or epididymectomy one establishes that the process has involved the whole vas deferens, and when before operation the lesion of the seminal vesicles had not been recognized. If in denuding the vas higher and higher, with the purpose of reaching a sound portion, one becomes convinced that it is diseased in its whole course, the seminal vesicle should be removed also. When the disease is unilateral the inguino-iliac route should be employed. The perineal route gives less space for the removal of the seminal vesicles than the sacral, but takes less time for the execution of the whole operation, and should be employed in feeble patients in whom the removal of all the diseased organs in two operations should not be done. In those patients in whom the removal of the diseased portions of the genital apparatus can be done in two stages, one may employ the temporary resection of the sacrum, by which more space and easy access to the seminal vesicles is afforded. The immediate results of spermatocystectomy are very satisfactory. The mortality in insignificant. There are some reasons for believing that the final results are satisfactory. A large number of cases followed for a longer or shorter time have not recurred.

The Late Results of Ureterocystostomies.—Boari (*Ann. d. mal d. org. gén.-urin.*, 1909, ii, 1232) reports on the late results of 13 collected cases of ureterocystostomy, which have been preformed in the past ten years. One was performed in 1908, and another in 1909. In still another an autopsy had been performed a year after operation. In all the others the time which has intervened since the operation varies from five to ten years. The operation in all these cases was performed with the aid of an anastomosis button, which was first suggested by Boari. Although, in the 9 cases studied, the operation were performed by eight different surgeons, the immediate results were good in all. It was generally agreed that the technique was easy. Boari believes that the failures reported from other methods are due to: (1) The fact that the anastomoses are poorly made, either because of a faulty technique, or the sutures are too close, together causing a consequent stenosis of the orifice; (2) healing by first intention

does not ocur, so that fistulæ develop and later stenosis; (3) the condition of the kidney and ureter prior to operation interferes with success, an ascending infection often causing fistulæ. In the 9 cases the operation was necessitated by a ureteral fistula, consecutive to a severe labor or to a vaginal hysterectomy. In order to preserve the functional value of the kidney, the operation should be done as soon as possible after a positive diagnosis has been made, since infection of the kidney occurs easily. The good results obtained, and proved by a number of cases during a prolonged period, indicate that the anastomosis by a button deserves every confidence. Late stenosis has been observed in some cases in which the anastomosis was made with sutures, but never in those made with a button.

The Double Filigree Operation for the Radical Cure of Inguinal Hernia.—McGavin (*Brit. Med. Jour.*, August 14, 1909) says that it is the general complaint of surgeons that in inguinal herniæ in patients of poor physique, in elderly subjects, in those whose occupations involve a constantly recurring increase of intra-abdominal pressure, and in any patient in whom such a hernia has recurred or has exceeded the more moderate dimensions, the prospect of accomplishing a really radical cure is extremely remote and in some cases quite impossible. In all cases in which hernia occurs or recurs, the chief factors are: (*a*) the presence or formation of a peritoneal sac; (*b*) the recession or pushing aside of the muscular walls of the inguinal canal; (*c*) the stretching of the fibrous structures covering the canal. The method of making the filigree is described. As perfect asepsis is necessary here, or more so than in the implanting of abdominal filigrees, they should be placed in ether for five minutes, to remove all grease from them, and should be left in the sterilizer in the centre of the boiling area until the moment of implantation, when they are lifted directly from the sterilizer into the wound. The operation is at first conducted exactly as in an ordinary Bassini, except that the aponeurosis should be split to a point rather farther out, and the peritoneum must be more freely separated from the under surface of the conjoined tendon, as must the latter from the aponeurosis overlying it. The sac having been isolated and dealt with, the cord is held out of the way, and the first two of the sutures which are to approximate the conjoined tendon to Poupart's ligament are inserted, and their ends caught by pressure forceps. These sutures being held aside by an assistant, the pubic section of the filigree is placed upon the peritoneum, its narrow end being close to the pubic spine and its wide end at the inner margin of the internal ring. The conjoined tendon is then brought in close contact with Poupart's ligament over the filigree, by the two sutures already inserted, and as many more inserted as are deemed necessary, the bed in which the filigree lies being kept as dry as possible. In cases in which the muscular wall external to the internal ring is strong, the cord is placed in position and the iliac section of the filigree is taken from the sterilizer and is placed beneath the aponeurosis in such a way that its inner end lies over the internal abdominal ring and upon the cord for a space of three-fourths of an inch, the outer end being carried outward and laid upon the surface of the internal oblique muscle, one or two sutures holding it in place. If the above mentioned weakness

is present, the muscular wall is divided from the ring outward toward the iliac spine for about an inch, and is separated from the peritoneum by the handle of a scalpel. Upon this peritoneum the outer end of the iliac section of the filigree is laid, being lightly sutured in place, and the muscles are brought together over it. The inner end lies as already described. Finally, the aponeurosis is sutured and then the skin wound. This method was employed with excellent results in 33 cases, in none of which was there a remote prospect of a cure by Bassini's operation.

THERAPEUTICS.

UNDER THE CHARGE OF

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The Karell Milk Cure: Its Modifications and Indications.—Roemheld (*Monats. f. d. Phys.-diät. Heilkunde.*, 1909, i, 32) reviews the Karell milk cure and its various modifications. Karell, in 1865, published his results in over 200 cases treated by the use of skimmed milk. He gave about four to eight ounces of milk three or four times a day, and made an especial point that it should be taken very slowly Oertel thinks that the beneficial effects of such a cure are due simply to the restriction of fluids. He believes that such a starvation cure tends to a loss of good weight and consequent lowered vitality. Leyden agrees with this point of view, and adds that a weakened heart requires an especially nutritious diet. Hirschfeld has found this diet useful in the treatment of cardiac insufficiency, but thinks that an easily digestible mixed diet restricted in amount has equally good results. Lenhartz has made use of a modification of the original Karell cure in the treatment of chronic bronchitis, in the treatment of heart disease with passive congestion, and in order to hasten the loss in weight in obesity when the heart is insufficient. Lenhartz gives four doses of 200 c.c. of milk at four hour intervals. No other nourishment or fluids are allowed for a period of from five to seven days. In the succeeding two to six days other articles are gradually added to the diet, so that at the end of twelve days the patient is receiving a fairly liberal diet. Roemheld himself makes use of from 1000 to 1200 c.c. of milk on two days of each week. During the rest of the week a mixed diet containing two-fifths to three-fifths of the necessary amount of calories is ordered. He has found these milk days especially useful in the treatment of obesity associated with heart disease, nephritis, or gout. He uses them when the ordinary method of treatment fails or to maintain the loss of weight after the ordinary diet. Thus, a patient may control his weight for years by the interposition of two milk days a week with an ordinary restricted diet. Moritz uses large quantities of milk on a definite plan. He does not consider rest in bed essential, though the patient should be under observation for

unfavorable effects, such as headache, malaise, etc. Roemheld thinks that the beneficial effects of milk cures are due to the restricted amount of food, which, combined with rest, has a favorable influence upon the heart. The value of such a cure is enhanced by rest in bed. This is especially true in the case of the severer diets. The low sodium chloride content of the milk cures also causes an increased diuresis. Consequently, he believes that failure of heart compensation is the chief indication for the use of milk cures. Roemheld advises the method of Moritz and his own method for the treatment of obesity. He thinks that a combination of the two plans is very valuable. Thus, the Moritz plan may be followed to secure the reduction in weight which subsequently should be maintained by the interposition of milk days in a restricted diet. The loss of proteid weight and the loss of iron can be prevented by this combination. Roemheld concludes by saying that the Karell milk cure and its modifications are not specifics for obesity, but that, with proper supervision, they are valuable adjuncts to the ordinary methods of treatment.

Calcium Lactate in the Treatment of Epilepsy.—Littlejohn (*Lancet*, 1909, 4472, 1382) has treated a number of epileptics with calcium lactate, giving it in doses of 15 grains, three times a day. He says that the improvement in these cases is sufficiently marked to justify his recording the facts. All of the patients (number not given) improved under this treatment. Littlejohn gives the detailed histories of two of the more successful results obtained, and hopes that others may be persuaded to try this treatment.

The Use of Cerium Oxalate for the Relief of Vomiting.—Baehr and Wessler (*Archives Int. Med.*, 1909, iii, 516) have endeavored to determine experimentally the usefulness of cerium oxalate as an antemetic. Their conclusions are as follows: (1) Commericial cerium oxalate is non-toxic. (2) Cerium oxalate has no inhibitory effect whatever on vomiting of central origin. (3) Cerium oxalate may inhibit vomiting due to local irritation of the gastric mucosa, but only if given in large doses for some time, so as to coat the stomach wall pretty generally. (4) Cerium oxalate is not absorbed from the gastro-intestinal tract. Baehr and Wessler say that these four propositions complete a very close analogy between cerium oxalate and bismuth subnitrate. Both salts are insoluble and, because of this property, neither is absorbed from the gastro-intestinal tract. This latter fact, in the case of bismush, has resulted in its use for various local disorders of the alimentary canal, but, so far as the literature furnishes any evidence, the use of cerium oxalate has been directed, in great part against the reflex vomiting of pregnancy, a condition in which no one could rationally think of using bismuth. They say that cerium oxalate ought to prove efficacious in alleviating all those conditions for which bismuth is now used. Cerium oxalate has been found valuable in relieving the irritability of the stomach in alcoholic gastritis and in allaying the gastric disturbances that occasionally occur in the course of the infectious diseases. They believe that there is no reason for doubting its value in these and similar conditions, such as gastric ulcer. However, they believe that most men

prescribe cerium oxalate in too small doses, 2 to 3 grains. Since it appears that cerium oxalate accomplishes its purpose by mechanically coating the walls of the stomach they advise the administration of cerium oxalate in doses comparable to those in which bismuth subnitrate is given.

The Treatment of Fibrinous and Serofibrinous Pleuritis.—Lord (*Boston Med. and Surg. Jour.*, 1909, clx, 469) says that it appears to be established that at least three-fourths of the primary pleurisies with effusion are tuberculous. It is also especially significant, he adds, that out of every ten cases of primary fibrinous or serofibrinous pleurisy, at least three or four develop pulmonary or other tuberculosis within an average period of from four to six years. Therefore, Lord suggests that it seems not too radical that all cases of primary fibrinous and serofibrinous pleurisy, even the mildest forms, in patients otherwise in apparent health, should be treated as if they were tuberculous until they can be proved otherwise. Lord discusses the question of thoracentesis in detail. He has collected a series of 500 cases of serofibrinous effusion, in which sudden death before tapping occurred in three patients. The autopsy showed the cause of death to be pulmonary embolism in two, of whom one had a double effusion, the second a large unilateral accumulation. In the third case, no other cause than pulmonary œdema could be discovered. The frequency with which effusions may be complicated by venous thrombosis also suggested itself to Lord by the occurrence of five cases of pulmonary infarction in a total of fourteen autopsies in this series. He thinks that the removal of fluid and the consequent change in the intrathoracic pressure may dislodge a thrombus and thus cause the infarction. He considers thrombosis with the danger of subsequent embolism and infarction more liable to occur in effusions of long duration, and consequently advocates their early removal. Furthermore, these dangers are increased in double effusions and in large unilateral effusions. Lord also discusses the advisability of tapping medium-sized and small effusions. Many clinicians maintain that a pleural effusion has a conservative function, limiting the respiratory motion on the affected side and thus giving the affected lung the opportunity of spontaneous healing. In addition, the fluid may contain protective substances. Lord says that the clinical evidence supporting these views is inconclusive. However, he believes that the removal of medium-sized and small effusions is not immediately necessary. A short delay may furnish evidence of spontaneous absorption. If this does not occur he advocates tapping. The relief of the pressure alleviates the symptoms, and the course of the disease seems to be shortened. The early removal of the fluid may prevent the formation of venous thrombi and consequent pulmonary infarction. In addition, the danger of pleural adhesions is lessened by early removal. In his series of cases there was less tendency for a re-accumulation of the fluid after early tapping. Lord also mentoins the fact that the early removal of fluid excludes the possibility of mistaking an empyema for a serofibrinous effusion. With reference to the amount of fiuid to be withdrawn at a single tapping, Lord advises immediate withdrawal of the needle whenever there are any symptoms of distress. These symptoms are usually cough, pain,

or dyspnœa. Usually 1500 c.c. should be the maximum amount withdrawn and more than 2000 c.c. should never be withdrawn. The operation, is usually without any danger in uncomplicated cases in young subjects. In cases of longer duration, however, or those with complicating pulmonary, cardiac, or mediastinal disease, and in patients past middle life, Lord says that the first operation may well be regarded as an experiment. The accidents that may occur are fatal pulmonary or pleural hemorrhages, pulmonary infarction, and pneumothorax. Albuminous expectoration usually results from the removal of too large quantities of fluid, though a few cases have been reported after removal of very small quantities. Lord prefers to use a trocar and cannula with only slight negative pressure exerted by the aspirator. He believes that the cannula is less irritating to the lung and pleura, and in the event of the cannula becoming blocked it may be opened by re-inserting the trocar.

The Employment of Iodopin in Syphilis.—Freshwater (*Brit. Med. Jour.*, 1909, i, 1228) gives the technique of the administration of iodopin. Iodopin is prepared by repeated iodization of sesame oil by means of iodine monochloride in alcoholic solution. It is a light oily liquid, and is prepared in two strengths, 10 per cent. and 25 per cent. More recently a solid iodopin has been introduced, and is supplied in yellow-coated tablets of 7½ grains each, corresponding to one grain of potassium iodide. Iodopin may be given by the mouth, or by subcutaneous or intramuscular injection. Freshwater prefers to use the 25 per cent. iodopin, intramuscularly. He gives it in doses of 20 grains, injected daily or every other day. These are given until a total of from 200 to 300 grains of iodopin have been injected. He gives one or two such courses of treatment during a year. No more than six courses of iodopin should be given to the individual patient because of the infiltration of the buttocks resulting form the injections. Freshwater sums up the advantages of iodopin as follows: (1) It is often necessary to give a long course of iodine to patients who are unwilling to take iodide, either because of its depressing effects or because they are the subjects of iodism. (2) All the iodopin injected is used up and must exert its specific action; an exact dosage is, therefore, possible. (3) Injections are painless, and there is no fear of sepsis if proper precautions have been taken. (4) Iodopin subcutaneously does not produce iodism. Patients who have an idiosyncrasy to potassium iodide can take it quite well. (5) Patients remain much longer under the influence of iodine than when iodine is given in other forms. After a short course of injections the system can be kept for a period of four to six months under the influence of iodine. (6) The body is under a slow, continuous, regular action of iodine, which is of prophylactic value. (7) In nearly all cases, after a prolonged course of potassium iodide, there are stomach and bowel troubles. This does not occur with iodopin. (8) It has a specific action in tertiary syphilis and arterial degeneration. Freshwater adds that the disadvantages to the use of iodopin may be briefly summarized as follows: (1) In cases of syphilis, when a rapid therapeutic effect of iodine is required, iodopin is of little use, as the absorption of iodopin is extremely slow, two to ten days elapsing before the iodine can be definitely demonstrated in the urine, so that in cases in which there is a threatened perforation of the

palate, cerebral gumma, etc., potassium iodide should be given. Iodopin is not a subsitute for potassium iodide when active lesions are in progress. (2) A further disadvantage is that only a small amount of iodopin can be absorbed per diem, about one-third what would be given in the ordinary way by the mouth. This can, however, be turned to account in various ways. For example, in tertiary syphilis, after potassium iodide has been administered somewhat vigorously, a course of iodopin injections may be given, and the patient may then be left without medicine for some months, during which time the physician knows that iodine is daily passing through his tissues.

A Contribution to Digitalis Therapy,—Müller (*Münch. med. Woch.*, 1909, xviii, 904) reports a series of cases treated, some with digalen and some with digitalis leaves. Müller says that digalen should not be termed soluble because it apparently does not go into solution in pure water, but only in a mixture of water, alcohol, and glycerin. He has found it unsatisfactory for subcutaneous or intramuscular injection, the injections being painful and at times causing considerable inflammatory reaction. Many of the French observers, Müller says, have found that it possesses no such advantages over other preparations of digitalis as have been claimed. Other observers have advocated the use of digalen because of its lack of cumulative action and of its less irritative action upon the stomach. Müller administered the digalen by the mouth in this series. He found that digalen was as effective as other digitalis preparations, but could determine no superiority. Digalen was apparently less irritating to the stomach, but Müller could not definitely determine a lack of cumulative action. He concludes, therefore, that digalen differs but little in its action from other preparations of digitalis, and has no marked advantages over the other preparations.

The Therapeutic Action of Potassium Bitartrate.—Eichhorst (*Med. Klinik*, 1909, xi, 381) reports three severe cases of ascites due to cirrhosis of the liver treated by potassium bitartrate. The potassium bitartrate owes its beneficial action to its marked diuretic properties. This diuretic action does not occur immediately, but after the diuresis is once obtained it persists for some time. In one of the cases described this medication was kept up for four months with marked benefit. The patients lose the ascites, their appetite returns, the bowels move normally, and they gain in strength. At the same time the subicteroid tinting of the skin and conjunctivæ disappears. Eichharst's experience has taught him that the operative treatment of patients with cirrhosis of the liver offers but little hope. He has also had disappointing results with other drugs, and, therefore, highly recommends the use of potassium bitartrate. Eichhorst first excludes tuberculous peritonitis in cases of ascites by means of tuberculin. He has the abdomen rubbed once a day with green soap, and puts the patients on a milk diet or on a light mixed diet with the addition of three pints of milk a day. He also gives potassium bitartrate in cases of pleurisy with effusion, but its effect in this is not so remarkable as in the ascites due to cirrhosis of the liver.

PEDIATRICS.

UNDER THE CHARGE OF
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The Relation of Infectious Diseases (Measles, Whooping Cough, and Influenza) to Tuberculosis in Children.—EDGAR COPELAND (*Archives of Pediatrics*, 1909, xxvi, No. 7) concludes that the development of tuberculosis as a complication or sequel to measles, etc., is all but invariably dependent upon the preëxistence of a latent focus of infection, and that its dissemination is directly due to the lymphatic activity accompanying the pulmonary inflammation associated with these diseases. In a large number of cases tuberculosis may develop a long time after the infectious disease which is its etiological factor—from a few months to several years. He emphasizes the prevalence of latent tuberculosis in children, which he has proved at autopsy findings to be 7 to 8 per cent. at one year. Some investigators, like Kelynack, claim 40 per cent. of all children under fifteen years show evidences of tuberculosis. The infectious diseases are characterized by marked catarrhal inflammation of the mucous membranes of the entire body, and by greatly lowered powers of resistance to secondary invaders. He believes the key to the connection between tuberculosis and these diseases is this catarrhal condition of the respiratory mucous membrane; not in predisposing to infection, but rather in promoting activity of a quiescent disease. The lymph streams swollen to many times their normal volume, flowing through the tuberculous foci, sweep the bacteria into the circulation for distribution over the entire body. Depending on the virulence and amount of the infectious agent, and on the resistance of the tissues, general miliary disease may or may not ensue. Many cases of bronchopneumonia on which tuberculosis is supposedly engrafted are really miliary tuberculosis from the beginning. The extreme grade of inanition following these three infectious diseases lower resistance and favor the disseminating tubercle. Influenza is rarely if ever followed by tuberculosis when such a condition did not previously exist. The incidence of tuberculosis as a complication or sequel of these three diseases is of relatively small importance, considering the morbidity of the diseases.

The Incidence of Tubercle Bacilli in New York City Milk.—A. F. HESS (*Jour. Amer. Med. Assoc.*, 1909, lii, 1011) has studied the milk of the city of New York to determine the number of instances of tubercle bacilli in it, to study the nature of these bacilli, and to report on the health of children known to consume this milk. He found virulent bacilli in 17 of 107 specimens examined, in all 16 per cent. of the milk retailed from cans in New York City. Animal inoculation had to be resorted to, to demonstrate them. Cream and sediment contained them, making it necessary to use both in making inoculations. The organisms were also found in one specimen of commercially pasteurized

milk, showing that this method, as now carried out, does not insure protection. The label "pasteurized" should only be given to milk heated for a length of time and to a degree of temperature sufficient to render it an absolutely safe food. In 16 of the specimens the type of bacillus was bovine; in the last a human variety was differentiated, showing that contamination from tuberculous individuals should be guarded against. A number of infants and young children who drank such contaminated milk, when examined one year later, seemed to be in average health. One fourth reacted to tuberculin, and one of these was in poor physical health, having suffered from a glandular infection. Even though 90 per cent. of tuberculous infections come from human beings, we are not justified in neglecting the bovine danger; a small percentage of infections means thousands of cases. Milk not coming from tuberculin-tested cows should be pasteurized or brought to a boil; all herds should be carefully examined, all cows tested with tuberculin, and all animals which react condemned or isolated.

Chronic Glomerulonephritis Treated by Double Decapsulation.—E. Garceau (*Boston Med. and Surg. Jour.*, 1909, clx, 707) reports the case of a girl, aged fifteen years, who had marked symptoms of nephritis since her fourteenth year, no disease having preëxisted except scarlet fever during her first year and whooping cough during her fourth year. Double decapsulation was performed, the kidneys at that time being large, soft, regular, yellow, and mottled. The capsule was thin and easily stripped. Marked improvement followed, and nothing was complained of for more than fifteen months, when renal symptoms returned gradually, death, however, not occurring for more than three years from the time of the operation. Her life was prolonged by the operation, she suffered less, and the improvement was directly due to the operation. At the autopsy the kidneys were small, rough, nodular, and pale, thus being in marked contrast to their appearance at the time of the operation; the capsules were thickened and stripped with difficulty.

Napkin-region Eruptions in Infants.—H. G. Adamson (*Brit. Jour. Dermat.*, 1909, xxi, 37) distinguishes between at least four distinct types of eruptions involving the napkin region of infants, which are of non-syphilitic nature and which must be carefully distinguished from the eruptions of congenital syphilis. The most frequently occurring of these are the various forms of erythema of Jacquet: (1) Simple erythema is found oftenest on the prominent convex surfaces of the buttocks and neighboring parts; in severe cases it may extend to other parts of the body. It is most common in very young infants. (2) The erythematovesicular type presents in addition toward the centre of the convex area, small bright red erosions; at the periphery of the erythematous areas are small vesicles; the erosions by coalescence denude large areas. (3) The erythematopapular type has flat red papules on an erythematous base. (4) In the ulcerating form the erosions have gone deeper instead of granulating. The last three forms occur in infants several months old. These four forms depend upon a vasomotor disturbance, possibly of gastro-intestinal toxic origin, with a determining factor of local mechanical irritation. The seborrhœic dermatitis occurs

in babies a few months old. The entire napkin region is involved. The eruption is bright red, small scales being over all. Other parts besides the napkin region are also involved, more particularly the scalp, umbilicus, behind the ear, etc. The cause of the condition is microbic and the cure a daily boric acid bath, with sulphur ointment (10 grains to the ounce). A third class of eruptions is the vacciniform ecthyma; it resembles the erosive and ulcerative stage of Jacquet's erythema, and is of streptococcic origin. The bullous impetigo is also streptococcic. It is usually found in other parts of the body besides the napkin region. The phlyctenular margins reveal the impetiginous nature of the bullæ denuded of their epidermis.

Rheumatoid Arthritis.—J. Porter Parkinson (*Brit. Jour. Children's Dis.*, May, 1909) reports a case in a child, aged two years and four months. There was swelling of the wrists ankles, knees, and elbows, for three months. There was a brown stain on the skin on the front of the legs and thighs and on the lower abdomen. Some of the joints were very tender. There was some effusion and considerable periarticular swelling, but no grating, and the skiagram showed no bone changes. The spleen was enlarged, as were the lymph glands of the groin and axilla. Examination of the blood showed hemoglobin, 30 per cent; red cells, 1,000,000; white cells, 5000. The temperature fluctuated daily, the highest evening rise being 104° F. During the febrile periods the joints became worse, the spleen and lymph glands enlarged, and the child seemed to be suffering from a general poisoning. In three months the temperature fell, the joint swellings diminished, as did also the spleen and lymphatic glands, and there was no tenderness or adhesions in the joints. Salicylate of sodium seemed to give the best relief. Parkinson believes the symptoms due to periodic intoxication from one of the affected joints. The skin pigmentation is a point of resemblance to the disease as it afflicts adults.

Purpura Fulminans.—Charles A. Elliott (*Archiv. Int. Med.*, April, 1909) reports a case in a girl, aged eight years and seven months. There was no history of hemophilia, and the child had had pertussis, measles, and varicella. Seventeen days before the onset of purpura she had a mild attack of scarlet fever, from which she recovered rapidly, with no albumin or casts in urine. The onset of purpura was marked by severe sore throat, swollen tonsils and cervical glands; systolic murmur at base of heart; temperature 102° to 104° F., and albumin and granular casts in the urine. On the third day a purpuric spot appeared on the right ankle and rapidly spread, covering the dorsum of the foot. The left ankle was next affected, until the dorsal and plantar surfaces of both feet were covered. There were large blebs on the dorsal surfaces and gangrene of the affected toes. Hematoxylin-colored areas, varying in size, appeared all over the body more or less symmetrically, and were usually œdematous and tender. Fine petechial spots were scattered over the skin of the legs and later on the abdomen and chest. There was nosebleed, excessive desire to urinate, and occasional delirium. On the fifth day an anemia developed, shown by blanching

of face, lips, and hands. Blood examination showed hemoglobin, 55 to 60 per cent.; red cells, 2,720,000; white cells, 65,400. Death occurred sixty-eight hours after appearance of purpura and twenty-two days after onset of scarlet fever. Autopsy report showed hemorrhagic subcutaneous infiltration; hemorrhagic infiltration of pelvic fascia, of submucosa of urinary bladder and of right ovary; cervical adenitis, persistent thymus, and cloudy swelling of liver and kidneys. Cultures from heart's blood, serum of bullæ, and smears from splenic pulp showed no growth. Elliott discusses 56 cases of purpura taken from literature, 9 of which were similar to his own, and followed scarlet fever during the second and third week. Fifty-two out of the 56 died, and the average course of the disease was fifty-two and a half hours after first appearance of purpura. In 32 cases no predisposing cause was given. In the remainder, scarlet fever preceeded 11, diarrhœa 3, and pneumonia, measles, and exposure each 2. The mental state usually remained clear to the end, and there was a remarkable absence of gross pathological change. It would appear that purpura fulminans differs from other forms of purpura in degree only, there being a complete chemical reaction and a progressive severity.

OBSTETRICS.

UNDER THE CHARGE OF

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Repeated Cesarean Section and its Complications.—Brindeau (*L'Obst.*, January, 1909) reports six cases of repeated Cesarean section. In one the placenta was attached to the uterine cicatrix of the first operation. Its separation caused considerable hemorrhage, but did not complicate the patient's recovery. In the second operation some adhesions were found between the omentum and the abdominal wall which were easily separated. The uterine incision of the first operation had perfectly healed. The incision was made parallel to the first incision. The mother recovered without complications. In the third case the omentum was found adherent and it was necessary to ligate it and separate it. The uterine wall was composed of connective tissue at the site of the first scar, and the incision was made parallel to it. The pregnancy was twins, and mother and children made good recoveries. In the fourth case the omentum was adherent to the abdominal wall and required ligation. The placenta was adherent to the border of the former uterine cicatrix. It was necessary to use caution in separating it. There was, however, no unusual hemorrhage, and the mother recovered without incident. In the fifth case the omentum was adherent and also the uterine and abdominal walls. At one point fibrous tissue had developed, which practically united the two cicatrices. Some difficulty was experienced in keeping the intestines out of the way. There was very little

hemorrhage. The next day after the operation the patient became cyanotic, and died with congestion of the lungs and anuria. The sixth patient had had a Cesarean section, and recovery was complicated by abscess in the abdominal wall. The second pregnancy was terminated by Basiotripsy. The third pregnancy was terminated by section, and there was found a considerable hernia of the abdominal wall. This was resected. The omentum was adherent, and it was necessary to resect a portion of this. The patient's recovery was complicated for several days by moderate fever. The patient presented herself some years afterward in labor with considerale fat in the abdominal wall. There was also a large umbilical hernia. On opening the abdomen the omentum was adherent, and was ligated in two portions. The intestine was wounded during the process, and the uterus was covered practically by the intestines, which were adherent to the womb. Difficulty was experienced in opening the uterus. This patient made a tedious recovery. The Fallopian tubes were ligated to prevent further conception.

The Treatment of Sepsis with Bacterial Vaccines.—HARTWELL, STREETER, and GREEN (*Surg., Gynec., and Obstet.*, September, 1909) report 24 cases of general infections, 22 of septic abdominal wounds, 41 of local sepsis, 6 of empyema, and 4 of osteomyelitis; in all 97, treated by bacterial vaccines. In the puerperal cases very decided improvement followed with the gradual recovery of the patients. Cultures from the uterus in these cases showed streptococci or pure cultures of staphylococci. In one case an abundant growth of pseudodiphtheria bacilli were present with slight growth of streptococci. Antistreptococcic serum was used with this patient without effect. She became profoundly septic, with a large indurated mass filling the left side of the pelvis. On the eighteenth day she was inoculated with 100,000,000 pseudodiphtheria vaccine, and three days later with 200,000,000. In the next thirty-six hours the temperature steadily fell to normal, and there was a marked improvement in the general condition. She received two further inoculations with continued improvement. The temperature fell to normal on the thirty-third day and the patient was discharged on the forty-first day. There was then a dense mass filling the left side of the pelvis to the base of the bladder, with a smaller mass on the right side.

Accidental Perforations of the Uterus and Vagina.—WETHERILL (*Surg., Gynec., and Obstet.*, September, 1909) describes the case of a patient suffering from subinvolution, in whom he used the ordinary Sims sharp curette, which perforated the uterus. The abdomen was opened and a valvular perforation, three-fourths of an inch in length, was found near the left cornu. This was closed with two fine sutures of plain catgut. Ventrosuspension was then performed, the patient making a good recovery. Wetherill adds two other cases in which the uterus was perforated by a curette, in one of which the small intestine was injured, necessitating the removal of six and one-half feet of the smaller bowel. These patients made good recoveries. Wetherill calls attention to Heineck's table, giving cases of perforation of the uterus with injury to the intestine, followed by resection of the bowel. Of these patients, 56 per cent. recovered and 44 per cent. died.

[The writer recently had occasion to operate upon a woman admitted to the Jefferson Maternity, giving a history of having tried to produce an abortion by the introduction of a glass catheter. The catheter had slipped and had gone farther than was intended, and the patient could not recover it. On admission, by vaginal examination, the catheter could be felt but could not be isolated, nor retained in any one position. An effort was made to open the posterior cul-de-sac and to grasp it, but this effort was unsuccessful. Twelve hours after admission I opened the abdomen, finding the catheter among the coils of intestine, and removed it without difficulty. There was no blood in the abdominal cavity, the uterus was slightly enlarged, evidently in very early pregnancy, and the point of entrance of the catheter could not be distinctly observed. There was, however, on the posterior wall of the uterus, where the lower uterine segment was forming, an area which, to the finger, gave evidence of marking the site of a perforation. The posterior vaginal fornix was freely incised and gauze drainage passed from above downward into the vagina. The uterus, tubes, and ovaries were inspected and brought into position, the gauze packing placed behind them, and the upper end of the gauze allowed to emerge through the lower end of the abdominal incision. Hot salt solution was poured into the abdominal cavity and the patient's shoulders raised to promote drainage. The gauze was gradually removed through the vagina, the patient making a good recovery.—E. P. D.]

Symphysiotomy.—Burns (*Surg., Gynec., and Obstet.*, September, 1909) reports the case of a multipara who had had two living children, the third dying soon after birth. The head did not engage, and an effort was made to bring it down with the obstetric forceps, but this proved unsuccessful. She was then taken to a hospital, where the cervix was incised, the patient placed in Walcher's position, and strong traction again made with the forceps, without success. The forceps was left on and symphysiotomy then performed and the head readily delivered with the forceps. The child gasped but could not be revived. There were no lacerations of the vagina, vulva, or bladder. The cervix was repaired with catgut, and the suprapubic incision with silkworm gut. The puerperal period was without fever, the patient having pain and tenderness in the pubes, hips and outer side of the left foot for the first ten days. The head of the bed was raised to favor vaginal drainage, and the patient was urged to lie upon the sides, which seemed to produce better apposition of the edges of the joint. The patient was discharged on the fifteenth day, in good condition. [While the reporter is to be congratulated upon the success of his symphysiotomy, his case illustrates the fact that the forceps should not be applied to the head unless the head engages and moulds in the brim of the pelvis. His symphysiotomy should have been done before the forceps was tried. Obstetric surgery will not reap its just reward until this mistake is eliminated from obstetric practice.—E. P. D.]

Differential Diagnosis Between the Hemolytic Streptococci.—Fromme (*Zentralbl. f. Gyn.*, 1909, No. 35) has pursued investigations with different sorts of nutritive material, using bouillon and lecithin to recognize

easily the more virulent forms of streptococci. He finds that by the use of these substances, employing a lecithin emulsion of 2 per cent., in from twenty-four to forty-eight hours, he can distinguish the more virulent sorts of streptococci. Obviously, any method which permits the prompt and accurate recognition of the most virulent germs must be of value.

GYNECOLOGY.

UNDER THE CHARGE OF

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Complete Restoration of Genital Function in Gynatresias by Abdominal Operation.—F. Cohn (*Zntralbl. f. Gynäk.*, 1908, xxxii, 1593) discusses the various methods of operation for the cure of gynatresia, and advocates the abdominal method as described by Pfannenstiel, which consists in the separation of the peritoneum between the bladder and the uterus, freeing of the posterior bladder wall down into the vaginal region; incision of the uterus and opening of the cervical canal, and suture to the opening made in the vagina. The last step may be completed by suture per vaginam. Cohn reports a case operated upon by this method in which impregnation and delivery of a healthy child occurred about seven years after the operation. He states that the ideal aim of every abdominal operation for atresia should be the restoration of communication between the vagina and the uterine cavity so as not only to insure undisturbed discharge of the menstrual blood, but in addition effect the possibility of birth by the natural passages.

Amputation of the Uterus in the Corpus to Preserve the Menstrual Function.—H. A. Kelly (*Amer. Jour. Obst.*, 1909, lix, 570) says in certain cases in which amputation of the uterus has to be done, and the conditions admit, it is desirable to preserve the menstrual function. He recommends two methods of procedure to that end: horizontal and vertical resections, and describes the technique of each. He reports nine typical cases in illustration.

Contributions to the Clinic of Gynatresia.—Emanuel Gross (*Ztschr. f. Geburtsh. u. Gynäk.*, 1909, lxiv, 70) reviews the literature on gynatresia, and as the result of personal observations arrives at the conclusions: (1) The Nagel-Veit theory of the evolution of deep gynatresias does not apply to all cases; the inferior end of the vagina arising from the sinus urogenitalis may be absent, although the remaining portion of the genital tube may be well developed (Case I); on the other hand, the upper portion of the vagina may be absent, while the lower third and the hymen are intact (Case II). (2) Hematosalpinges may occur in congenital deep occlusion of the single or a double genital tube. The adnexa of a

uterus unicornis, with total absence of the pars mullerica of the vagina may give rise to inflammatory symptoms. (3) The main danger in hematosalpinx lies not in rupture and primary infection in opening the atresic vagina, but in ascending secondary infection. The presence of tubal sacs is by no means to be treated lightly, as Nagel still does, but should be emptied or removed at once. That a cure with retention of menstruation may follow secondary pyosalpingitis was demonstrated by one of Gross' cases. (4) In atresia acquired during childhood there may occur later pyocolpos formation as the result of beginning sexual activity without preceding hematosalpinx. This was clearly shown in Case III, in which imperfect development of the uterus bicornis and permanent amenorrhœa existed.

The Choice of Time for Operation for Pelvic Inflammation of Tubal Origin.—In considering the choice of time for operation for pelvic inflammation of tubal origin, SIMPSON (*Surg., Gyn., and Obst.*, 1909, viii, 45) says several important and suggestive questions arise: (1) Will operation always be necessary for complete restoration of health, comfort, and functional activity? (2) If operation is decided upon, will the coincident occurrence of acute illness and operation entail more or less danger than their separate occurrence? (3) If interval operation is decided on, by what means may we determine that a safe time has been reached? The first of these Simpson answers in the negative, mentioning types of conditions that recover with non-surgical treatment. To the second question Simpson replies by stating more danger will be entailed by operating during the acuteness of the attack. Simpson answers the third question as follows: (1) The patient shall have recovered from her acute illness and shall have regained a satisfactory margin of reserve strength. (2) The temperature shall not have arisen above normal a single time for a minimum of three weeks. (3) The inflammatory exudate surrounding the focus of infection shall have been completely absorbed. (4) There shall have been no marked or persistent rise of temperature following a careful bimanual examination. These rules will permit of a mathematically exact selection of a safe time for operation.

The Involuntary Muscle Fibers of the Pelvic Floor.—STUDDIFORD (*Amer. Jour. Obst.*, 1909, lx, 23) states that on microscopic study of the tissues lying between the halves of the levator ani and posterior to the anterior end of the external sphincter in the perineal body, a large amount of involuntary muscular tissue will be found. It is in close relation to both the levator and the sphincter, some of the bands being attached to the fascia covering the upper ends of the sphincter, and running in a longitudinal direction, while other bands are connected with the fascia covering the levator on either side, and are arranged to pass transversely. It is claimed that no fibers of the levator pass between the rectum and vagina, and Studdiford believes the involuntary muscular tissue mentioned pulls the halves of the levator together in the perineum, thus assisting in the supporting function of that muscle, and particularly when special strain is put upon the perineal structures. Upon this anatomical study Studdiford has constructed a new perineorrhaphy that is used by H. C. Coe.

Indications for Operation in Chronic Inflammatory Adnexal Disease.—Prochownick (*Monatsschr. f. Geburtsh. u. Gynäk.*, February, 1909, 174) summarizes the deductions from his experience as follows: (1) In severe cases resisting all treatment operation should be done after careful observation for five or six weeks, provided tuberculosis is not present as a complicating factor. (2) In women who show no objective signs of improvement after prolonged conservative hospital treatment, and who readily suffer recurrences, operation is to be advised. Only when prospects of abscess drainage obtain should repeated conservative measures be employed. Early intervention in such cases frequently effect conservation of organs and of function. (3) Early operation is indicated in all unilateral tumors if association with appendicitis is apparent or strongly suspected. (4) In concurrence of tuberculosis with other infections—particularly gonorrhœa—as well as tuberculosis alone, operation is indicated only in severe local disease when the lungs and urinary organs are not involved, or only slightly so. The operation should always be radical. (5) Permanent fistulous tumors, particularly suppurative adnexal tumors connected with the intestine, should be operated upon as early and as radically as possible if not accessible for extensive drainage extraperitoneally.

Wertheim's Panhysterectomy for Carcinoma of the Cervix.—Berkeley (*Jour. Obst. and Gyn. Brit. Emp.*, 1909, xv, 145) discusses the various steps in surgical procedures which were worked out by various surgeons and culminated in the Wertheim operation for extirpation of cancer of the cervix uteri. This discussion is followed by a resume of the collected cases of this operation done in Great Britain. Berkeley regards Wertheim's operation as productive of a greater ultimate curative effect than all others for cancer of the cervix, and exhibited a clamp known as the Berkeley-Bennie clamp for clamping the vagina by the plan of Wertheim. The various phases of the operation—complications, bladder and ureteral injuries—causes of death, and mortality are each in turn carefully considered.

Dry Heat as a Therapeutic Factor in Gynecology.—Gellhorn (*Amer. Jour. Obst.*, 1909, lx, 31), after discussing the therapeusis of heat, refers especially to dry heat, which he recommends in three classes of cases: (1) Chronic exudative processes within the pelvis; (2) certain menstrual disorders; and (3) a number of postoperative conditions and complications. The principal indication for the employment of dry heat is parametric and perimetric exudates. These, when the acute symptoms have passed and absorption has been but partial, may be successfully treated by dry heat, a special apparatus for which Gellhorn recommends as a result of his experience with it. Due warning is given against relaxation in carefulness in the applications of this agent.

Suture of Recent Perineal Tears.—Sigwart (*Zentralbl. f. Gynäk.*, 1909, xxxiii, 329) states that Mayer recently reported 30 cases of perineal rupture in which the perineal surface was sutured by means of Michel's staples. Healing by first intention occurred in 27 cases; in 3 cases granulation occurred at the site of the perineal and the vaginal surface

union. Sigwart sutured 125 cases of perineal rupture with Michel's staples; only two failed to heal entirely by first intention. The staples are placed along the perineal surface and deep catgut sutures along the vaginal lesion. Removal of the staples is simple and painless by employing a forceps, one jaw of which contains a hollow groove, into which the convex jaw fits. The hollow jaw is placed under the staple, and by compressing the handles the ends of the staples are separated and release the tissues.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

UNDER THE CHARGE OF
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Regeneration of a Vocal Cord after Total Extirpation for Cancer.—Lannois and Durand (*Revue hebd. de laryng., d'otol., et de rhin.*, January 9, 1909) report a case of a man, aged sixty-four years, from whom the one vocal cord was removed in its entire ligamentous extent and who was able to speak eight or ten days after the intervention. They were able to watch a progressive reproduction of a new vocal cord so well that less than two months after ablation, interesting stereoscopic photographs could be presented to the French Society of Oto-rhino-laryngology. The new cord was a little smaller than that of the other side, and a little less mobile, but it had the aspect of a true vocal cord and fulfilled its functions in respiration and in phonation.

Intranasal Tuberculin Reaction.—Lafite-Dupont and Moulinier (*Ann. des maladies de l'oreille, du larynx, du nez, et du pharynx*, May, 1909) describe their procedure for producing a diagnostic reaction of tuberculin on the nasal mucous membrane. An exudate ensues which desiccates and forms a yellowish crust reposing upon a congested mucous membrane, of which the extravasated red corpuscles cover the crusts with little hematic points. The experimentation was made upon one hundred patients, taken at random, and a reaction occurred only in tuberculous subjects. The method is claimed to be as certain as the ophthalmic reaction, and has the advantage over the former of being innocuous.

Epithelioma of the Rhinopharynx.—Dupond (*Revue hebd. de laryng., d'otol., et de rhin.*, January 2, 1909) reports a case of fungous epithelioma in a man aged fifty-eight years, who had complained for sometime with obstruction in the back part of his throat and impairment of hearing in the left ear. He appeared robust and constitutionally strong, and had never had any serious illness. The fungous growth was found completely filling the left side of the rhinopharynx and obstructing the left choana, the Eustachian outlet of the same side, and extending

down the pharynx to the posterior part of the tonsil. All surgical interference being contra-indicated, treatment was confined to topical applications with the tincture of thuja and of celandine, under which an enlarged parotid lymphatic gland diminished in size, while a similar carotid enlargement increased in volume. The patient was kept comfortable for a considerable time with improvements and relapses, and finally succumbed to one of the complications common to malignant rhinopharyngeal growths.

A New Pharyngoscope.—Utilizing the refraction of the prism, HAROLD HAYS (*Laryngoscope*, July, 1909) describes a new laryngoscope modelled on the electrocystoscope. The tube carrying the lens passes through a horizontal shaft used as a tongue depressor, at the distal extremity of which are two electric lamps which illuminate the pharynx and prevent clouding of the prism. By throwing the prism in the proper directions, the pharynx, posterior nares, Eustachian outlets, epiglottis, and larynx can be more or less satisfactorily examined. Instruments passed through the nasal passages such as Eustachian catheters, for instance, can be manipulated under control of the image and seen in the pharyngoscope. The apparatus is much easier of manipulation than the laryngoscopic mirror.

Lipoma of the Larynx.—GOLDSTEIN (*Laryngoscope*, September, 1909) reports a case of his own of which a careful study was made before and after operation, and reproduces the records of twelve cases recorded by previous writers, illustrations accompanying the records in several instances. His own case was a double lipoma occurring in a married woman, aged thirty-three years. A globular growth occupying nearly the entire lumen of the larynx was removed with the cold snare, revealing a second growth beneath. In an attempt to remove this growth with the snare it became impossible either to cut through the growth or to disengage the wire. The entire mass, snare, tumor, and larynx, was pulled forcibly upward and forward into the cavity of the pharynx, and then the pedicle of the growth was cut through with a pair of long, curved scissors. By microscopic examination the growth was verified as a typical lipoma.

Laryngostomy.—JACKSON (*Laryngoscope*, September, 1909) describes and depicts the technique of the operation as now performed by him after a personal experience of seven patients, the last five of which are reported in the paper. Six were cured, and one still remains under treatment, the duration of treatment having varied from five months to three years.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF
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The Pathogenesis of Rabies.—Among the curious phenomena of rabies are the great length of incubation period (two weeks to two years) and the fact that only a small percentage of people bitten by rabid animals develop the disease (probably less than 10 per cent.), and yet when once developed, the outcome is invariably fatal. In this connection PALTAUF (*Wien. klin. Woch.*, 1909, xxii, No. 29) observed that the medullas of four people bitten by mad dogs, but dying of an intercurrent disease, produced rabies in rabbits, though no signs of rabies were apparent in the patients at any time. Two died of delirium tremens, one of pulmonary embolus, the fourth of encephalomalacia. All had been bitten within three weeks, and had received less than six injections of antirabic serum. In the case of four other people, bitten by rabid dogs, and dying of an intercurrent disease, who, however had undergone a complete Pasteur treatment, the injection of the medulla into rabbits was negative. From these observations, Paltauf deduces that the virus of rabies in the human organism is for the most part gradually weakened and destroyed without the formation of rabicidal bodies, so that in only 6 to 10 per cent. of the cases does it survive and cause symptoms. In each case, however, the patient undergoes a latent form of the disease, and their organism contains the etiological factor, whatever that may be. In the second group, however, after the immunization from the Pasteur treatment, rabicidal bodies are developed in the human organism and the infectious agent is actually obliterated.

Changes in the Hypophysis Cerebri during Pregnancy.—The morphological changes in the anterior lobe of the hypophysis during pregnancy, first observed by Comte in 1898, are discussed in great detail by ERDHEIM and STUMME (*Ziegler's Beiträge*, 1909, lxiv, 1) in 126 pages with fifteen excellent illustrations. In the normal histology of the anterior lobe, they recognize three kinds of cells: (1) The eosinophilic, which are the most numerous and usually occur in the outer circle of the alveolus; (2) the basophilic, which with the eosinophilic constitute the chromophile cells; (3) and least numerous, the chief or chromophobe cells, usually lying in the centre of the alveolus. In pregnancy, these latter cells increase enormously, so that the chromophile cells, which remain the same, are completely overshadowed. They also change in character from round nuclei, with very scanty, ill-defined protoplasm, to large, pale, oblong nuclei, surrounded by considerable, well-staining protoplasm that occasionally contains vacuoles. In this characteristic form, called "pregnancy cell," they remain until the puerperium, when

they undergo involution and either disappear or return (by vacuolization of the protoplasm) to the original chief cell. These, however, remain more numerous than at first, so that the hypophysis of a multipara can be distinguished by an experienced observer from that of a nullipara by histological examination. The whole gland, during pregnancy, increases in size (posterior lobe remaining unchanged), the main growth being toward the side, as the anteroposterior diameter is limited by the dorsum sellæ. The average weight of a nulliparous gland was found to be 61.8 cgm., of a primiparous, 84.7 cgm.; of a multiparous, 106 cgm. Clinically, some of the symptoms of pregnancy may be explained by these changes. The bloated face, thick lips, and enlarged hands (compare with akromegaly) may be due to the same cause, that is, hypersecretion of the hypophysis.

In this connection, a case reported by von Reuss is most interesting (*Wien. klin. Woch.*, 1908, No. 31). Bitemporal hemianopsia occurring during pregnancy, and disappearing during the puerperium, was thought to have been caused by the pressure of an enlarged hypophysis upon the optic chiasm.

The Influence of Cardiac Insufficiency on the Disposition of the Blood in the Organs.—H. C. Thatcher (*Deut. Archiv f. klin. Med.*, 1909, vol. xcvii) has possibly explained the rather paradoxical findings of Lowi, who showed that in states of cardiac decompensation, when the venous system was greatly engorged, the arterial pressure might even be raised. Thatcher's experiments consisted in determining the changes of volume of the various organs by means of an oncometer when sudden cardiac insufficiency was produced. This condition was produced by means of a small balloon introduced through the right jugular vein in a collapsed condition into the right auricle or inferior vena cava. By sudden dilatation of the balloon almost complete block of the returning venous blood was effected. Enormous engorgement of the liver at once followed. The liver was found capable of distending to such a degree that an amount of blood, amounting to one-third to one-fourth of its own weight, could be accommodated. The brain also showed a considerable increase in volume, but not so marked as the liver. The spleen, intestines, kidneys, and extremities, on the other hand, all showed a distinct decrease in volume. More complete investigation of the kidney indicated that with this organ the decrease in size was due to a contraction of the arteries, following the arterial anemia resulting from the venous block. This arterial contraction was so great that notwithstanding the venous engorgement of the organ its volume was diminished. The brain and liver, organs which are well known to have weak vasomotor mechanisms, followed the curve of the venous pressure; whereas, the kidney, spleen, intestines, and extremities, with their active vasomotor apparatus, adapted themselves to the sudden arterial anemia by means of powerful vasoconstriction. The nervous control of this action, Thatcher believes to be largely central, because when the nerves of one kidney were cut the organ did not contract as markedly as its fellow. Blood pressure curves were made with all the experiments, and were of great interest. With the first shock of shutting the right heart the systemic pressure fell abruptly.

But very soon it rose rather rapidly, and reached a level somewhat lower than the original. This was held as long as the right heart was blocked. Evidently, then, the organs with powerful vasomotor mechanism, by their arterial constriction, served to bring up the fallen pressure and maintain it at a life-sustaining level. In conclusion, Thatcher points out that the artificial nature of the lesion does not similate absolutely the acute cardiac insufficiency of man, nor does it have any bearing on the chronic venous congestion of old weak hearts. But his experiments indicate what an important role the vasomotor mechanism plays in the pathology of the circulation.

Hypophysis Tumor and its Bearing on Akromegaly.—The theory of Tamburini and Benda, that akromegaly is caused by a hypersecretion of the hypophysis, as shown by an unusually large number of chromophile cells, receives important support from a case reported by Erdheim (*Ziegler's Beiträge*, 1909, lxiv, 233). In a case of akromegaly of ten years' duration, no enlargement of the hypophysis was found at autopsy, but instead, a tumor composed of tissue identical with the chromophile cells of the anterior lobe of the hypophysis, and occupying the body of the sphenoid bone, immediately beneath the sella turcica. The nature of the tumor is explained in the following manner: When the "anlage" of the anterior lobe of the hypophysis separates from the pharyngeal mucous membrane, it ascends through what will later be the sphenoid bone to join the posterior lobe in the sella turcica. The canal thus formed not only sometimes persists (canalis craniopharyngeus), but always contains small remnants of hypophyseal tissue. This is most thoroughly worked out by Haberfeld in the same number "Ueber Rachendach Hypophyse" (*Ziegler's Beiträge*, 1909, lxiv, 133). The tumor described has obviously sprung from one of these remnants, accommodating itself by forming a cavity in the centre of the sphenoid, and leaving the hypophysis unchanged. As it was composed almost entirely of typical chromophile cells, the occurrence of akromegaly is amply accounted for. At the pharyngeal end of the canalis craniopharyngeus, that is, the original "anlage" of the anterior lobe, Erdheim had previously found in fœtuses a small mass of tissue identical with the anterior lobe of the hypophysis. Haberfeld now finds this to be constant in all ages, and adds the latin name—hypophysis pharyngea—to Erdheim's Rachendach Hypophyse. It may well be that many of the rare cases of akromegaly without hypophysis tumor could be explained by an unobserved tumor in one of these two places.

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ORIGINAL ARTICLES.

THE EMMANUEL MOVEMENT.

ITS PRETENSIONS; ITS PRACTICE; ITS DANGERS.

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Even before the Emmanuel movement began, the doctors had been hearing so much about mental healing that we were growing a little tired of being instructed and exhorted by the laity; then came the flood of literature let loose by the new agitation, and our interest was renewed until we discovered that we were only being offered an old, well-tried, and respectable remedy (with a few new adornments, to be sure) under a novel and flashy name—psycho-therapy—soul-treatment! It looked a little like Christian Science, it sounded rather like "mind-cure," it had some elements of the "faith-cure," but every combination of psyche and psycho was fashionable; it seized the fancy, and here it is, so well established a phrase that one must not waste time on it except to cast a doubt on its scientific accuracy.

Let us say at once that there can be no question that the Emmanuel movement has done good, and that in the hands of cool-headed men of wide knowledge of the world, deep versed in human nature, and possessed of an unusual combination of caution, courage, and acute judgment, it may do more. Its important service in the end will be to have called attention anew to the fact, already always and everywhere held and taught by thoughtful doctors, that the mind and the body cannot be treated independently of one another.

Some of the more direct and immediate benefits received by—are they to be called its patients?—we have heard of. There are yet certain doubts to be expressed on the statistics of these cures. Let us admit them for the present in evidence and agree that much has been accomplished. Even then we ought not to approve, as a whole, a method which proposes that the clergyman shall call in the physician, not to direct or even aid in the treatment of the case, but to relieve him of some responsibility by settling whether the sufferer shall be turned over to the spiritual arm. We hold our profession as seriously in honor as the clergy do theirs, and it is not asking too much that they should admit that both callings have in this discussion one object in view: the welfare of those whose health, whose happiness—yes—whose souls are in our hands. There need be, there should be, no jealousy—but the tone used by some of the supporters of the Emmanuel movement toward our profession will bear improvement. It is childish for them nowadays to rake up ancient slurs on physicians, to talk about doctors being materialists or atheists, and such medieval gibes as should have been long barred by a statute of limitation on old jokes that never were good ones.

This is no place for consideration of whether doctors are mostly good Christians or religious men, but it may be safely asserted that "idealist" rather than "materialist" would be the word fitly to describe the largest number of doctors to-day Not very long ago Miss Cobbe complained, in an article published in England, that the doctors "were setting up a new priesthood which was to replace the care of the soul by the care of the body." But since some of the clerical champions of soul-healing seem in their zeal almost to have forgotten that souls have bodies, they may well be reminded that the opinion of the best men of our profession has ever been that he was but a poor physician, a mere mender of broken bodies, who was not concerned for the spirit as well as for the flesh. Most earnestly should we insist that the *treatment* of a patient, whether it be surgical, medical, or psychic, should, for the safety of the public, be in the hands of the doctor.

A physician's first duty is to cure his patient. We who are doctors belong to no school, owe allegiance to no medical sect, admit no creed which binds us to a limited system. We stand on one principle and one only. We will use any honest means to heal our patients. The wisdom of the ancients, the science of the moderns the art of the practitioner—we use them all with such minds as we have and such skill as we can acquire by painful years of study and laborious work. Our professional hospitality has turned no man away who offered us any means to this end, whether he were a Jesuit missionary bringing cinchona bark from South America, or an African voodoo-man with an ordeal poison. We listen, we try, we adopt or lay aside. Always the best of our calling—and it is by these we may claim to be judged—have been willing to be taught by

anyone. Of late years the medical man, far from being a reactionary, has been almost too ready to accept the new, often receiving half-fledged and doubtful remedies rather on faith than by works. It cannot, therefore, be said that opposition to the new movement is opposition to an innovation because it is an innovation. Rather is it opposition to the form of *re*novation of an old heresy. What is new in the Emmanuel movement has been forced upon it by medical criticism after a years' exploitation of the plan: that the doctor shall be called in to decide whether he shall gracefully retire and hand over the patient to the—shall we say unlicensed?—practitioner. What is old is that an appeal to the best in man, confidence in the goodness of God, the stimulation of the patient's self-helpfulness, courage and faith, are useful means of aiding those who are in trouble, sickness, or any other adversity. The thoughtful physician has used these means since there were physicians, since the days, in fact, when the functions of the priest and the medicine-man were differentiated. Before that time pure psychotherapy was practised by the ecclesiastical authorities, shaman, voodoo-man, or witch-doctor, who put feathers in their hair, painted their faces, and beat tom-toms to scare away the evil spirits that produced the illness. Further development brought the medical man out of the medicine-man and gave up the treatment of disease by these wholly spiritual and suggestive means as unwise. How far are we to return on our steps now? and shall the specialized physician be replaced again by his primeval ancestor, the combined priest and medicine-man?

Our clerical critics disclaim any intention of finding fault with physicians, and then explain that they only mean that doctors are materialists and materialistically taught, imperfectly educated, and not acquainted with modern science; fifty years behind Europe, and quite unequal to the task of healing nervous patients, which is to be assumed by the ministers, who presumably are fifty years ahead of Europe,[1] in which benighted land treatment of the sick is still strictly a duty of the physicians.

It can scarcely be seriously contended that the clergy in general are better fitted to undertake the cure of mental disease than the doctors in general. I desire to speak of the ministers as I regard them, with every respect for the men, and more for the office. Nevertheless, since we are frankly criticising one another, I must say that in my experience the minister when called in to perform his recognized and desirable functions in aid of patients in spiritual

[1] The Rev. Lyman P. Powell, far the coolest and most level-headed of the Emmanuelists, and author of a book which has some semblance of the scientific and statistical method so lacking in the "official" publications, said at the Episcopal Congress last May that "physicians in America were fifty years behind Europe in this regard" (psychotherapy). "Its success depended on no theory of the subconscious; it put the responsibility on the physician;" exactly—the responsibility, but neither the authority nor the credit for good results. (Reported in The Churchman, May 22, 1909.)

need, has not often been so helpful as one would expect or hope. Of course, we are speaking of his functions as pastor and priest, not as a psychic assistant. It has seemed sometimes as if he were unable to comprehend that the minds of such patients work in contradictory and perverse fashion, and could not be judged by normal standards, or that he must distinguish the normal disturbances due to religious doubts from the self-accusatory mental attitudes of persons ridden by fixed ideas of sin or mere unformulated vague indecisiveness. Even these two latter troubles, though frequently they are heralds of serious mental or brain diseases, may be helped to some extent by strong, simple authoritative reassurance and reenforcement of the courage. It is not from want of good-will that the minister is of so small helpfulness in these instances, but sometimes from want of knowledge of the world of men, and oftener rom inexperience in mental disorders and from lack of training—exactly the defects charged to the medical profession. One does not speak of the failures to help where we all may fail, namely, in the cases of those patients who, whether they consult a minister or a doctor, are seeking rather a novel emotional stimulant than the real help which comes only through stern discipline and self-control, two measures very unpopular among neurotics. The causes of this imperfect comprehension and consequent inability to help lie not only in the absence of training in the study of abnormal mental states and, without disrespect be it spoken, the credulity with which the patients' overaccented statements are usually received by the inexperienced, but also in another important and rather contradictory fact—the notion, no less widely held by the clerical profession than by the laity, and not unknown even among medical men, that nervousness in almost any form or expression is an "imaginary" ailment. This attitude on the part of the adviser from whom the patient has every right to anticipate understanding, consolation, and spiritual strengthening has frequently unfortunate results: if the too easy belief give the prevailing tone, the patient, delighted with the prospect of a new and sympathetic auditor, goes on from exaggeration to exaggeration, or if his case be one of genuine need of spiritual comfort, and he finds his trouble regarded as imaginary, his depression is deepened and his spiritual helplessness intensified by the feeling that the one of all others whom he has looked to for comprehension does not understand his state. The authors of *Religion and Medicine* have admitted some of these deficiencies and pointed out themselves the much greater success with which the Roman Catholic priest handles such difficulties, though they attribute this success to the confessional, which is probably only one factor, and neglect to mention his elaborate training, the intimate personal character of his relations with his congregation, and his more absolute authority, as other important elements.

Whatever the shortcomings of the doctor in psychological knowl-

edge may be, the shortcomings of the minister at present are no less, and the latter not only wants the practical experience which serves to guide the physician in these dangerous waters, but may wreck the ship if, instead of the lamp of knowledge, he steers by the unsteady light of emotional sympathy. It seems improbable in such matters as these, involving judgment of character, knowledge of men, and acquaintance with the tortuous workings of the neurotic mind, that a few lectures in a seminary course will enable the average divinity student to do better than his far longer study and more practical contact with men and minds will the medical graduate, assuming both equally equipped in the beginning with the necessary human comprehension and with the gift of sympathy in its best sense.

Nor is it only against overdoses or misapplications of sympathy that the minister who would help the nervous must be cautioned. More serious ills may follow if the use of religious emotion as a motive or stimulant is pushed to excess. It should be remembered that emotional instability is characteristic of many and various nervous disorders, and emotional overstrain among the commoner causes of not a few of them. Care must be used, then, in any appeal to religious feeling, with constant recollection that the force may be a terribly dangerous one to use, and may increase or even create that very overexcitable condition which should be lessened or prevented. Dreadful moral disasters have resulted because a clergyman could not see that religious emotion, a good thing in itself, might prove too strong a medicine for a moral constitution already weakened by emotional excess, or that there were degrees in its applicability and must be graduation in its doses. A thousand times more strongly should this be said of the uses of suggestion as a treatment, since oversuggestibility is in itself a diseased condition, and every use of suggestion (that is, hypnotism), however mild, increases the suggestibility just as cultivation increases the fertility of the soil, so that chance sown seeds of evil suggestion take ready root in ground prepared to receive them, and grow as bad habits are apt to do, even more strongly than good ones.

There is another question raised by the mention a little while ago of the unlicensed practitioner. The man who treats patients by the light of nature we put in jail for the protection of the public. The State holds even the graduate and experienced physician legally responsible, liable to be amerced in damages should he by a mistaken diagnosis or from want of a reasonable sufficiency of knowledge treat his patient wrongly, or confound one disease with another. We have had no suggestion as to how the minister will stand in this respect. Assuredly the time will come when a judicial definition of his responsibility and of his clinical capacity will be demanded.

From what has been said and written it appears that the great field of usefulness of the movement is to be in "functional nervous diseases." The victims of these are to be committed, soul and body, to the clergy. This is the present conservative claim, but certain signs indicate that they will soon take all diseases for their province and omniscience for their foible, as has already happened in the English "Emmanuel movement."

Now what are "functional" nervous diseases? You and I are uncertain, but then we are students and scientific workers. There is no gap in the ministerial knowledge, no hesitation. The authors of *Religion and Medicine* appear to regard any disease as functional that is not accompanied by external evidence of alteration of structure as obvious as a broken bone. This, of course, is not their definition. Indeed, in this matter of diagnosis, as in some others, they avoid definiteness. We know that almost every year new knowledge, closer observation, takes one or two diseases out of the functional list and puts them in the organic class. Moreover, even at the risk of being charged with materialism, one may be permitted to express doubt if there is *any* purely functional disease. Pain in the stomach represents a disturbance of function, but also, according to its cause, hyperemia, anemia, deficient or excessive secretion, etc. The most typical of the functional nervous diseases is neurasthenia, which is also indiscriminately called nervous collapse, breakdown, exhaustion, and, in the forms chiefly affecting the mental side, psychasthenia. We describe this whole class of disorders inclusively as being fatigue neuroses. Is fatigue a functional or an organic symptom? Organic, most certainly. Muscle fatigue is due to poisoning of muscle by waste products of energy. Nervous fatigue is due to changes in the ganglion cells distinctly visible under the microscope. Besides, the diagnostic distinction between organic and functional nervous disease is a convenient rather than a fundamental one. Take again this much discussed prototype of the functional troubles, neurasthenia; many of us think it absolutely due to disturbance of nutrition in the nervous system; that is, in short, to some form of localized tissue starvation dependent upon a great variety of climatic, occupational, and other causes.

The evidence of this is plentiful, clinical and physiological, but too technical for brief statement here. The best wits of the most expert clinicians often find it difficult to decide whether a disorder is functional or organic, even on repeated examination. Still more complicated is the question when the patient is the victim of both functional and organic disease—a combination which may test the acuteness of the best diagnostician and utterly baffle one less well trained and experienced, and might demand not one but a dozen examinations for final judgment.

It is scarcely worth while to waste time on a discussion of the

refinements of diagnosis with an author who says: "All the functional neuroses are to be regarded as diseases of the subconscious mind. By recognizing this we simplify diagnosis."[2] We do, indeed! Diagnosis is a part of medicine just as much as therapy is—and psychotherapy is but one part of general treatment. One main cause of disagreement with the movement I am discussing is the overinsistence upon mental methods to the neglect of physical ones. The possible need of physical treatment is grudgingly admitted, but one might read all their publications and conclude at the end that physical methods were seldom or never required. One author, the Rev. Dr. Batten, in an article on "Psychotherapy," has extracted instances of psychotherapeutic treatment from the Old Testament. Among others, he cites the fact that the witch of Endor gave Saul food, to compose his soul. The witch (even a doctor may read the Bible) had just announced his approaching death to Saul, so that he may have been in need of suggestive therapeutics, but a few verses further on comes the explanation: "There was no strength in him, for he had eaten no bread all the day nor all the night," and the woman begs him to "eat that thou mayest have strength when thou goest on thy way." But one should beg pardon of Dr. Worcester, who, however one may differ with him, must be taken seriously, for speaking of his arguments in the same paragraph with those of Dr. Batten. The latter may be left to the tender mercies of Dr. Joseph Collins, who has handled him and his circular letter with a bitter playfulness which it is to be feared was wasted on a critic whose sense of humor must be as ill developed as his faculty for criticism.

Certain other peculiarities and contradictions should be mentioned before leaving the question of diagnosis, since that involves the selection of cases for treatment. One is that, in spite of much undigested assertion about hysteria and Myers' theory of the subconscious mind, neurasthenia and hysteria are constantly confused. We all know how they run into one another—and how troublesome the borderland cases are—but how can it be asserted by anyone with knowledge that "In almost all such cases (functional neuroses) the real cause of the disorder is moral or psychical?"[3] This would be interesting and valuable, if true, but it is not true. In a vast majority of such cases the real causing cause is physical—and the psychic difficulty is a later addition. It is probably also true that in many of these cases the physical inheritance is bad—not in the sense of inherited disease but in providing a suitable soil for the growth of neurotic troubles and in giving an ill-balanced or oversusceptible nervous system. For a single example of physical cause of neurasthenia take a railroad shock. A man in perfect condition is badly shaken up in a train wreck, with slight external injuries. In two

[2] Religion and Medicine, p. 112. [3] Ibid., p. 48

or three days he goes all to pieces nervously and remains so perhaps for many months—to be called weakminded by his friends and accused of simulation by the railroad. It has been frequently proved that if after such a shock the victim is kept quiet, body and mind, for two or three weeks, the bad effects are minimized or entirely prevented. Often a person seriously injured in such an accident escapes altogether the nervous shock—because of the enforced rest required for the treatment of his wounds.

"Psychical disorders give rise to . . . physical disturbances . . . and these may require physical . . . treatment." Many times oftener, probably, the reverse takes place and physical disorders give rise to psychical disturbances. Dr. Weir Mitchell, who is quoted a good deal in *Religion and Medicine*, has advised in *Fat and Blood* and other works on the treatment of nervous diseases, that physical treatment should *precede* moral in order that the latter may be more effectual. It is only in trifling cases of neurasthenia that moral suasion, suggestion, explanation can make a complete cure. When extreme anemia, digestive feebleness, absence of appetite, numberless pains, constant fatigue, asthenopia, and the rest of the endless catalogue of ills and aches are found, the inability of the patient to concentrate attention is often so great, and the mind so distracted and confused as to render it inaccessible to useful or lasting improvement by suggestion. In such conditions suggestion may temporarily cover up the symptoms, but it does not reach the disease. This sort of success is in the eyes of its practitioners the justification for its use, and in those of sober critics a grave argument against it. To cover or to mitigate the more conspicuous and annoying symptoms results in a neglect of the physical states which are at their roots; for example, if, as so good and sane an authority as Dr. Münsterberg asserts, the anemia accompanying neurasthenia is bettered by psychotherapy, it is probably a temporary stimulation in blood distribution that makes this seem to be so, and a careful cellular count would show a deficiency in the blood elements still present, a deficiency which for permanent improvement will need such profound alteration as iron, ample food, and massage will bring about; and so with the other symptoms—the headache, whether due to anemia, to constipation, or to eyestrain, will only be finally better when the causes are removed, not when it is bettered as a glass of wine betters it until the passing effect disappears. In hysteria, on the other hand, miraculous cures may be wrought almost instantaneously by suggestion, no matter what we call it, and thousands of such miracles have been reported by doctors and many more thousands gone unrecorded, partly because these "cures" are prone to relapse.

The experienced neurologist who has seen many cases of nervous break-down in any of its forms not hysterical will agree that cases suitable for purely mental treatment are few and far between. In discussing this subject with Dr. Weir Mitchell last winter,

case records for the previous eighteen months were examined to settle for our own satisfaction how many cases in a large practice would have been suitable for such purely mental therapy. The conclusion was that *three* of our patients might have been so handled with success, but that the result of combined treatment even in these cases was, in all probability, both more rapid in action and more permanent in effect.

The neurologist sees daily patients with organic disease in whom the nervousness has been so conspicuous that the general practitioner has been deceived, stuck on his tag of "neurasthenia," and left it to the neurologist to discover that the patient had structural disease, such as Bright's disease or cancer—or often enough incipient insanity in some form. The delay of a few weeks for the treatment of these diseases by suggestion, by exhortation, by enthusiasm without knowledge, may render impossible their ultimate cure. Is it probable the examination will be more careful, the diagnosis more accurate, when the question is only whether the doctor shall give up the case altogether?

But, of course, the final result is that if there are mistakes they will be the doctor's fault, and if successes, they will be the clergyman's virtue! We Americans have always been like that, and I suppose we shall be until we reach a higher level of civilization. We boast of our education, but it has never gone deep enough to uproot a superstition in favor of amateurs. One has known plenty of educated people who would not consider the opinion of a surgeon if they could get that of a member of a family of hereditary bone-setters. We have insisted in several wars that a saloon keeper would make a better general than a West Point graduate—an opinion which we backed with our best blood. We consume vast quantities of patent medicine, we offer the chief field of the world to quacks, medical, spiritual, and commercial, we hinder every effort at raising professional standards—because we always feel sure that a trained man is never so good at his job as an untrained one.

In Dr. Worcester's latest pronunciamento[4] he forgives his critics beforehand, but has "noted with satisfaction that no radical criticism of our work has proceeded from a man who has studied it at first hand." If by this, as one must suppose from the context, he means that no one is competent to criticise the Emmanuel performances who has not been in actual contact with them, he lays an impossible condition upon his critics, and they might easily ask if this rule would not work both ways and apply equally to the Emmanuel criticism of physicians. How many years' service in hospital must a minister have before he can criticise the practice of medicine? Without comment, it will be enough to set down a few names of those who have adversely criticised the new plan

[4] Century, July, 1909.

and leave to the reader to decide if they are men able to form opinions from the vast and discursive literature of the subject. These publications are presumably meant to enlighten our ignorance, but if we cannot learn enough from them to form views, and must make pilgrimages to the original shrine to see suggestion and hypnotism properly carried out, Dr. Worcester is laying on our shoulders heavy burdens and grievous to be borne.

Conspicuous among these critics, then, stands Dr. Weir Mitchell, who expressed himself upon the subject before the American Neurological Association in 1908. On that occasion Drs. Mills and Dercum, of Philadelphia, agreed with his views, as did Drs. Sachs and Spitzka, of New York, and Putnam, Taylor, and Knapp, of Boston. Of these gentlemen, several have since published detailed and precise criticisms, as have Collins, Hamilton, and Darlington, of New York, Hun, of Albany, and Benedict, of Buffalo—a fair array of names of authority in neurology and in general medicine. Besides these, some psychologists have been unwilling to accept the theories or methods of the clerical psychotherapeutists, and Mr. H. R. Marshall, a former president of the American Psychological Association, Professor Witmer, of the University of Pennsylvania, and Professor Münsterberg, of Harvard, have pronounced against the movement on very various grounds.

To return again to the question of diagnosis, which is interesting, too, in view of the rosy character of the results reported from the churches. They are nearly as completely perfect as the cures at St. Médard. The habit of mind induced by years of observation of cases makes one doubtful of too uniformly successful treatments. One questions if many of the cases must not be of the hysterical type that chase from cure to cure and are always seeking new sensations in treaters and treatment. For these, soothing suggestions and darkened rooms and "gentle pressure of the hand" furnish novel and delightful emotions and fill them with that sense of their own importance so dear to those who "enjoy ill-health."

If one takes up a medical journal, in its melancholy columns he will often find an author recording his failures: he treated, he says, so many patients by such a method, so many recovered, so many failed to improve, so many died. Are there no failures to be recorded under this new plan? What of the ones without faith? The statistics are too good—80 per cent. of cures of alcoholism—indeed! Let us have a definition of alcoholism. If periodical drinking is meant, we can all cure that—between sprees! and the cure will be perfect until the next spree, at which point if one can catch the toper and head him off at the start, he may be stopped (*i. e.*, "cured") again. But what about the enlarged liver, the inflamed kidneys, the atrophied stomach of the alcoholic? Are these cured too? Even taking the broad statement of successes with inebriety or alcoholism, the time since the beginning of the Emmanuel treatment is too short

to be certain of the results remaining satisfactory. The medical profession is rightly suspicious of one of its members whose published statistics are too good. We like to see the failures recorded too. The assertion of the cure of 80 per cent. of alcoholics was made in a lecture in New York, by the Rev. Dr. McComb, assistant rector of Emmanuel Church. At the meeting in Philadelphia, where the paper was read upon which the present article is founded, Dr. McComb first denied and then reasserted this statement, and added that anyone who knew his job ought to be able to "cure" that percentage of "alcoholics." Mere physicians cannot hope to rival the therapeutic efficiency of this gentleman—and since no definition was forthcoming of alcoholism and alcoholics, we must be polite enough to accept his statement on its face; though the description, largely inferential, of the "alcoholism" in cases in *Religion and Medicine* reads for the most part like what might better be described without technicality as "drinking" or even as "hard drinking." Either of these is very different from "alcoholism," as doctors, at any rate, will know. Absent treatment is given to drinkers[5] and reported (by the still absent patient) to be successful. Again, a mere doctor, knowing the morals of drinkers, would want to feel the pulse and perhaps smell the breath of that patient while he is writing his letter.

More serious still appears to me the use of hypnotism in any form or degree. It is perhaps worth while to say that my experience in the practice of hypnotism extends back not two years (the Emmanuel movement began in 1907), but twenty-two. The first case that I can recall in which direct medical use was made of hypnosis was in 1886, in Vienna. After that I worked at it in Vienna and Paris, in 1886, and again in 1890, and have used it occasionally since, but less and less frequently and more and more cautiously as conviction of its dangers grew.

One reads in the official publications of the Emmanuel movement a great deal about hypnotism, in which its use is described and recommended in one place, condemned and denied in another. Suggestion! Suggestion! Suggestion! is the continual word on every page—waking suggestion, suggestion in sleep, autosuggestion, suggestion by another. If suggestion as a medical or medicoreligious measure means anything, it means hypnotism. If not, it had better be called by some other name than by this entirely technical term. If it means only encouragement, persuasion, and reasonable presentation of the case to the patient, or such use as everyone in the world makes every day to bring another person to see and apprehend his point of view, why call it by a technical name? The fact is it does mean hypnotism. Moreover, the descriptions given in *Religion and Medicine*, in Dr. Worcester's lectures, in the letters and literature of a number of the disciples,

[5] Religion and Medicine, p. 54.

speak of hypnotic suggestion as if in constant use. Side by side with these letters and descriptions are found letters denying hypnotism and saying that hypnotism plays no important or frequent part in their treatment.

Dr. Worcester, in a lecture in Philadelphia, described the use of a crystal ball—the oldest and most mechanical method of hypnotization, and said he used it "to fix the attention." To fix the attention for a few moments on a crystal ball, if the eyes are held to it, will hypnotize, to a certain degree, almost anyone, and still more if the other methods described are in use at the same time—quiet surroundings, concentration of mind, and the soothing voice directing attention to the ball and making suggestions. It is unnecessary to labor this point. We may simply say that anyone who says that suggestion in the form which these letters, lectures, and books advise is not hypnotization, does not understand hypnotism.

As to the possible ill effects of hypnotization without bad intent, while numerous instances could be quoted, it may suffice to recall some experiments at the University of Pennsylvania years ago. Two gentlemen, both promising young medical men then in various lines, and one of them now a most distinguished practitioner, experimented upon self-hypnotization or autosuggestion in the production of attacks of convulsion. After a few weeks of occasional experiment at such times as they could spare from other work, they found themselves able to bring on convulsive seizures of a most violent cataleptic description, sometimes with clonic movements. They soon stopped, finding the consequent nervousness and other effects disagreeable, and especially that the convulsions were *beginning to get beyond their control.* One of them told me that he had had convulsive attacks—controllable, to be sure, but sufficiently lively to be disagreeable—from so small an irritation of his nerves as a heavy cart rattling by as he walked in the street would cause. These were not sick men, nor neurotic patients, but strong, active, hard-working, unimaginative young doctors. Plenty of worse cases could be cited, but I will simply repeat the assertion which is backed by almost everyone with large practical medical or psychological experience—that repeated hypnotism decreases the powers of self-control and does *not* increase them, thus most obviously weakening the will; that the surrender of one's personality, one's mental independence, into the hands of another is a serious danger, can be denied by no one who reflects upon the ease with which such mental states become chronic enfeeblement.[6] The quotations in

[6] Mr. Henry Rutgers Marshall (a former president of the American Psychological Association) in a letter to the New York Times, makes the following statement, which needs no reinforcement: Suggestion, he says, is "an effect of one person's act of will upon the other's power of willing. An individual's acquiescence in the reception of radical forms of suggestion is an abrogation by him of his power of willing, and the process by which this suggestion becomes effective is dependent entirely upon the subserviency of the mind of one individual

Religion and Medicine to the contrary, are some of them fitted only to exceptional cases, and some of them are from persons of no recognized authority whatever. Indeed, it is astonishing to find the miscellaneousness with which authors—medical, psychological, and others are quoted in this book, as if the mere names were sufficient to give weight. Bramwell is quoted to offset Janet, for example, or Schofield is cited against Maudsley, as if their views were of equal value. No one could tell from the way they are quoted that Charcot, who touched so many medical subjects and added something of value to almost every one that he did touch, was any better authority than Dubois, whereas every educated physician knows that it would be moderate to say that Charcot's opinion was about six thousand times more valuable than Dubois' on any matter.

I desire to repeat, as the most important contribution to be made to this subject, that the use of hypnotism in any form, manner, or degree is a matter which should be under legal control. It is a dangerous remedy in the hands of the best qualified medical man, and utterly to be condemned when used by any one not a qualified and responsible physician. In Europe our Emmanuel friends would quickly find themselves in the hands of the police, who would probably prove less patient of amateur medical practice than our Boston confrères, who have with scarcely an audible protest consented to the subordinate and undignified position in which the new plan places them.

Nevertheless, I may conclude as I began by saying that in criticising the Emmanuel movement I am finding fault not with the use of proper mental means of cure, but with their improper, unwise, exaggerated, and unscientific application by persons not fitted to judge the needs and limitations of the patients treated. Mental and moral therapy is as good and valuable a remedy as it always was, and although it will suffer discredit, as other good remedies have done, from ignorant enthusiasm, the new attention which the discussion has brought it will in the end do good.

to the mind of another." The one thus guided "agrees to allow his mental states to be determined by outside influences as far as this is possible," and thus the control of the individual's self, so efficient in the building of character, is broken down. He points out, too, that freedom from pain or bad habits or from worry when due to the suggestion of another may be paid for by a weakening of personality. This result is a familiar one to those who have watched the effects of Christian Science treatments and beliefs and observed the calm and cheerful selfishness, the happy readiness with which the convinced and converted "Scientist" shifts his burdens of responsibilities on the nearest person he can find willing to carry them.

THE TREATMENT OF CHRONIC BRONCHITIS.

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To any one who has treated many cases of chronic bronchitis, it is needless to say how unsatisfactory it is. The best we are able to do in most instances is to relieve somewhat; but as to cure, that seems rarely, if ever, to occur—at least among our hospital and dispensary patients. Of course, if we should get, as we sometimes do, a patient who states that during the previous one or more winters he had persistent cough and expectoration, but during the intervals was apparently well, we are hopeful. And this hope is increased, if, after careful inquiry and examination, we find no distinct evidences of heart, lung, or kidney disease, or, indeed, anything abnormal other than what is easily explained by changes of the bronchial mucous membrane.

Now, what should we do, so far as may be, to prevent those conditions from arising as complications or sequels which, sooner or later, would make life miserable and notably shorten it? If the patient be well to do and the sacrifice be possible, we would say, first of all, and as soon as the weather becomes bleak, raw, and changeable, go to a warm and genial climate where life in the open air is agreeable and curative. But few men or women can properly do this when means and obvious duties are considered; at all events, not until it is imperatively necessary. If we wait until this period, secondary changes in different important organs have already taken place, and the best we can do is to keep them stationary, or prevent their getting rapidly worse.

In chronic bronchitis, hygienic measures are of primary importance. Good ventilation, plenty of air and sunshine are essential; so are good food and rest. Clothing suitable to the season, and well selected, is necessary. Apparel too heavy, which overheats in the house and causes perspiration, only brings on fresh colds and aggravates the chronic disease. Food should be simple, well prepared, and nutritious; but heavy meals, especially at dinner, must be interdicted. Strictest moderation in sweets and alcohol should be enjoined; and tobacco, as a rule, hurts notably, except in mild quality and very small quantity in the evening. All the foregoing, and much that could be added about the risks of draughts, wet feet, exposure to cold, biting winds, etc., are among the platitudes.

As to medication: Internally, the iodides are the most useful drugs we have when properly used, and without them our armamentarium in this line would be very defective. For my part my two standbys are iodide of potassium and syrup of hydriodic acid. When there

is dyspnœa, even slight, or nervous irritability shown in any way, we should combine the iodide with Hoffman's anodyne. Hydriodic acid may be alternated advantageously with terpene hydrate in fairly large doses. In the use of the latter drug I agree with my friend, Dr. R. H. Babcock, of Chicago. Invariably counterirritation to the chest should be insisted upon, and kept up for many days, or weeks, with occasional intermissions when the skin becomes tender. Nothing equals the compound tincture of iodine for its resolutive qualities, and the derivative effect toward the skin is all that is desirable. Internally I am opposed to the use of sedatives or anodynes, unless imperatively required. The least objectionable are the combined bromides, henbane, or codeine. A mercurial, followed by Rochelle or Epsom salt, is useful once a week or oftener, and diminishes cough and expectoration for a time in a pronounced degree.

Vapor inhalations, especially of creosote, are very valuable when properly used, and if persisted in are more curative than any other one thing, unless it be change of climate and, at times, habits and occupation. The inhalations should be used with the perforated zinc inhaler; my well-known formula of equal parts of creosote, alcohol, and spirit of chloroform is unequalled. Internally, creosote may also be given with the happiest effects, in small repeated doses, and, combined with the best whiskey and glycerin, will rarely disagree with the patient.

These patients cannot, should not, be housed. If so, they soon get worse, and their bronchial mucous membrane will not bear the slightest change without increased cough and expectoration. If a change of climate may be indulged in, one should go, preferably, to the sand hills of Georgia in winter, and in summer to the Adirondacks, at a moderate elevation. If permanent banishment seems desirable, California, not too near the coast, is the one place of best resort.

In these remarks I have considered the treatment of chronic bronchitis before the advent of emphysema or asthma or dilated heart. When one or the other or all these occur, the indications are somewhat different, more difficult and complicated. When these are not present, moderate exercise in the open air each day should be urged as imperative, unless the weather is very inclement or the patient is suffering from an acute attack grafted on the former trouble. Of all exercise, walking, moderate golf, and riding are the best.

Whenever there is an acute attack, with increased cough, tightening of chest, and less and difficult expectoration, with or without fever, benzoin may be substituted for creosote, and hot water used to vaporize it. In like manner, ipecac, sweet spirit of nitre, and spirit of mindererus take the place of the iodides; and mustard or soap liniment to the chest, and pediluvia at bedtime, take the place of compound tincture of iodine.

It would seem as though one should have more and better facts

to offer. I confess I have none. I have tried many, many things, internally and externally; all sorts of cough mixtures; all sorts of sprays and inhalations, including the globe inhaler, with vaporized oils and divers drugs, and finally, I have adopted what precedes, as best. Of course, judicious tonic medication, with iron, bark, cod-liver oil, arsenic, strychnine, etc., are at times desirable, and undoubtedly indicated. But above all, let it be understood that nothing practically equals dry or moist vapor inhalations of creosote, after the manner I have so frequently affirmed. Indeed, for almost all affections of the respiratory tract, including laryngeal and pulmonary tuberculosis, there is absolutely nothing now known quite or at all equal to them in efficacy and great power for good. But in order to get good, the best, results, one must have practical knowledge how to use them, and faith in their power to help when other things without them fail or are incomplete.

In the way of spa treatment, the only resorts I believe in specially on the eastern side of the United States are Sharon and Richfield Springs, giving the preference to the former, despite the fact that it is not an amusing place. Aix-les-Bains and Homburg, abroad, are the two valuable springs in my judgment. At Aix, one gets Marlios vaporizations; at Homburg, a specially useful innocuous alkaline spring, the Elizabethan, to drink.

AN ANATOMICAL STUDY OF PERICARDITIS.[1]

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Pericarditis is among the most venerable of recognized cardiac lesions in the history of medicine. Galen is said to have observed pericarditis in animals, and to have suspected its existence in man. Among the ancients the "hairy heart" was supposed, according to Haller, to indicate great valor and bravery. Among those heroes who after death were found to be so endowed are said to have been

[1] Read at a meeting of the Pathological Section of the Buffalo Academy of Medicine, April 20, 1909.

Leonidas and Lysander. Morgagni and Vieussens described the obliterative type of pericarditis, and Corvisart, in 1811, noted the significance of the bulging pericardium. Although Laennec was familiar with the lesion, he expressed his doubts as to the possibility of diagnosis, and the characteristic to-and-fro rub of the acute condition was not recognized until 1824 by Collis.

Notwithstanding the antiquity of the anatomical recognition of pericarditis, Osler confesses that it is a condition more frequently recognized in the dead-house than in the ward, referring chiefly to chronic pericarditis. Great difficulty in diagnosis of the acute lesion should no longer exist, unless it occur as a terminal condition in the late stages of some disease when the clinician's attention for detail oftentimes becomes relaxed in the study of obviously fatal cases. In such instances, however, the recognition or its failure is of little practical importance.

During the past three years our attention has been especially drawn to the subject, for the most part, because we have found the lesion so frequent at the autopsy table, and so rarely recognized by us in the wards. Yet the conditions in which pericarditis arises as a frequent complication are in general well understood and fully recognized. We have been constantly on the watch for it in such instances, but nevertheless our percentage of ward diagnoses in cases subsequently demonstrated in the dead-house has been disappointingly small. This study has, therefore, been undertaken not only in order that we might, if possible, correct these errors of diagnosis in the future, but also especially that we might more satisfactorily determine the nature of the process and better estimate the bearing of so marked and striking a lesion, which, however, often exists with very few or no obvious clinical signs.

The cases on which this study is based have been for the greater part taken from our own personal service; a few are from the routine protocols of the pathological department at the University and Bellevue Hospital Medical College. Our clinical observations have been chiefly made in the service of one of us at the City Hospital. In all, 1000 protocols have been examined, and of these, 150 have shown pericarditis.

Only frank and outspoken pericardial lesions of true inflammatory nature have been considered in this study. In the acute cases, for example, no instances of hydropericardium have been included, and we wish to make a sharp distinction between pericarditis, a true inflammatory lesion, and the hydropericardium, which occurs in various œdematous conditions. This may, indeed, be followed by true inflammation, but it is not in itself primarily of this nature. Of the chronic, only such instances have been included as showed manifest inflammatory thickening of the membrane or adhesions of considerable extent. The small granulomatous patches which are found so frequently, especially over the auricles and cardiac base,

for example, have not been included in these statistics, although these changes are also, in our opinion, unquestionably inflammatory.

Classification and Pathological Anatomy. For clinical purposes pericarditis is best classified as acute, subacute, and chronic. Subacute or chronic pericarditis occurs in many instances as an extension or terminal state of the acute forms in which there has been a fibrous or granulomatous replacement of the acute exudate.

In both acute and chronic forms the process is usually found most marked on the visceral layer of the membrane, even in those cases in which the lesion has been produced by direct extension, as from a pleural, pulmonary, or mediastinal inflammation. The most logical explanation of this fact appears to be the greater physical activity and the greater vascularity of the epicardium, as compared to the parietal layer of the membrane.

The acute forms are subclassified as fibrinous, serofibrinous, hemorrhagic, and purulent. We have found the division into fibrinous and serofibrinous unsatisfactory, since in both instances the lesions are practically identical. In the one, more clear serum is exuded; in the other, this serum is richer in coagulated fibrin; we shall, therefore, in this study group together all cases ordinarily classed separately. Similarly, hemorrhagic pericarditis is almost without exception the simple adding of extravasated blood cells to the serofibrinous exudate or to a purulent one, and we have, therefore, for statistical purposes considered hemorrhagic cases under the heading of one or the other of these forms. By some authors, however, hemorrhagic pericarditis has been considered as diagnostically definite; thus, Thorel states that it is present in tuberculous tumors of the pericardium, in uremia, the various hemorrhagic diatheses, and in leukemia. We have found it to have even a broader etiological basis and to be shown in practically any severe type of pericardial inflammation. Stewart,[2] for example, reports it present in a case of infection of the pericardium by the colon bacillus.

Acute serofibrinous pericarditis is a lesion characterized by the primary appearance of minute granulomas on the surface of the pericardium. The capillaries are injected and proliferation of the endothelial and connective-tissue cells is set up, together with inflammatory infiltration of the entire depth of the membrane. The change is seen most frequently or most marked over the base of the heart and notably over the walls of the auricles. Only the epicardial layer may be primarily involved, but sooner or later a similar change appears in the adjacent parts of the parietal layer. At the outset the lesion is very apt to possess a patchy character, but eventually in progressive instances it finally becomes diffuse. Coincident with the inflammatory changes mentioned a deposit, first, as a thin pellicle of fibrin gathers over the eroded surfaces, and this may increase in

[2] Zentralbl. f. innere Med., 1904, xxv, 863.

thickness, varying in different instances from a very delicate web to a coat as much as 1 cm. in thickness. Serum and leukocytes exude into the sac from the distended capillaries, and the amount which may so collect may become very great, as much as 2 to 3 liters, according to Osler and Gosselin, while Kestner claims to have found from 3 to 10 liters in scorbutic cases—all amounts far in excess of the fluid which can be artificially injected into the normal sac post-mortem, as shown by the experiments of Smith, Lusk, and others. Minute petechial hemorrhages appear in a good many instances, either into the fibrinous deposit or more frequently into the sub-endothelial connective-tissue layer, and in some cases actual granulation tissue may appear. Of course, when the pericarditis develops in such conditions as the hemorrhagic diathesis, in purpura or scurvy, the hemorrhagic exudate is very pronounced. Hemorrhage is also frequent in tuberculous inflammation and in those inflammations which arise from neoplastic invasion of the pericardium. In these last-mentioned circumstances blood extravasation takes place directly into the free fluid, and is not limited to the substance of the membrane or its adherent exudate.

It is generally admitted that in the healing of serofibrinous pericarditis fibrous substitution of the exudate takes place, and when abraded surfaces are apposed, adhesion, of course, with resulting synechia at these points follows.

The bacteria found in serofibrinous pericarditis commonly bear a direct etiological relation to the process. Those most commonly present are the streptococcus, the pneumococcus, various staphylococci, and, according to Poynton and Paine, Diplococcus rheumaticus. Secondary infecting organisms of all sorts may, of course, be present, and in some cases the exudate is sterile.

Purulent pericarditis in our experience occurs either from direct purulent infection of the pericardial sac or as a sequence to the serofibrinous form when infection with pyogenic organisms takes place. In most instances the effusion is less abundant than in the cases of serofibrinous inflammation, but it may become great before death ensues. As a rule, especially when the condition follows the serofibrinous form, the exudate is rich in fibrin, although the shreds are apt to become more or less liquefied. Pus, eroded connective tissue and endothelial cells, and general inflammatory detritus are also abundantly present in the fluid. The portions of the membrane previously most deeply covered by fibrin are now commonly overlaid by granulations. The color of the purulent effusion is, of course, chiefly dependent on the nature of the infecting organisms. Gas may also appear due to the action of microbic growth. Whenever the case has been of long standing one is very apt, in addition to whatever organisms may have been first present to find the colon bacillus or members of the proteus group. We have twice found Bacillus aërogenes capsulatus in purulent pericardial effusion when general

infection with this organism was present; gas was, of course, abundant in both these cases. The tubercle bacillus, streptococci, and staphylococci are the most common organisms found, and they usually bear a direct etiological relation to the disease.

When healing follows purulent pericarditis, extensive fibrous adhesions almost invariably form, and in some cases we have found patches of calcification.

Chronic pericarditis is most conveniently divided into a chronic fibrous variety, in which adhesions either do not exist or are present to very limited extent, and those cases which show chronic fibrosis with adhesions. The latter is by far the most frequent form in our experience. In the simple chronic fibrous form the essential process is a thickening of the fibrous layers of the membrane due in many instances to inflammatory changes, but frequently to a general fibrosis with this local manifestation, as in certain types of syphilis, akromegalia, arteriocapillary fibrosis, and the like. Hess[3] thus describes this lesion as a local manifestation of a general chronic serositis. The thickening may be entirely limited to a single layer of the pericardium, in which instance it is almost invariably the visceral layer which is so involved, but in most examples, and especially when it follows a previous serofibrinous or purulent inflammation both layers are diseased, but the visceral to the greater degree. Occasionally one finds extensive plaques of thickening without general involvement of the sheet, and here the changes are most prominent about the base and over the auricles.

Microscopically, the alterations are briefly those of a fibrous hyperplasia, usually with more or less still active round-cell infiltration, and commonly with exudation of some extent. In this variety, and also in cases of fibrous pericarditis with adhesions, when the etiology is tuberculosis or syphilis, the characteristic lesions of these infections may be present, although by no means invariably.

In chronic pericarditis, with adhesions, a very wide range exists between those cases in which only a few fibrous strands unite the visceral and parietal membrane and those in which the union is complete and of dense scar tissue. In our statistical studies we have placed in this class only those cases which showed fairly extensive synechia, but it is, of course, manifest that all degrees of adhesion may exist, depending probably on the nature and extent of the primary process and on its rapidity of absorption or replacement. In our experience when the union has been incomplete we have found the most numerous adhesions immediately about the apex or just over the auricles, but in this particular the cases vary greatly.

Tuberculous Pericarditis. As has been stated by Osler, pericarditis caused by tuberculosis may manifest itself either with distinct and demonstrably tuberculous lesions, as miliary tubercles or caseous

[3] Eine Klinische-experimentelle Studie, Marburg, 1902.

masses, or it may be found as a simple fibrous thickening of the membrane with or without adhesions. Although in the latter case the change is unquestionably indirectly due to the tubercle bacillus, if the specific bacilli or distinctly tuberculous changes cannot be demonstrated, we have chosen to class such examples as chronic fibrous or adhesive pericarditis, due to tuberculosis and not as true tuberculous pericarditis. In most instances of this nature it has been found that the pericardial change is but a local example of a more or less general fibrosis. As will be shown later in our study, tuberculous infection of the pericardium may cause practically any sort of anatomical pericarditis, from serofibrinous or hemorrhagic to purulent, fibrous, or adhesive, a fact which we think should be more generally recognized. This may depend on the varying degree of virulence of the organism, on the resistance of the body and its reaction, but probably still more on the nature of the contaminating organisms.

In tuberculous pericarditis of the chronic variety the membrane is, in our experience, generally much thickened and is studded with yellow necrotic areas, which are softening tubercles. When, on the other hand, the change is but a chronic stage of an acute tuberculosis of the serosa, as in some slow cases of general miliary tuberculosis, obliteration of the cavity may not be present, but only occasional shreds of adhesion are demonstrable. In practically all cases of tuberculosis of the pericardium, tubercle formation is not diffusely present, but is associated with inflammatory changes of clearly simple nature, and organisms other than the tubercle bacillus are also etiologically concerned. Indeed, in most cases, from a study of the lesions one is impressed with the idea that the tubercle bacillus is here a relatively inactive organism, and that other agents are probably much more directly involved in the process.

Syphilitic Pericarditis. Practically the same conditions obtain as in tuberculosis. Unless distinctly syphilitic lesions can be demonstrated in the pericardium, an admittedly rare condition, the lesion should not be classed as syphilitic pericarditis, although, as we show later on, various forms of pericarditis may be caused by syphilitic infection, a fact not generally recognized. There is now no doubt that any of the various anatomical forms of pericarditis, either acute or chronic, may be caused by the syphilitic virus. As might be expected, however, only a relatively small proportion show typical syphilitic pictures. In these, accompanying syphilitic changes in the myocardium are the rule. In passing, however, it is interesting to note that in the two instances of gumma of the heart, which occurred in the service of one of us, no pericardial changes of note were present. In the average case of syphilitic pericarditis the membrane shows a more or less diffuse thickening, and it is only on microscopic examination that changes suggestive of syphilis appear. It is manifested usually by perivascular infiltration, nuclear fragmentation, and a distinct tendency to necrosis, as well as toward hyperplasia of the

connective tissue. It is probably partly for this reason that syphilitic pericarditis has been so rarely reported. Gumma of the pericardium has never occurred in our experience, and McPhedran states that but three cases have been recorded.

Traumatic pericarditis does not deserve a separate classification from the pathological standpoint, since in most instances the actual pericarditis is due not to the traumatism, but to infecting agents introduced with it. It occurs mostly in surgical practice, and the changes induced depend on the nature of the contributory infection. The cases of perforation of the pericardial sac with foreign bodies from the œsophagus and stomach are very interesting, and the consequent pericarditis is invariably of a purulent type. The elder Flint thus cites in his text-book a case following perforation and infection from the swallowing of false teeth. Personally, we have never seen a case of this nature in man, but it is common in the ruminants, and several years ago one of us reported such a case in a buffalo, when the perforating body was a bale wire, quite a common circumstance, I am told, among domestic animals.

Mechanical pericarditis also does not exist as a distinct pathological entity, although it is a clinical condition of some importance. It is seen most frequently as a serofibrinous inflammation, occurring in such conditions as dilatation of the heart or from overaction, oftentimes with more or less dilatation. It is seen, for example, in Graves' disease, two marked instances of which have been observed in our service. Although the most common anatomical change found in these cases is a serofibrinous inflammation, fibrous hyperplasia also occurs with relative frequency. Rubino produced acute pericarditis in animals by experimental trauma, and we all have noted its frequency in tube-fed animals.

Knox[4] describes a further form of pericarditis characterized by the appearance of small fibrous nodules along the course of the arteries of the epicardial layer, not entirely unlike the syphilitic form described by Balzer.[5] According to Knox this pericardial inflammation is caused primarily by lesions of a degenerative character in the walls of the coronary arteries. We have not personally observed this form, except, perhaps, in those cases due to marked coronary arteriosclerosis, when the superjacent epicardium was thickened, hyperplastic, and more or less infiltrated; we have not considered such instances as frank pericarditis in this study.

Etiology. It is very questionable if a primary idiopathic pericarditis exists. At least, if it ever occurs it is a very rare condition, and even in reported instances Thorel[6] discusses the probability of most of the recorded cases being tuberculous or the result of hemic infections of other varieties. It is certain then that pericarditis is, almost with-

[4] Jour. Exp. Med., 1899, iv, 245.
[5] Archiv der Phys., 1883, vi, 93.
[6] Ergeb. der allg. Path. u. Path. Anat., 1909, S. 921.

out exception, a secondary lesion or a complication in some other disease or dyscrasia. This statement is now generally accepted, and holds true except for the relatively unimportant traumatic cases. In general our study upholds the usual conceptions in regard to the etiological factors chiefly concerned in the production of pericarditis, but in the investigation of the literature of the subject we have been impressed with the fact that in most reports the statistical conclusions are based on clinical findings only, or in some instances on bacteriological investigation alone, and are, therefore, less apt to be accurately based than when the data have been derived from complete postmortem examinations aided in many cases by histories, clinical notes, and bacterial examinations, as in our statistics.

In the acute serofibrinous type of pericarditis lobar pneumonia leads as the most frequent cause, and was present in 26 out of the total of 67 instances. This is in direct contradiction to the statement of von Schrötter, who states that pneumonia is not shown in his observation as an especially frequent cause of pericarditis. This apparent discrepancy is largely explained by the clinical observation, which I am sure has been apparent to all of us, namely, that certain epidemics of pneumonia have been characterized by the frequency of pericarditis as a complication, and in other years other complications have been more common. For example, nearly all the hospital physicians in New York City remarked on the great frequency of pericarditis in the pneumonia epidemic of 1906, during which time my statistics were partly collected.

In most instances pericardial infection in lobar pneumonia has taken place by transmission by direct contact in so far as could be determined by the local condition of the adjacent pleura and mediastinal tissues, although, of course, lymphatic or hemic transmission cannot be excluded in this general infection. Four cases of pericarditis of the serofibrinous type were found to have developed in the course of bronchopneumonia, and three from simple pleurisy without pulmonary involvement.

Rheumatism has been quite generally reported as the most frequent condition in which pericarditis of the serofibrinous variety develops. When we recall that under the head of rheumatism must be included many presumably septic conditions incorrectly diagnosticated, it is readily understood why this disease has been so generally accepted as the most frequent cause of pericarditis. In our observations, which it must be remembered are founded on postmortem and not on clinical evidence alone, rheumatism was found to be the factor in but 7 out of the 67 instances. Von Schrötter reports acute rheumatism as the cause in 30 per cent. of cases, von Harrass in 7 per cent., Pribram in 5.2 per cent., and Eichorst in 3 per cent. We must, however, not fail to recollect that in this respect clinical data may sometimes be the more valuable, since the larger percentage of these instances of rheumatic pericarditis recover, leaving no traces which

can be afterward definitely ascribed to rheumatism anatomically. Again, in regard to rheumatism all clinical observers are united in admitting that the percentage of pericarditis as a complication varies greatly in different years. Poynton, in his book, states that he has isolated Diplococcus rheumaticus from the pericardial effusion in a high percentage of cases of pericarditis, and he, therefore, assumes that all these instances are examples of true rheumatic infection. Until the relationship between acute rheumatic fever and the diplococcus of Poynton and Paine has been more definitely demonstrated most of us will be inclined to accept this evidence as far from positive.

Six of our cases developed in the course of general sepsis and two took place in generalized tuberculosis, without, however, the presence of tubercle bacilli or tubercles in the pericardium; in other words, they were the result of the mixed infection.

But five instances of serofibrinous pericarditis developed in the course of renal disease, a considerably smaller percentage than is shown in most statistics, and probably accounted for by the fact that our data were collected from postmortem and not clinical records. Four were due to acute and one to chronic nephritis.

Five cases developed as a result of syphilitic infection, four in the tertiary stage. In these examples no typically syphilitic lesions were demonstrable in the pericardium, although the predisposing influence was clearly syphilitic infection. Alcoholism, acute myocarditis, and asthenia appear as the etiological or determining factors in two instances each. In but one case could no probable etiology be found, but this instance was not submitted to bacteriological investigation.

Finally, in regard to the etiological factors concerned in the production of acute serofibrinous pericarditis, it will be observed that general bacterial infections predominated in a high percentage (46 out of 67).

In our eighteen instances of purulent pericarditis generally septic conditions naturally lead, although this was demonstrable in but seven. Lobar pneumonia with apparently direct transmission of the infection was present in three instances and purulent pleurisy in two. Three formed the terminal picture, following rupture of a tuberculous abscess of the lung with direct invasion of the pericardium, but without the formation of tubercles, although doubtless these would have developed had the patient not promptly died from the resulting general septic condition, of which the pericarditis was evidently but a part. Three cases of apparently idiopathic purulent pericarditis appear in which, while a generally septic condition was present, no other presumably primary focus of suppuration could be discovered.

But eight instances of chronic fibroid pericarditis independent of adhesions occur in our series. Of these, tuberculosis (without tuberculous lesions in the pericardium) leads in four examples, and chronic nephritis appears as the probable exciting cause in two.

In chronic adhesive pericarditis, sixty-one instances of which occur

in our list, tuberculosis, which was demonstrable in 17 cases, appears as the most constant etiological factor. In none of these were tuberculous lesions present in the pericardium, and the change seems then to have been induced by the general toxemia of the disease rather than by the immediate action of the tubercle bacillus. This statement is in general accord with the conclusions of most of those who have made a special study of pericarditis in its relationship to tuberculosis. In most instances the fibrosis does not appear to have been the result of a direct transmission of the inflammatory process from the lung and pluera, but to have followed the toxemia of the disease. Osler, for example, states that as a cause of pericarditis tuberculosis follows hard upon acute rheumatic fever.

Arteriocapillary fibrosis, with the pericardial lesion as a local manifestation, appears as the second most frequent cause of this form of pericarditis. It was present in 11 of our cases. We believe that the importance of this etiological factor has been very commonly overlooked by most writers, who have in many instances attributed the changes to nephritic disease, the "uremic pericarditis" of Banti, whereas the renal changes were but a part of the end result of the primary arteriocapillary disease. Independent and clearly etiologically important, nephritis could be traced in but 2 of our cases, while Bulil finds it causative in 35 per cent. of his, Mamberger in 14 per cent., and Frerichs found it probably causative in 18 out of 292. Banti,[7] in two carefully observed cases, came to the conclusion that the nephritic type of pericarditis is caused by the toxic factors present in nephritis, and states that he was able to reproduce the lesion experimentally. It seems highly probable that the frequency of terminal infection in uremia may bear some relationship to the development of pericarditis in nephritic conditions; although perhaps the distant cause of the lesion, it would not be so considered in our study.

Syphilis, with its general tendency to fibrosis and inflammatory changes, has been found as the most probable factor in 10 out of our 61 cases. In none of these, however, could definitely syphilitic alterations be demonstrated in the pericardium, and we, therefore, assume that the pericardial lesions were but local evidences of the general tendencies shown in this disease. We believe that the relationship of syphilis in this respect has been very generally unrecognized by previous writers on this subject, most of whom exclude syphilis as the probable cause unless definitely syphilitic changes are demonstrable in the membrane. This is by no means in accord with the accepted bearing of syphilis on such conditions as hepatitis, nephritis, and myocarditis, all lesions very similar in nature histologically to chronic adhesive pericarditis. Alcholism, with an anatomical picture somewhat similar to the changes found in cases of syphilitic

[7] Ueber urämische Pericarditis, Zentralbl. f. path Anat., 1894, 461.

nature, appears in nine instances out of the sixty-one, and we find in going over the literature that the relationship of this chronic drug poisoning has been also very generally overlooked by most authors, many of whom do not even mention it. Even a slight knowledge of the nature of the general changes which occur in chronic alcoholism is such as fully to convince one of its probable important bearing on pericarditis, particularly of this form.

Chronic endocarditis was concerned in but four examples of pericardial adhesions, in all of which the mechanical factors resulting from irregular action and cardiac dilatation appear to have been prominent.

The relatively small occurrence of rheumatism as an etiological factor in these chronic cases is notable in our series. It was found in only four cases. This probably is misleading, for again our statistics have been mostly founded on anatomical studies, and in some instances the histories were either entirely wanting or deficient in regard to this important question. Further, other chronic conditions are very apt to be associated with rheumatism to such a degree as to distract one's attention from the rheumatic factor; thus, alcoholism, tuberculosis, syphilis, and arteriocapillary fibrosis are all conditions frequently following or associated with rheumatism, any of which present more characteristic and definite anatomical changes. Personally, we are fully convinced that the general opinion in regard to the close relationship especially between rheumatism and the chronic adhesive form of pericarditis is correct.

But 6 cases of true tuberculous pericarditis were found in our series of 150. None of these were primary, and, indeed, primary tuberculosis of the pericardium is a most rare condition in the literature; that is, cases in which actual tubercle bacilli or tubercles have been demonstrable in this membrane and not elsewhere. All of our six instances developed in the course of a more or less generalized tuberculosis, that is, in tuberculous bacteremia rather than in merely localized tuberculosis of the lung or pleura. This fact is especially interesting when we recollect that up to recently it was rather generally conceded that a high percentage of cases of pericarditis with adhesions were really tuberculous. This false impression seems to have originated from the great frequency of tuberculous toxemia as a cause of chronic pericarditis, and because of inadequate anatomical study of the thickened membrane in cases of pericarditis with adhesions. Considering the great frequency of tuberculosis in its relationship to pericarditis, the relative immunity which the pericardium enjoys against this type of inflammation is, indeed, striking.

A very similar condition of affairs exists in regard to syphilis in its relationship to pericarditis, and the relative immunity here, as in tuberculosis, can perhaps be best explained by the theory of McPhedran, who asusmes that it is due to the relatively slight vascularity of the membrane. In but a single case of our 150 could definite acute

syphilitic changes be shown. This was an example of early but very severe secondary syphilis, in which death followed from perforation of an acute syphilitic ulcer of the aorta, but in which an acute syphilitic granulomatous pericarditis with inflammation of nearly all the serous membranes was demonstrable. We have already discussed, however, the important relationship which syphilis probably bears to the development of the chronic adhesive form of pericarditis. Rosenthal,[8] however, states that although it is generally assumed that syphilitic pericarditis occurs only in conjunction with syphilitic disease of the myocardium, it may do so independently, and Schönemann also reports such an instance. The cases recorded by Virchow, Ricord, Mracek, and Herxheimer all showed definitely syphilitic involvement of the myocardium.

No discussion of the occurrence of pericarditis in diseases of infancy, as in measles, scarlatina, and the like, has been presented, as our experience has been limited to adults.

Although there is more or less general unanimity of opinion in regard to the conditions in which pericarditis is prone to arise, the immediate local determining factors which in any given case cause the onset of the pericarditis are by no means clearly shown. It has already been pointed out that on account of the relatively small blood supply of the membrane it is rather less likely to become involved in bacteremic processes than other similar membranes, as those of the brain, for example. In this regard the observations of Charrin, however, tend to demonstrate that the metabolic products of bacterial growth when in the proximity of the pericardium tend to favor involvement of the membrane.

The predisposing influence of local traumatism, even though of slight degree, has been apparently fully demonstrated, the principle involved being apparently that of the locus minoris resistentiæ. This well-recognized fact has been corroborated by the experiments of Banti, who first caused irritation of the membrane by the injection of turpentine or by the mere puncture with a platinum needle, subsequently producing an experimental bacteremia with the pneumococcus, when a pneumococcic pericarditis was quite constantly produced. Rubino caused a typical pericarditis by the preliminary infliction of a blow over the pericardium or even over the thorax at large, and the subsequent injection into the blood stream of virulent staphylococci. Somewhat similar experiments have been conducted with the diplococcus rheumaticus by Poynton and Paine. Stern[9] thinks that other and less definite injuries to the thorax may be the determining factor, especially when mediastinal hemorrhages are induced. These observations are in a way corroborated by certain experiments of our own, in which we have noted the great fre-

[8] Berl. klin. Woch., 1900, Nos. 47, 48.

[9] Ueber traumatische Entstehung innere Krankheiten, Jena, 1900.

quency of pericarditis, especially of the serofibrinous form, in animals tube-fed for considerable periods, in which traumatisms to the mediastinal tissues resulted from the frequent passage. Pericarditis is very prone to develop in these animals even when no bacteremia preëxists, although in most of our experiments the general resistance of the tissues was already reduced by drug administration and terminal infection was, of course, thereby favored.

Clinical observation has also led us to consider the role which overaction of the heart plays, as in Graves' disease, in nephritis with elevated blood pressure, and in the numerous conditions in which rapid action of the heart oftentimes with more or less dilatation of its chambers occurs. Although as yet our data do not warrant us in more than a very tentative statement, we believe that these factors do play a very important role in the determination of pericarditis in cases in which it might not otherwise develop.

The Association of Other Lesions with Pericarditis. In this relationship more interest is derived from the bearing of the concomitant lesions upon the pericarditis than of the pericarditis upon them, for while it may be caused by or follow in the wake of several other lesions, there are very few lesions which follow or result primarily from pericarditis. Nevertheless the association of concomitant lesions in the heart and other viscera with pericarditis becomes at times of diagnostic and prognostic value, more so, however, as indicative of the general process or condition in which the pericarditis has originated than of any direct value in the immediate diagnosis or management of the pericardial change itself.

Entirely as one would expect, the lesions most constantly associated with pericarditis are changes in the pleura. In our 150 cases pleural lesions were present in 136. In well over half of these cases it appears that the pleural change is the more anterior, and that the pericardial alteration occurred later, perhaps even as a result of the pleural lesion, though still back of this relationship is the general disease or condition in the course of which the two membranous inflammations developed.

A direct relationship between pericardial and pleural lesions is best shown in the acute serofibrinous cases, in which out of the total of 67 instances, 60 showed pleural changes; in 36 of these the lesions were identical in character. In 11 others chronic inflammatory changes were present in the pleural sheet, which, nevertheless, also appears to have had a direct bearing on the onset of the adjacent pericardial inflammation. Four cases of purulent pleurisy appear to have been the point of origin of the acute serofibrinous pericarditis, so that in 51 out of 67 cases the pericarditis seems to have appeared as secondary to a like or similar process in the pleura.

This pleuropericardial relationship is even more clearly shown in the cases of purulent pericarditis when associated changes were present in all except one. Purulent pleurisy was present in 9 out of the

18 cases, and in 5 others the pleural change was serofibrinous in nature; possibly, however, in these last five, secondary to the pericarditis. In 3 out of the 4 remaining cases the pleural changes were chronic inflammatory.

The association between chronic adhesive pericarditis and pleurisy is less definite, but the pleura was found involved in 48 out of the 61 cases. In 31 of these the pleural change was like that in the pericardium, which would seem to suggest a common origin. It is very important to note in this regard that in but two instances was the pleural inflammation demonstrably tuberculous when the pericardial change was simply chronic inflammatory in nature. In three examples the pericardial lesion certainly was not secondary, but perhaps rather causative of the pleural change, since the pericarditis was a chronic adhesive one and the pleurisy acute. In six instances both acute and chronic alterations were demonstrable in the pleura.

In our 6 cases of tuberculous pericarditis, tuberculous pleurisy was present in 4, and the other 2 were examples of general miliary tuberculosis, in which for some reason the pleural membranes appear to have escaped invasion.

Although some authors have assumed a more or less direct relationship between pericarditis and endocardial disease, this does not seem to be borne out by the study of our series. From our records it seems even highly problematical if there be any significant relationship between these processes. Thus, of our serofibrinous cases, 46 of which were caused by bacteremia of some variety, only 10 showed acute endocardial changes; 7 of these were unquestionably rheumatic, and bore no other relation to the pericarditis than that both had a probably common etiology. The fact that 28 out of these 67 instances showed chronic endocardial changes is believed to be of no bearing, but to be explained by the great relative frequency of chronic endocarditis. All these cases were very carefully gone into under the supposition that deficient muscular action, with perhaps dilatation and mechanical friction might have predisposed toward the pericarditis. This probability could not be sustained in any of these instances except when other changes to be mentioned later on were present.

This lack of correlation between endocardial and pericardial disease is further emphasized in the purulent cases, which although, universally due to bacteremic conditions were unassociated with endocardial lesions of an acute character, except in one instance. From these cases one may apparently more or less definitely assume that infection is rarely borne by the route of the cardiac vessels from the endocardium to the pericardium, or vice versa.

Acute endocardial alterations were entirely absent in the 61 cases of chronic adhesive pericarditis, but in 35 chronic endocardial inflammation was present, but in no such degree or form in any as to indicate probable dependent or etiological relationship.

Myocardial alterations are present in the majority of cases of pericarditis, but that this lesion has more than an accidental or common etiological relation in most instances does not seem likely. Nineteen of the acute serofibrinous cases were accompanied by acute parenchymatous degeneration of the heart muscle. Study of these shows that in but a very small number was the pericardial lesion in any likelihood responsible for the myocardial disease, which in most cases was clearly due to the general toxemia of the disease in which the pericarditis had developed. On the other hand, a certain number of cases are present in which the dilatation and deficient muscular action of the myocardium consequent upon the myocardial disease may have had a predisposing or determining effect in the evolution of the pericarditis. This suggestion is given further probability by the occurrence of eighteen examples of fatty degeneration of the myocardium in which no derivative effect of the pericarditis can be assumed, but rather the contrary, and this, in our belief, is one of the factors which favors the appearance of terminal pericarditis in the numerous conditions in which myocardial degeneration or deficiency is shown. That fatty degeneration bears an especially important relationship is also suggested by the fact that but six instances of fibroid myocarditis were associated with the pericarditis, a very sharp contrast to the occurrence of the fatty degeneration, which in most cases was further suggestive by the concomitant occurrence of more or less dilatation. In 20 of the acute serofibrinous cases absolutely no myocardial lesions could be made out, and it is most striking to note that only 2 of the 67 showed acute inflammation of the myocardium.

This lack of effect on the heart muscle of the adjacent inflammatory process is still more vividly illustrated in the purulent cases, of which only two showed acute inflammatory lesions in the muscle. Nine of the 18 naturally showed parenchymatous degeneration of greater or less degree, and in 6 fatty degeneration was associated.

All of the 8 cases of chronic fibroid pericarditis without adhesions showed myocardial lesions, among which there were 2 of fatty degeneration, 3 of fibrosis, 2 of brown atrophy, and 1 of an acute and incidental parenchymatous degeneration.

Of the 61 cases of chronic pericarditis with adhesions, myocardial changes were entirely absent in 13. Fifteen showed fatty degeneration, 14 fibrosis, and 7 brown atrophy. Careful study of these cases in particular was made in order that, if possible, some conclusions might be drawn as to the effect on the myocardium of pericardial adhesions. In so far as we can conclude in those instances in which extensive effects on the heart were associated with pericardial adhesions, the myocardial disease was in most cases clearly the earlier, and we fail to find in our series of cases what we have also failed to demonstrate clinically, namely, that adhesive pericarditis has any very important or serious effect on the anatomical condition of, or on

the action of, the heart. In so far as we have been able to observe, some of the cases showing most pronounced adhesions clinically manifested little or no evidence of cardiac embarrassment *unless the myocardium itself was diseased*, usually primarily or independently so.

None of our six cases of true tuberculous pericarditis showed tuberculous invasion of the myocardium, and in none were the myocardial lesions apparently due to the pericarditis.

Renal changes are so universally present in all cadavers that it is very difficult to make an intelligible analysis of the occurrence of changes in these organs in association with pericarditis. Manifestly, in those cases showing arteriocapillary fibrosis alterations of this character were present in the kidneys, but in no instance were we able to find anything indicative of any inter-relationship between pericarditis and renal disease. Neither do we find from our study any indication that renal disease per se is an important factor in the causation of pericarditis, except in a very general or indirect way, for example, when, as suggested by Thorel, pericarditis arises as a terminal infection or perhaps follows myocardial disease of renal origin.

The Cause of Death. One of the most interesting parts of our study has been the investigation of the cause of death in pericarditis. This, we find, is only very rarely due to the pericarditis itself. In but 6 of our total of 150 cases was pericarditis mentioned as among the causes of death. In five of these instances the lesion was septic in nature, and in the one remaining, serofibrinous, due to a general infection, with its most pronounced focus in the pericardium. In most cases the causa mortis has been the general disease or condition in which the pericarditis had arisen as a complication. It is also interesting to note that notwithstanding the admitted frequency with which rheumatism acts as the cause of pericarditis, death was found to result from definitely rheumatic changes in but two instances, both examples of serofibrinous pericarditis.

Naturally, septic conditions are found to be the most frequent cause of death in pericarditis, and in 86 of our 150 cases death resulted from infections exclusive of tuberculosis and syphilis. In some of these, as in 17 cases of chronic adhesive pericarditis, the infections were apparently terminal in character; 8 of them were thus pneumonic and 9 simple sepsis. In the serofibrinous cases the infections were for the most part not only the cause of death, but also the obvious cause of the pericarditis. In the serofibrinous cases it is interesting to note that the cause of death was an inflammatory process adjacent to the pericardium in 30 instances of lobar pneumonia, 4 cases of bronchopneumonia, 1 case of empyema, and 9 cases of acute endocarditis. Not included in this list are four instances of death from acute myocarditis and four from acute tuberculosis.

In chronic adhesive pericarditis the cause of death appears to be absolutely independent of the pericardial lesion, which we have come

to consider as of very little prognostic importance. Pericarditis was not even mentioned as among the causes of termination in any of these cases, and in but 5 instances was a cardiac dilatation the immediate cause of exitus. After a careful study of these cases, in three only can the pericardial inflammation or adhesions be considered as probably actively concerned in the cause of death. Chronic endocarditis was the terminal factor in 4 instances, acute endocarditis in 1, lobar pneumonia in 8, sepsis in 9, empyema in 1, and tuberculosis in 9. General changes attributable to alcoholism appear as the probable cause of death in 6 instances, and definitely syphilitic lesions in 4, myocarditis of non-syphilitic nature in 4, and traumatism in 2.

In the chronic fibrous form of pericarditis without adhesions the cause of death is even more indefinitely connected with the pericardial lesion, and, taken as a whole, we again wish to express our conviction of the very minor role which pericarditis in any form plays in prognosis, except as a measure of the degree or extent of any general infection.

Conclusions. In conclusion, we wish to summarize those points which have especially impressed themselves upon us in the course of this study, although some of the evidence leading us to these conclusions has been but incompletely expressed in the paper:

Pericarditis is a lesion secondary in nature, rarely or never primary. The complication, for such it must be considered, is in most cases not in itself serious except when of the suppurative variety, and in all instances it is more noteworthy as indicating the general condition in which it arises than on account of its own importance. It is, therefore, of little relative clinical importance, except as a diagnostic index, and in suppurative cases. True myocarditis is infrequently associated with pericarditis, but myocardial degeneration is commonly found, and is due in most instances not to the pericarditis, although generally caused by the same condition as the pericardial inflammation. Death rarely results in pericarditis from this lesion. Independent myocardial degeneration leading to dilatation of the heart, and especially fatty degeneration of the myocardium, is a predisposing or determining factor toward pericarditis. Overaction of the heart may induce pericardial inflammation. Serofibrinous pericarditis is in most instances an evidence of generalized bacteremia. Chronic adhesive pericarditis is a lesion of great frequency, often impossible of diagnosis, and in itself of very little clinical significance or importance. Serious symptoms arise from adhesive pericarditis only when the myocardium itself is seriously diseased, either concomitantly or quite independently. The signs usually cited as characteristic of pericardial synechia develop only when mediastinal inflammation or adhesions of marked degree are present in addition to the pericarditis.

NORMAL AUSCULTATORY DIFFERENCES BETWEEN THE SIDES OF THE CHEST.

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Differences at the Bases Behind. From those who are concerned to make the earliest possible diagnosis of pulmonary tuberculosis we hear more and more loudly each year the command to study with all possible care and attentiveness the auscultatory differences between corresponding portions of the two sides of the chest, as well as between different portions of the same lung. The slightest variation in pitch, quality, intensity, or duration of either inspiration or expiration at any point, as compared with conditions in the corresponding portion of the other lung, is, we are told, a sign to be noted, since it may be one of the factors in a decision affecting the patient's whole future life.

If this is true, and I see no good reason to doubt it, it behooves us to know as much as we can about any differences existing between the auscultatory phenomena in the right lung and those in the left in the healthy chest. It has been clearly recognized, at any rate since Austin Flint's time, and by the great majority of competent practitioners, that the apices of the two lungs differ in a large proportion of healthy persons, the apex of the right being more like that we expect to find in beginning solidification (from any cause) than is the apex of the left. All this is too familiar to need repetition. I wish, however, to call attention to what I believe to be a fairly common difference between the right and the left base posteriorly.

In a series of 250 healthy cases recently examined with reference to this point, I found the difference to which I allude to be present in 67 per cent. In these cases I noticed that the breathing at the left base was notably louder and harsher than that at the right. In the majority of these it had also the quality described by the Germans as "rough." I have been unable to see that age, sex, the time of day, or the amount of food in the stomach had any effect upon this phenomenon; yet I found it sometimes present in the morning and absent in the evening, for reasons unknown to me. It seems to be distinctly less common in persons under twenty years of age.

I have nothing of importance to say as to the cause of this difference. Thinking that it might be due to the amount of gas in the stomach, I inflated a patient's stomach with air and auscultated the backs of the lungs before and after the inflation, but I could not see that any considerable effect was produced by this procedure. One might conjecture that the slightly lower position of the diaphragm upon the right, or the greater impingement of

the heart upon the left than upon the right lung, might have some influence in producing the auscultatory differences to which I have referred, but these are unverifiable conjectures, and seem to me practically valueless. I have not found any differences in percussion or palpation accompanying the phenomena to which I have just referred.

Obviously, the only importance of the sign which I have described is that in case it is confirmed and established by the observations of other physicians it will slightly complicate our process of reasoning in regard to the soundness or unsoundness of the lungs as based upon physical examination. It may be that we shall have to make allowance for the differences to which I have just referred, as we have been in the habit of doing with the differences between the two apices.

Differences in the Lateral Recumbent Position. Almost every good clinician is familiar with the fact that when the patient is lying upon his side the restriction of the motion of this side of the thorax leads to differences in the phenomena obtained by palpation, percussion, and auscultation in the lower lung, as compared with the upper. Hence we are all of us chary of drawing conclusions from what we hear in a patient's chest in the lateral recumbent position. We all prefer to examine the patient, either sitting up or lying on his face, whenever one of these positions can be assumed without serious inconvenience. But I wonder how many of us would be able to state, promptly and correctly, just what are the differences between the upper and the lower sides of the chest of the patient lying upon his side. As a matter of permanent and accurate record, it seems to me worth while to formulate as accurately as we can the knowledge which we all of us now possess in a more or less inchoate form.

With this in view, I recently examined fifty normal chests with the subject in the lateral recumbent position. Briefly stated, the differences are as follows: (*a*) On palpation, increased tactile fremitus on the lower side; (*b*) on percussion, a combination of dulness with a tympanitic quality on the lower side; (*c*) on auscultation, an increase in the intensity of the spoken and of the whispered voice, with a slight prolongation of expiration and a raising of its pitch.

Summarily stated, these signs amount to the indications of a slight degree of condensation of the lung, such as we see in the upper part of a chest when a pleural effusion is present below.

It is perhaps also worth noting that in patients, both of whose lungs contain approximately an equal number of rales when the patient is examined in the sitting position, lateral decubitus increases the number of audible rales on the lower side.

THE PRACTICAL VALUE OF SPINAL PERCUSSION IN DISEASES OF THE MEDIASTINUM.[1]

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Curiously enough, the systematic study of normal vertebral resonance and of its pathological modifications has never come into general vogue as a routine method of physical examination, so that to most clinicians the technique of vertebral percussion, in comparison with the more familiar procedure of what may be termed mural percussion of the thorax, is virtually a closed book. Despite this indifference on the part of medical men in general, several noteworthy contributions have been published in recent years on the subject of spinal percussion and the various tonal changes thereby demonstrable in diseases of the thoracic and abdominal cavities. Twelve years ago, von Korányi[2] drew attention to the fact that in pleural effusions displacement of the posterior mediastinum abolished the normal resonance of the lower spinal vertebræ and produced a triangular patch of dulness extending therefrom to the unaffected side. The more recent investigations of Grocco[3] and his followers along these lines are too well known to call for more than passing comment. Nagel and de la Camp,[4] in a recent series of experimental and clinical studies, have clearly demonstrated the value of percussing the spinous processes as an aid to the diagnosis of enlarged bronchial and mediastinal lymph nodes. Ewart's careful original work[5] on percussion of the vertebræ and its correlation with dorsal percussion also forms a most important contribution, and will be referred to at more length in a subsequent paragraph, as will von Korányi's elaborate studies on the vertebral percussion zones.[6] In passing, it is of interest to recall the fact that almost half a century ago Piorry[7] advocated "pleximetry of the spine," and described in this connection a vertical band of universal vertebral *dulness!* Obviously, this cardinal error in acoustics is sufficient to dismiss this investigator's conclusions from serious consideration.

The subject matter of this communication deals primarily with the technique of spinal percussion and the application of this method of research to the clinical investigation of mediastinal lesions, which in the cases under consideration consist chiefly of enlarged bronchial and mediastinal lymph nodes met with in connection

[1] Read by title at the meeting of the Association of American Physicians, Washington, D. C., May 11 and 12, 1909.

[2] Handbuch d. spec. Path. u. Therap., 1897, iv, 717.

[3] Rivista crit. di clinica med., 1902, iii, 13.

[4] Jahrbuch f. Kinderheilk., 1908, lxviii, 46.

[5] Lancet, 1899, ii, 261.

[6] Ztschr. f. klin. Med., 1906, lx, 295.

[7] Traité de Plessimétrisme. Paris, 1866.

with tuberculosis and other pleuropulmonary infections, and which also include several examples of neoplasms implicating the middle and posterior mediastinal spaces. Lesions such as these, as will be explained presently, affect the percussion sound over the thoracic vertebræ, and with this segment of the spinal column alone is the present paper concerned.

The normal osteal percussion resonance of the spine as a whole, elicited by rather *forcible percussion* of the vertebral spinous processes, is characterized by a sound of well-sustained volume and duration, of relatively high pitch, and of a peculiar osteal quality—the last two criteria being judged by their comparison with the pitch and the quality of healthy pulmonary resonance. Vigorous percussion blows, then, bring out this type of resonance along virtually the entire length of the spinal column, from the nape of the neck to the coccyx, for, when sharply struck, an individual spinous process transmits the impact to neighboring vertebræ, above and below, and the vibrations set up in this manner induce a sonorous tone whose dominant element is resonant. This synchronized and concerted pleximeter action of the vertebræ under the action of a sharp percussion blow provokes a general resonance of the spine, despite the possible existence of certain acoustic factors, which, were they not smothered, so to speak, by the loud osteal tone, might damp less powerful vertebral vibrations, and thus modify the percussion findings along areas of the spine that abut upon both normal and morbid structures of the thoracic and abdominal cavities. To all intents and purposes, the spinal column acts as a long pleximeter, and the vibrations thereupon set up by the percussion impact travel not only up and down the vertebræ and laterally along the ribs, but also to some extent are conducted toward and even into the organs and other structures of the thoracic and abdominal cavities.

In contrast to the foregoing, by *gentle percussion* it is possible to distinguish, in the healthy subject, differences in the percussion sounds over certain areas of the spine, which differences are explained partly by the resiliency of the segment percussed, and partly by its proximity to various pre- and para-vertebral structures. The former condition, that is, the inherent vibratory properties of the spine itself, is an all-important factor in determining the character of the percussion findings over the different spinal segments. The dulness ordinarily afforded by the cervical region is to a large extent attributable to such a cause, for, aside from other physical influences, this part of the spinal column is anatomically unfitted to generate a resonant sound: to the pleximeter finger it presents a short, uneven, concave column of bone, composed of compact vertebræ tipped with short bifid spines, held close and rigid by a tense ligamentous network, and unprovided with articulating ribs for the lateral conduction of vertebral vibrations. On the

other hand, the thoracic zone seems peculiarly well adapted to yield resonance on percussion: to the plexímeter finger it presents a long, even, convex segment of bone, unaffected by disturbances of tension referable to excessive flexion, and made up of large vertebræ having long, stout spines and costal paths for the transmission of vibrations. The influence of contiguous structures in modifying the spinal percussion sound is to be considered in intimate correlation with the anatomical factors just mentioned, and in pathological conditions of the mediastinum such influences must, naturally, dominate. In the cervical region, the tympanitic effect of the prevertebral air passages and gullet is effectually negatived by the structural peculiarities of the spine here existing, so that the sound remains dull so long as the percussion impact is gentle, though a tinge of laryngotracheo-oral tympany is appreciable on forcible percussion while the subject's mouth is open. The thoracic vertebræ, which abut anteriorly and laterally upon the lungs, are consequently clear and resonant on light percussion, and they become increasingly hyper-resonant as the force of the blow becomes greater. The fact that this normal resonance of the thoracic spine is definitely modified by certain mediastinal diseases demonstrates the advantage of spinal percussion as a routine clinical procedure. Lesions of this intrathoracic space, it should be remembered, may impinge against the spine so closely as to damp its vibrations most effectually, without affecting in the slightest degree the percussion sound of the thoracic wall proper.

The Normal Spinal Percussion Zone. Percussion of the vertebral spinous processes by the method subsequently to be described delimits a vertical zone overlying the twelve thoracic vertebræ and affording percussion sounds which differ acoustically according to the situation of the vertebra percussed. In health, percussing the successive spines downward from the vertebra prominens, the sound is frankly dull, the tactile resistance exaggerated, and the pitch high over the first three, sometimes four, thoracic spines, below which level the remaining eight or nine thoracic spines afford clear, low-pitched resonance tinged with a distinctive osteal quality. With a hammer and ivory plexímeter one can furthermore distinguish certain circumscribed patches of impaired resonance along this stretch of uniform resonance elicited by ordinary mediate finger percussion. Thus, Ewart[8] lays great stress upon the isolated dulness of the fifth thoracic spine, which modification he considers an invariable normal finding, attributable to "the cessation at the level of the fifth vertebra of the resonant influence of the trachea and to the replacement of this resonant influence by the dulling influences due specially to the infratracheal glands." That dulness in this situation is clearly demonstrable in instances

[8] Loc. cit.

of bronchial glandular tumors is a well-recognized fact, but it is not easy to understand how it is possible for a group of normal-sized bronchial lymph glands thus locally to damp the vertebral vibrations, despite the lost resonance of the windpipe at this level. The authority just quoted also speaks of "postcordial" impaired resonance over the sixth and seventh thoracic spines; of "left auricle" dulness over the eighth and ninth; and of "posthepatic" modified dulness over the tenth and eleventh. While recognizing the academic interest of the foregoing refinements of the percussion sound, their practical service as diagnostic aids must appear questionable.

In an elaborate study of vertebral percussion von Korányi[9] has mapped out the entire spine, from occiput to coccyx, into five separate zones, each of which, he asserts, furnishes distinctive percussion findings. In brief, these zones, with the sounds therein obtainable with identical percussion technique, are as follows:

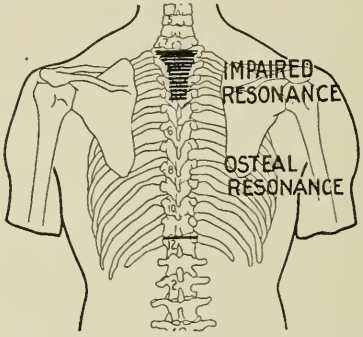

FIG. 1.—Normal percussion zones of the thoracic spine. Impaired resonance and increased tactile resistance from the first to the third or the fourth spinous process; clear osteal resonance from the fourth or the fifth to the twelfth spine. (The figures have been prepared from Keen's Clinical Charts, published by P. Blakiston's Son & Co.).

(1) first to seventh cervical, dulness; (2) first to fourth thoracic, dulness blending below with impaired resonance; (3) fifth to eleventh thoracic, clear resonance; (4) twelfth thoracic to fifth lumbar, dulness progressively diminishing until tympany appears over the last lumbar spine; (5) sacrum and coccyx, tympany. These take into account the whole extent of the spinal column, and constitute a somewhat more formidable and complicated percussion map than is necessary for routine clinical work. Of the five different zones outlined, but two (the cervical and the thoracic) can be depended upon invariably to show definite findings, and of these two the cervical, being so far removed from the predominant influence of the pulmonary structure, can safely be dispensed with, save under

[9] Loc. cit.

most exceptional circumstances. The lumbar and sacral segments, whose physiological tympany is so materially interfered with by the dulling action of solid matter within the bowel, do not enter into the consideration of mediastinal affections, and in the investigation of lesions of the abdominal organs which should impair the tympany of this lower vertebral zone, the percussion findings of this level must needs be interpreted with great caution, for the reason just specified.

The accompanying illustration (Fig. 1) shows the two spinal zones corresponding to the crevical and thoracic vertebræ, to be reckoned with in dealing with the diagnosis of mediastinal disease.

Technique of Spinal Percussion. In order to obtain interpretable data from spinal percussion one must observe consistently certain details of technique relating chiefly to the subject's posture and to the method of eliciting the percussion sounds. As a preface to remarks on this score, it should be stated that it is useless to attempt the accurate percussion of a diseased spinal column, the plexímeter action of which is interfered with by various inflammatory processes and deformities affecting the osseous structure and its vibratory properties. The latter are also altered in thoraces having such an unnatural convexity of the vertebral extremity of the ribs that the spine lies in a deep gutter walled on either side by a high costal ridge. Excessive rigidity and overdevelopment of the musculature (especially of the vertebral, vertebrocostal, and vertebroscapular groups) are two additional bars to successful percussion of the spine, and it is quite obvious that a generous layer of fat upon the back must have a similar effect.

The subject's posture must be such as perfectly to relax both the vertebral muscles and the interspinous ligaments, to insure which the patient should sit upon a cushioned stool (or upon a chair, facing its back), with the trunk inclined forward so that the crossed arms repose lightly upon the knees, and with the head bent forward at an angle of about 50 degrees from the spinal column. In this position mistakes due to undue spinal rigidity from musculoligamentous tension need not be feared. On the contrary, when the subject sits upon a hard stool or stands upright the spinal column must necessarily be subject to the increased tension inseparable from the proper maintenance of the given posture. It is, of course, manifestly impracticable to percuss the spine with the subject lying in lateral decubitus.

Mediate finger percussion, as ordinarily practised, is the most satisfactory method of percussing the spine, the stroke being directed so as to fall upon the dorsum of the plexímeter finger applied individually to the tips of the spinous processes, beginning at the first thoracic vertebra and thence percussing downward toward the hyper-resonance of the lumbar segment. The stroke should be delivered somewhat sharply, but, and this detail is all important,

with moderate force, the exact degree of which can be determined only by personal experience. Too much force may bring out a confusing hyper-resonance which wholly masks the dulness actually existing; too little force may fail to demonstrate anything tangible. Some idea of the proper degree of force to be used can be learned by percussing, with strokes progressively increasing in strength, the spinous processes at the level of the inferior scapular angle, until the spine emits a clear, sonorous, resonant tone and at the same time conveys to the plexímeter finger a sense of moderate resiliency at the moment the percussion impact occurs. With precisely the same technique percussion of the cervical and upper two thoracic spines should, under normal conditions, produce high-pitched dulness with unmistakably increased tactile resistance. In judging the character of the vertebral percussion findings, the resistance appreciated by the plexímeter finger is a far surer guide than the sound produced by the percussion impact, in view of which point it follows that mediate finger percussion is preferable to instrumental percussion with a hammer and ivory or metal plate.

Summary of Case Reports. The twenty mediastinal cases herewith presented to illustrate the utility of spinal percussion were selected from some 300 odd records of examinations made during the last two years in routine hospital practice. For the sake of clearness, the individual percussion findings in these cases have been diagrammed, with a brief *résumé* of the clinical histories. In every instance the diagnosis of the mediastinal affection was verified by an *x*-ray examination, as a supplement to the evidence presented by the patient's symptoms and by the more familiar mural physical signs. The last two sources of information were sufficient to warrant almost certain clinical opinions in eleven of the cases (Cases I, III, VII, VIII, IX, XII, XIII, XIV, XV, XVI, XIX), and in these the vertebral percussion findings were confirmative of the other objective symptoms, save in a single instance (Case XII), in which, despite a pertinent clinical picture and a convincing radiogram, careful percussion of the upper part of the thoracic spine afforded nothing more than a modification of the osteal resonance. This discrepancy, for lack of a more tangible cause, seems best explained by assuming that the subject's concomitant emphysema, which was typically developed, must have exerted a disproportionately hyper-resonating influence on the spinal vibrations. This influence, it may be added, was readily demonstrable by percussing the vertebral column immediately below the strip of interscapular impairment. The predominant effect of emphysematous hyper-resonance upon the spinal percussion sound is clearly shown by the chart of Case IV, which suggests a zone of excessive resonance at the levels of the second and third thoracic spines (Fig. 5). Much the same peculiarity is also shown, though somewhat less conspicuously, by Cases VI, X, and XII,

but only in the last instance did the physical signs spell hypertrophic emphysema (Figs. 7, 11, 13). It is conceivable, however, that the dulling effect of an intrathoracic solid mass may be more or less

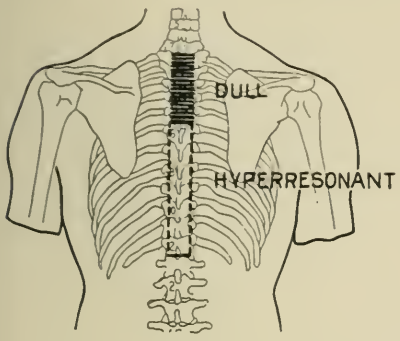

Fig. 2

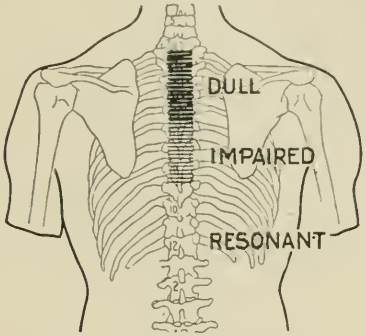

Fig. 3

Fig. 2.—Case I. Mary S., tuberculous lymphadenitis of the cervical and tracheobronchial glands; persistent venous congestion of the upper thorax and neck; cough, dyspnea, and symptoms referable to pressure upon the trachea, primary bronchi, superior vena cava, and pneumogastric nerves. *Absolute dulness and greatly increased pleximeter resistance from the first to the fifth thoracic spine, below which percussion yields loud sonorous hyperresonance.*

Fig. 3.—Case II. James S., tracheobronchial glandular enlargement; tuberculous infiltration (disseminated) of both lungs; small serofibrinous effusion in the left pleural cavity. *Frank dulness from the first to the fifth thoracic spine, with impaired osteal resonance over the vertebral column from the sixth to the eighth thoracic vertebra. Grocco's paravertebral dull triangle not demonstrable.*

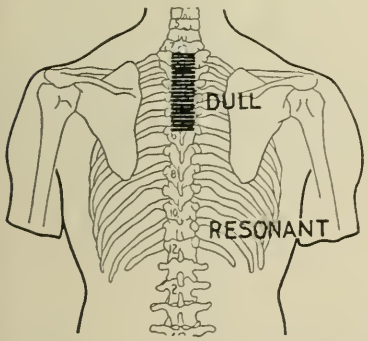

Fig. 4

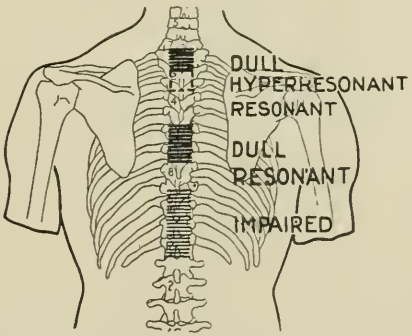

Fig. 5

Fig. 4.—Case III. Rebecca S., disseminated lymphadenitis of the cervical, supraclavicular, and mediastinal glands; postural congestion and tumefaction of the head, neck, and arms; dilatation of the superficial veins of the upper thorax, neck, and face; pressure symptoms relating to the trachea, bronchi, superior vena cava, and inferior laryngeal nerve. *Uniform osteal dulness from the first to the fifth thoracic spine, with clear resonance over the remaining vertebræ of the interscapular zone.*

Fig. 5.—Case IV. Elizabeth S., mediastinal and cervical adenitis; chronic ulcerative phthisis; bilateral pulmonary emphysema. *Striking dulness over the first thoracic spine; hyperresonance over the second and third spines; dulness over the sixth and seventh spines; impaired resonance from the ninth to the twelfth spinous process of the thoracic zone.*

negatived by the interposition of areas of compensatory vesicular dilatation affecting the inner and posterior pulmonary borders—which assumption, if true, must largely invalidate the real usefulness of spinal percussion in emphysematous subjects.

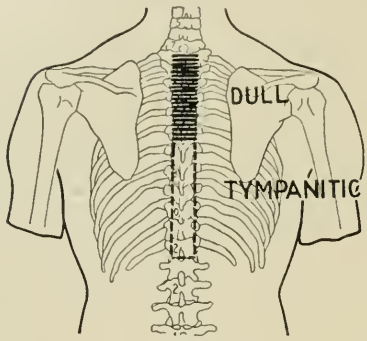

Fig. 6

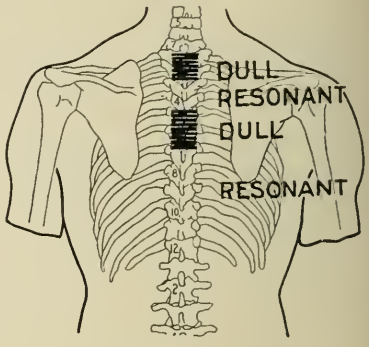

Fig. 7

Fig. 6.—Case V. Annie K., enlarged bronchial glands; chronic ulcerative phthisis; chronic adhesive pleurisy of the left side. *Uniform dulness from the first to the fifth thoracic spine, with clear, low-pitched tympanitic resonance of osteal quality from the sixth to the twelfth process.*

Fig. 7.—Case VI. John D., enlarged bronchial glands; chronic fibroid phthisis. *Dulness over the first thoracic spine; resonance over the second and third spines; dulness from the fourth to the sixth spine; then downward to the twelfth, normal osteal resonance.*

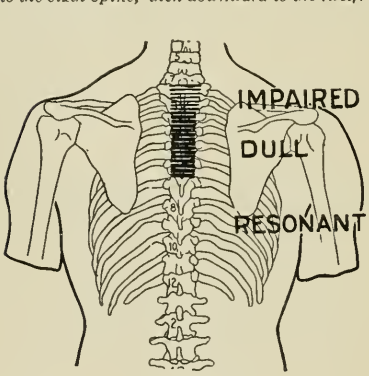

Fig. 8

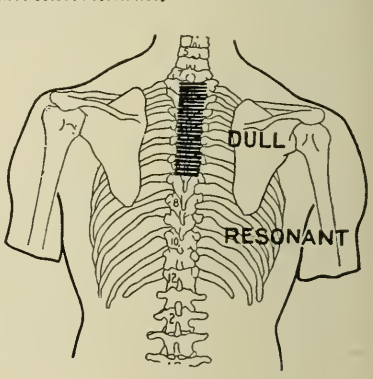

Fig. 9

Fig. 8.—Case VII. William R., mediastinal adenitis; tumefaction of the face and neck; substernal and interscapular pain; dyspnœa; cough. *Impaired resonance over the first and second thoracic spines; striking dulness and increased plexímeter resistance over the third, fourth, and fifth spines; normal osteal resonance over the remainder of the thoracic zone.*

Fig. 9.—Case VIII. Ella E., tuberculous adenitis of the cervical, axillary, and tracheobronchial glands; bilateral dry pleurisy; permanent jugular engorgement; postural congestion of the face and neck; dyspnœa, cough, and mucopurulent sputum; constant pleural pain. *Dulness from the first to the sixth thoracic spine, with normal osteal resonance thence downward to the base of the thoracic zone.*

To sum up the advantages of spinal percussion in the study of obscure mediastinal lesions, it may be concluded that the method is distinctly useful, particularly in that class of cases presenting

indefinite pressure symptoms and mural signs, and that it also has a certain corroborative value in the face of a more tangible clinical

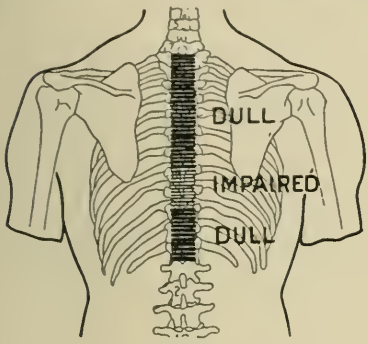

FIG. 10

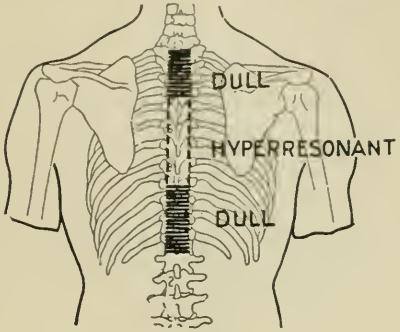

FIG. 11

FIG. 10.—Case IX. Margaret T., enlarged bronchial glands; small effusion in the left pleural cavity; paroxysmal attacks of dyspnœa, dry cough, vertigo, and syncope; substernal and epigastric pain; bulging of the left parasternal region; enlargement of the superficial veins of the upper thorax. *Frank dulness and greatly increased plexímeter resistance from the first to the sixth thoracic spine; impaired resonance over the seventh and eighth; dulness (Grocco's) from the ninth to the twelfth spines.*

FIG. 11.—Case X. John K., enlarged bronchial glands; cardiac hypertrophy; right lateral cardiac displacement; small serofibrinous effusion in the left pleural cavity; constant thoracic pain, suppressed dry cough, dyspnœa, and shallow respiration. *Uniform dulness from the first to the third thoracic spines; vertical (spinal) limb of Grocco's triangle extends from the ninth to the twelfth spines; intermediate spinous processes (fourth to eighth) afford loud hyper-resonance.*

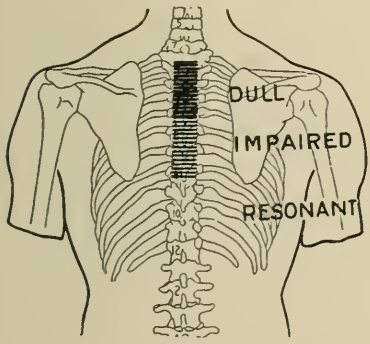

FIG. 12

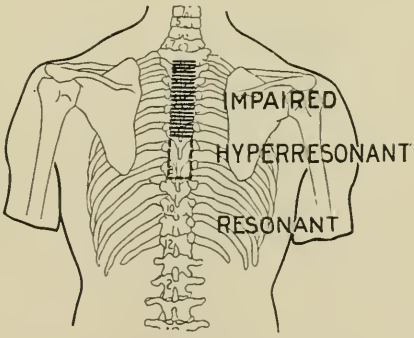

FIG. 13

FIG. 12.—Case XI. James H., enlarged bronchial glands; chronic bilateral adhesive pleurisy. *Uniform dulness from the first to the fourth thoracic spine, with decidedly impaired resonance over the lower four spinous processes of the interscapular region.*

FIG. 13.—Case XII. George R., enlarged bronchial glands; bilateral disseminated pulmonary fibrosis and hypertrophic emphysema; cardiac dilatation; habitual cyanosis; paroxysms of precordial pain, orthopnœa, deep cyanosis, and vertigo. *Impaired resonance (but not absolute dulness) from the first to the sixth thoracic spine, with hyperresonance over the next two processes of this zone.*

picture. In the former category belong those very frequent examples of enlarged bronchial and mediastinal glands, so fre-

quently consecutive to tuberculosis and to other specific infections, in which vague and puzzling pressure symptoms and physical signs are the rule. In such instances the discovery of a dull vertebral

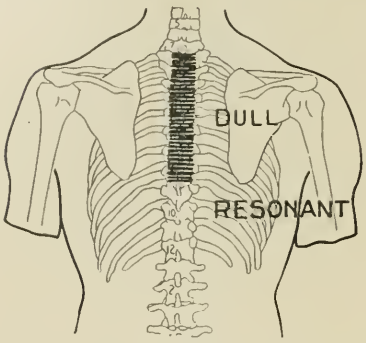

FIG. 14

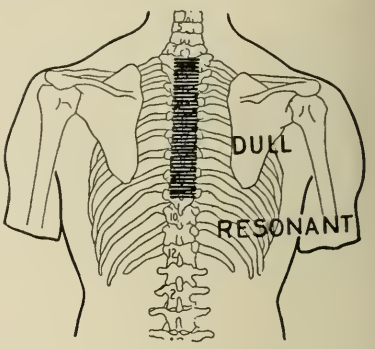

FIG. 15

FIG. 14.—Case XIII. William C., mediastinal neoplasm; bronchial compression; recurrent laryngeal nerve irritation; dyspnœa, tracheal tugging, laryngeal voice, substernal pain, "brassy" cough, acute paroxysms of cyanosis, orthopnœa, and cough excited by physical exertion and by forward inclination of the trunk. *Uniform dulness from the first to the eighth thoracic spine, the greatest degree of impairment being over the upper six vertebral processes.*

FIG. 15.—Case XIV. Robert B., mediastinal gumma; postural congestion and tumefaction of the face, neck, and arms; dilatation and unnatural tortuosity of the superficial veins of the head, trunk, and arms; pressure symptoms referable to the œsophagus, trachea, bronchi, superior vena cava, and vagus; bulging of the sternum and of the left parasternal region; downward and left lateral cardiac displacement. *Uniform absolute dulness from the first to the eighth thoracic spine, with conspicuous exaggeration of plexímeter resistance of the entire thoracic zone.*

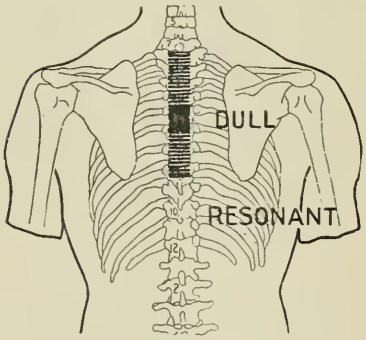

FIG. 16

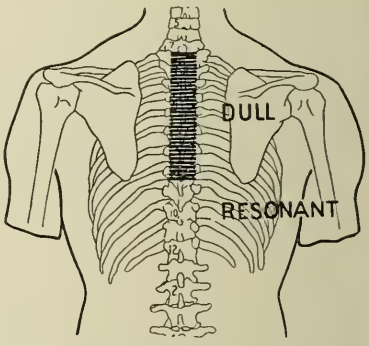

FIG. 17

FIG. 16.—Case XV. John B., fibroid (?) tumor of the middle and posterior mediastinal spaces; downward and left lateral cardiac displacement; compensatory dilatation of the left upper pulmonary lobe; paroxysmal cough, dyspnœa, and shallow respiration; habitual laryngeal voice; constant substernal pain. *Spinal dulness extends uniformly from the first to the eighth thoracic spine, the impaired resonance and increased resistance being especially conspicuous over the fifth and sixth spines.*

FIG. 17.—Case XVI. Edward K., Hodgkin's disease; enlarged mediastinal glands; progressive enlargement of the cervical, axillary, and inguinal glands; symptoms of bronchial and venous compression. *Dulness and greatly exaggerated plexímeter resistance from the first to the seventh thoracic spine.*

strip below the fourth thoracic spine gives a most significant clue, and one ordinarily corroborated by a subsequent *x*-ray examination. Mediastinal neoplasm, aneurysm of the aortic arch, tumor of the

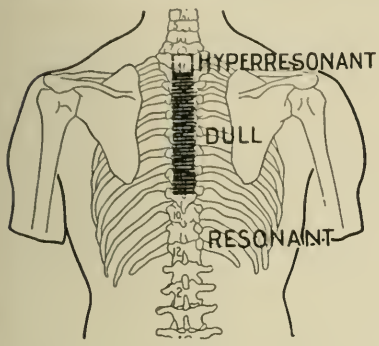

FIG. 18

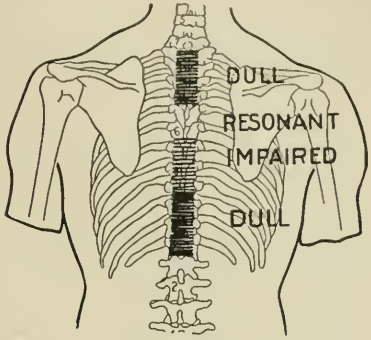

FIG. 19

FIG. 18.—Case XVII. John S., myelogenous leukemia; enlarged mediastinal glands; splenic tumor 51 x 36 cm.; upward displacement of the heart and of the left phrenic dome. *Impaired resonance from the second to the eighth thoracic spine, with an unnaturally high-pitched and clear percussion sound over the first thoracic spine.*

FIG. 19.—Case XVIII. Henrietta S., myelogenous leukemia; enlarged mediastinal glands; splenic tumor 42.5 x 26 cm.; retrodisplacement of heart. *Dulness over the first, second, and third thoracic spines; clear resonance over the fourth and fifth; impaired resonance and increased plexímeter resistance over the sixth, seventh, and eighth; frank dulness from the ninth to the twelfth thoracic spines.*

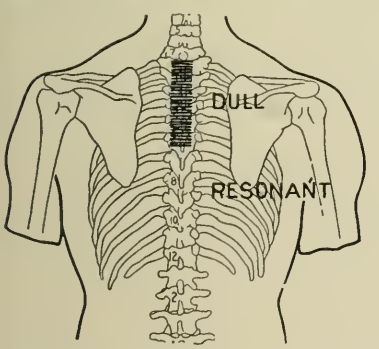

FIG. 20

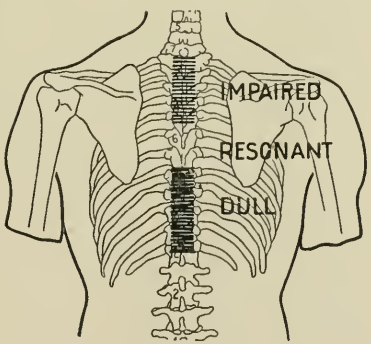

FIG. 21

FIG. 20.—Case XIX. Laura F., myelogenous leukemia; enlarged mediastinal glands; splenic tumor 30 x 22.5 cm.; cough, dyspnœa, and other signs of tracheobronchial pressure. *Impaired resonance and increased plexímeter resistance from the first to the fifth thoracic spine, with clear percussion sound below the latter level.*

FIG. 21.—Case XX. Henry S., lymphatic leukemia; enlarged mediastinal glands; splenic tumor 38 x 21 cm.; enlarged cervical, axillary, and inguinal glands. *Impaired resonance from the first to the fourth thoracic spine; resonance over the fifth and sixth; dulness from the seventh to the twelfth spine.*

œsophagus, atelectasis, consolidation of the lung, and pleural effusion also may encroach backward so as to damp the spinal sonorousness; these lesions should always be taken into account as potential factors of unnatural dulness over the thoracic vertebræ.

RESEMBLANCES BETWEEN THE CLINICAL EFFECTS OF PNEUMOCOCCIC AND MENINGOCOCCIC INFECTIONS.

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The systematic employment of blood culture methods in all cases of obscure infections has shown that the pneumococcus is frequently the cause of clinical pictures which differ widely from one another and from the commonest effect of this organism, croupous pneumonia. However, careful review of a very large number of these cases will show that there are certain features which recur with great constancy, no matter how much the cases may differ from one another. These phenomena I have come to regard as the specific effects of the pneumococcus, and from them I have been able repeatedly to predict the results of the blood culture. These effects are four in number, and while no one of them is peculiar to the pneumococcic infection, the combination of three of them, much less all four of them, is seen in the course of no other infection except one.

The first of these is the acuity of the onset of the symptoms of infections. Pneumonia is conspicuously a disease of sudden onset, the patient in most instances being able to fix not only the day, but in many instances the hour, and even the minute, when the symptoms began. This is equally true of other forms of pneumococcic infections. The sudden onset of symptoms is, of course, not peculiar to this infection, for there are numerous other organisms which initiate symptoms equally suddenly.

The second effect is the polymorphonuclear leukocytosis, and it, too, is common to many other infectious processes, some of which are also characterized by sudden onset, although there are several of the very acute infectious processes unaccompanied by leukocytosis.

The third effect of the pneumococcus, an herpetic eruption, is much more highly specific, being present in a large, though not constant percentage of the cases, and being common to but two other infections, namely, malaria, a process due to an organism of so widely differing nature that no consideration of it is here necessary, and secondly, meningococcic infection, in which, like the pneumococcic infections, herpes is very common. Moreover, the herpetic eruption occurs usually over the same nerves, one or the other of the lower branches of the fifth cranial nerves, although in either it may occur over any other nerves. The herpetic eruption in the cases of meningitis is not to be referred to the meningitis, for in no other form of inflammation of this membrane

is herpes common, except in those cases which are due to the pneumococcus.

When we consider these three effects, not singly, but in a group, one finds that while there are many infections causing a combination of the first and second and one infection causing the combination of the first and third, there is no other infectious disease in which all three of them occur, except in cases of the so-called epidemic meningitis.

Now, if to these three one other is added, the resemblance between the two processes is not increased by any mere arithmetical, but by a considerable geometrical, progression.

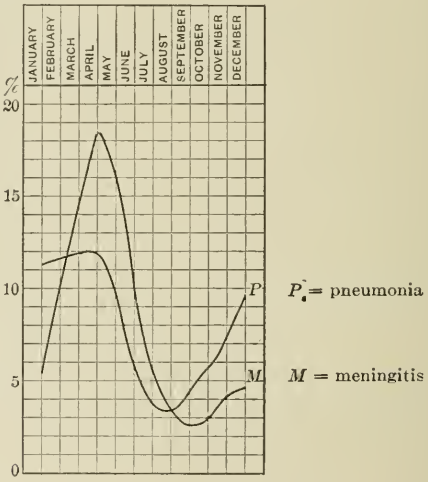
For years the extremely low chloride excretion in the urine in cases of pneumonia has been appreciated and has been utilized both as a means of diagnosis and prognosis, but it is not yet as generally known as it should be that in other forms of pneumococcic infections, such as tonsillitis, endocarditis, meningitis, sepsis without localization, the same extremely low chloride excretion is found. This I regard as a fourth specific effect of pneumococcic infection.

Naturally this has suggested a study of the urine of cases of meningitis as to the chloride excretion. No light upon the subject is thrown by a study of the literature, for as far as I have been able to find no mention is made of it, but in all the cases of epidemic meningitis which I have seen of late not a large number, it is true, have shown the same low chloride excretion which is shown by cases of pneumonia. We find, therefore, that while there are several infectious processes which show combinations of two of the specific effects of pneumococcic infections, there is no other except the meningococcic infection which shows a combination of three, much less one of all four of these effects.

These, however, do not exhaust the similarities between the two infections. There are certain complications which occur with greater or less frequency with pneumococcic infections, such as endocarditis, pericarditis, arthritis, and otitis media, and it is interesting to note that these same complications occur also with the meningococcic infections. Not much stress can be laid upon this, for these same things occur in many other infections. There is, however, one point upon which I should like to lay some stress, namely, that of the endocarditis. It has long been my opinion, based purely upon clinical observation, that endocarditis of pneumococcic origin occurs when the cocci are of low virulence, and this opinion has recently been experimentally confirmed by Rosenau. If, then, the suspicion is correct that the meningococcus is a pneumococcus of lowered virulence, then one would expect to find endocarditis more common as a complication of the epidemic meningitis than it is in pneumonia in the young. Pneumococcic endocarditis as a complication of pneumonia of the old or of those having an antecedent valvular lesion must be excluded from con-

sideration. One must also make allowance for the fact that certain of the cases of meningitis run so acute a course that there is not time enough to recognize clinically a developing endocarditis. Nothing very definite can be said upon endocarditis as a complication of meningitis, because of the lack of data in literature; even in the reports upon autopsies made but few details are stated. I feel sure that the lessened mortality from this disease, such as we seem warranted in expecting from the antimeningococcic serum, will be accompanied by numerous reports of subsequent endocarditis.

The frequency with which meningitis is accompanied by pneumonia may have some bearing upon the question of the relationship of the two organisms, but here again we lack data. Practically all of the early literature upon the question is valueless, and no doubt many of the instances in which the two processes were associated are due to the pneumococcus, which admittedly may cause either or both.



Another point of resemblance between the two diseases is that of their seasonal distribution. The accompanying curves (see chart) show the percentage distribution according to months. They are rather grossly alike, but would probably more closely resemble each other if they were upon anything like a similar number of cases. The pneumonia curve by its smoothness suggests that it is based upon a large series of cases, and this is true, for it is made up from tables including over 600,000 cases, while the meningitis curve is based on but 6000 bacterially confirmed cases.

It was formerly stated that the epid mic meningitis did not end by crisis, one of the most striking peculiarities of the pneumococcic infections, and this was true, but since the introduction of the

Flexner-Jobling serum, this is no longer so, for all who have observed the effects of this serum in many cases report endings as critical as any seen in pneumonia.

Another interesting thing about the epidemic meningitis is that cases occur sporadically separated from all known sources of infection and this, too, though the meningococcus is an organism known to have but little resistance to outside influences. This fact is difficult to explain if one admits that the meningococcus is an independent organism, but not so difficult if one assumes that it is merely a modified pneumococcus, an organism which is practically ubiquitous.

It must be admitted that many, possibly the great majority of the cases of sporadic epidemic meningitis are not truly instances of this disease, but even so, there seems nowhere any disposition to question the occurrence of sporadic cases.

In contrast with these clinical resemblances one finds certain others of clinical differences which are significant, namely, the striking difference in the age distribution of the two diseases, one being conspicuously a disease of childhood, while the other occurs most often in adult life. The second point is the frequency with which the meningitis is associated with a hemorrhagic diathesis, as shown by petechial eruptions. Such a diathesis is not unknown in pneumonia, but it is extremely rare. I can recall but two instances of it, if one excludes the not infrequent cases of early hemoptysis in pneumonia.

It appears to me that the points of resemblance between the two infections are sufficiently striking to warrant serious consideration by the bacteriologists who, without exception, I believe, regard the two causal organisms as absolutely distinct, although admitting that they have many points in common, so many, indeed, that their differentiation is so difficult that much of the early work upon the bacteriology of meningitis and notably that of the nose and pharynx in this disease must be disregarded.

THE DIAGNOSIS AND SURGICAL TREATMENT OF ACUTE PANCREATITIS.

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The diseases of the pancreas are, at the present time, being subjected to an ever-increasing amount of clinical observation and to experimental research of the most varied forms. Especially is this true of acute pancreatitis, a disease which in its several varieties

touches upon some of the most important problems with which the abdominal surgeon and the physiologist of today must deal. Acute pancreatitis is a rare disease, although close study reveals the fact that many cases must in former times have been overlooked. It presents difficulties in diagnosis and treatment, and it is but rarely that either the surgeon or the medical man has the opportunity of seeing a large series of cases. I consider myself fortunate in having had the opportunity of observing closely several, and of operating on six cases. These are as follows:

Case I.—A white male, aged forty-four years, in the early part of 1901 suffered an attack of epigastric pain, lasting three hours and followed by diarrhœa. Pain recurred about every six months, was cramp-like in character, and relieved usually by mustard plaster. From 1901 to 1902 he felt very well, but afterward had pronounced symptoms of indigestion, loss of appetite, vomiting, belching of gas, and paroxysmal pain in the region of the stomach. During the week previous to the last attack the abdomen was distended, there was constipation, and some yellowness of the sclerotics. The diagnosis by his attending physician was intestinal obstruction. Operation showed acute inflammation of the pancreas, which I believe supervened upon a chronic pancreatitis. The gall-bladder was drained, but no stones were found. The patient recovered and is perfectly well at the present time, having had no further trouble referable to the abdomen.

Case II.—White, male, aged forty-two years, admitted to the German Hospital November 11, 1902.

The patient's past medical history is negative, except for occasional attacks of indigestion. His general health is good, but for the last twenty-five years he has had attacks of pain in the epigastrium lasting three to four hours, and accompanied by jaundice. No history of constipation was noted. Three days before admission he was seized with abdominal cramps, and the next day had epigastric pain and vomiting associated with much belching. Bowels did not move. On admission the patient was suffering with violent paroxysms of epigastric pain, was belching frequently, and vomiting. The vomitus was a clear, green, bile-stained fluid. The patient's bowel movements while in the hospital were clay-colored.

Physical examination showed the presence of slight icterus. The abdomen was markedly distended and tympanitic, the distention being most marked in the epigastric region. The *x*-ray examination showed the excursions of the diaphragm to be somewhat limited, and revealed a shadow in the region of the pancreas. The temperature on admission was 102.8° F., the pulse 104, and the respirations 30. The leukocyte count, November 14, was 8200, and on November 21, 1902, 13,850, with 81.3 per cent. of polymorphonuclears. The stools showed free fat. There was no glycosuria at any time.

The patient appeared to improve somewhat. The pulse dropped

to between 86 and 96 to the minute; the respirations to 24, and the temperature to between 99.6° and 101° F. before the operation.

On November 22, 1902, the patient was operated upon, under ether narcosis. The incision was made through the upper portion of the right rectus. Some fat necrosis of the gastrocolic omentum was found. The gastrocolic omentum was opened, exposing the pancreas, which was found to be diseased, with the head of the gland thickened. Some hemorrhage was present. The pancreas was drained at the site of the hemorrhage with a rubber tube, around which was packed sterile gauze. Death occurred soon after the operation.

Autopsy. The preperitoneal fat and all the fat in the abdomen, omentum, and mesentery was studded with little areas of fat necrosis. The pancreas was much diseased. The tail was gangrenous and surrounded by a reddish, purulent fluid. The head and body of the organ had remained relatively normal. The lesser peritoneal cavity contained much purulent, reddish-yellow fluid, containing broken-down pancreas. The gall-bladder was full of calculi, but it was impossible thoroughly to examine the biliary ducts owing to adhesions and necrosis. About the sloughing gangrenous pancreas there were adhesions; but there was no general peritonitis.

The salient features of this case, to which I shall refer more in detail in the general discussion, are: (1) General abdominal distention, most marked in the epigastrium; (2) the presence of biliary vomiting and frequent belching; (3) the drop in pulse rate, respiration, and temperature, with improvement in the patient's general condition, after the severe initial symptoms; (4) the presence of fat necrosis; (5) the presence of biliary calculi; (6) the involvement of the tail of the pancreas almost exclusively; (7) the presence of free fat in the feces and the absence of glycosuria; (8) the absence of free fluid in the abdomen; and (9) the mistake of not opening the lesser peritoneal cavity through the left loin posteriorly, instead of transperitoneally.

Case III.—C. K., white, female, aged sixty-one years. Admitted to the German Hospital January 20, 1905.

The patient was admitted with the diagnosis of acute intestinal obstruction, and was operated upon immediately. No history was obtainable. On admission the pulse was 76 to the minute and the temperature was 99° F.

Under ether narcosis, an incision was made in the left inguinal region, in the belief that the sigmoid was at fault. The descending colon and sigmoid were examined and found normal, and the original wound was closed. The abdomen then was opened in the median line, the incision extending from the ensiform to the pubis. The entire intestine was searched for an obstruction, but none was found. At the greater curvature of the stomach near the pylorus a dense mass of adhesions was found, with hemorrhage beneath. The adhesions

were separated and the pancreas was found hard, nodular, enlarged, and the seat of a large hemorrhage. Four pieces of gauze were carried to the head of the pancreas and a glass tube was placed in the pelvis. An antiseptic dressing was applied. The patient died fifteen hours after the operation.

Postmortem. The pancreas was 24 x 8 x 4 cm. in size and weighed 290 grams. It was dark red in patches, and densely adherent to the stomach and the transverse colon. Areas of fat necrosis were found in the surrounding tissues. The gall-bladder contained bile and about fifty stones; the ducts were patulous.

This was evidently one of those extremely acute cases of pancreatic hemorrhage, that is, hemorrhagic pancreatitis, referred to by some authors as fulminant. The operation took place within a day of the onset of the illness, even before fat necrosis had an opportunity to occur, yet the very severity of the disease rendered the prognosis hopeless. Especially to be noted is the diagnosis of acute intestinal obstruction. As will be seen on reading case reports of this disease, this diagnosis is very commonly made—far oftener than the true one. We find in this case also: (1) Fat necrosis; (2) the presence of calculi; and (3) a very slow pulse rate—76—with a temperature of 99° F.

Case IV.—White, female, aged forty-one years. Admitted to the German Hospital October 17, 1905.

No history of tuberculosis or malignancy was obtained. The patient had always had general good health. Nine weeks before admission she became gradually ill, complaining of pain in the right side of the abdomen referred to the back, epigastrium, and right iliac fossa. Three weeks ago she had severe abdominal pain in the right side of the abdomen, but most severe in the upper portion. The pain was followed by vomiting and was almost constant. Some soreness in the upper abdomen was also noted. The nausea and vomiting had been more or less continued. On admission the patient's abdomen was distended, especially above the umbilicus. Rigidity of both recti muscles was noted, but most marked above the umbilicus on the right side. There was an indefinite mass in the epigastrium, which was moderately tender. On admission the temperature was 100° F., pulse, 76. While the patient was in the hospital the leukocytes varied from 8600 to 32,900, and the hemoglobin from 45 to 68 per cent. Free fat and occult blood were found in the stools.

Operation, October 18, 1905. Under ether narcosis, an incision was made in the median line above the umbilicus; the stomach was found distended and pus escaping through the foramen of Winslow. The wound was closed and a second incision made along the right costal margin, exposing the lower margin of the liver adherent to the stomach. The adhesions between the stomach and the liver were separated, and the gastrohepatic omentum was found to be thickened and œdematous. An aspirating needle was passed through the thick-

ened gastrohepatic omentum in the direction of the lesser peritoneal cavity, and blood and pus were obtained. A free incision was then made, allowing the escape of considerable pus. A rubber tube, surrounded with gauze, was then placed in the lesser peritoneal cavity. The gall-bladder was searched for but not found. After the operation the patient developed a septic temperature. The leukocytosis continued, and she left the hospital in poor condition, with a discharging sinus. She died after some weeks of continued sepsis.

In this case we may note particularly: (1) Gradual onset of the illness, indicative of a subacute suppurative process rather than an acute necrotic one; (2) continued vomiting; (3) low pulse rate—76—before operation; and (4) an indefinite mass in the epigastrium.

Case V.—White, male, aged twenty-seven years. Admitted to the German Hospital March 18, 1908.

One year before admission the patient had four or five attacks of abdominal pain, accompanied by jaundice. Two and one-half weeks before admission he had a severe attack of epigastric pain, accompanied by nausea and vomiting. The pain continued up to the date of admission, with frequent exacerbations. It started in the epigastrium, and was referred all over the abdomen, and to the back and shoulders. The patient has been jaundiced more or less ever since the onset of this attack.

On admission, the patient was jaundiced, the respiratory excursions were limited, and the respirations short. The liver extended from the sixth interspace to two fingers' breadths below the costal margin in the mamillary line. There was slight epigastric fulness and spasticity of both recti muscles in this region. Some tenderness was noted over the entire epigastrium, and it was quite marked over Mayo Robson's point. The pain continued without relief up to the time of operation. The patient's temperature on admission was 98.4° F., and remained febrile during the entire course of the illness, but only for about three days after operation, with a maximum of 100.4° F. The pulse on admission was 88.

The operation was performed on March 21, 1908, under ether narcosis. An incision was made through the right rectus. The gall-bladder was found adherent to the colon and omentum, and contained calculi. Posterior to the stomach there was a soft fluctuating mass about the size of two fists, pushing the stomach forward. A finger placed in the foramen of Winslow found this to be in the position of the pancreas. The gall-bladder was walled off with gauze pads and aspirated; 40 c.c. of mucopurulent fluid was removed. This was sterile, as shown subsequently by culture. The gall-bladder was opened and four large and twenty-four small stones were removed from it and the dilated cystic duct. A tube drain was introduced into the gall-bladder, and it was sewn to the peritoneum. The choledochus was found to be patulous. The wound was closed and the patient placed

on his right side and an incision made in the left loin, extending down 7 cm. from the costal margin, and just external to the outer border of the erector spinæ. Within the fatty capsule of the kidney there was much fat necrosis. An abscess was evacuated in the location of the pancreas, and about half a liter of bloody purulent fluid escaped. The cavity was drained with a large rubber tube and two pieces of gauze.

The patient made an uneventful and practically afebrile recovery. The drain was left in the gall-bladder eleven days, and that in the posterior incision for several weeks, although the drainage gauze in this incision was removed in six days. The discharge from this wound was found to be very irritating to the skin.

The points of interest here are: (1) The slow pulse and afebrile course after operation; (2) the presence of biliary calculi; (3) the presence of fat necrosis around the abscess cavity; and (4) the irritating character of the pancreatic discharge.

Case VI.—H. M. N., a white male, aged thirty-nine years. Admitted to the German Hospital December 28, 1907.

The case, which has been reported by Dr. Jurist,[1] was one of gangrenous pancreatitis, in which following a posterior incision purulent and necrotic material was removed from the region of the pancreas. Ten days after the operation, and again a week later, large pieces of necrotic and gangrenous pancreas were discharged through the wound. Thereupon the patient's condition improved markedly, and eventually he made a perfect recovery. He now enjoys excellent health, his metabolism apparently is undisturbed, and he has no glycosuria.

Of special importance in this case are: (1) The primary shock with a slow pulse; (2) extreme prostration thereafter, with signs of profound toxemia; (3) the operation in the second stage; and (4) the use of the posterior incision alone.

In discussing this series of cases, it will be admitted that in each case there was an acute pancreatitis. It is true that they are of various types. Thus, in Case II we have an example of a slowly progressing type, of the hemorrhagic variety. This may perhaps be considered one of those cases in which the inflammation preceded the hemorrhage by some time, as the latter does not furnish the changes most marked pathologically, nor is the course that of an acute hemorrhagic case. Case IV represents the ultra-acute hemorrhagic pancreatitis of Mayo Robson's classification, in which the hemorrhage is the primary factor. The rapid course and extreme disorganization of the gland itself make this apparent. This represents that class of cases which is least promising for any form of surgical intervention—indeed, when the pancreatic disintegration has

[1] Amer. Jour. Med. Sci., 1909, cxxxviii, 180.

reached the stage which it had in this instance, it is almost hopeless to expect a recovery. Cases IV and V represent the acute suppurative variety of pancreatitis: not a localized abscess in the pancreas, but a more or less diffuse suppuration with involvement of the omental bursa. Case VI is of the gangrenous type. Doubtless this commenced as an acute necrosis also, probably hemorrhagic in type, which involved nearly all of the gland, as is shown by the extent of the gangrene. The subsequent well being and perfect assimilation of the patient would lead us to suppose that a portion of the head of the pancreas was entirely unaffected by the disease.

Etiology. As to the etiology of acute pancreatitis much has been written, largely from the standpoint of experiments. Clinically several factors have been mentioned. Many authors have called attention to the fact that people in whom acute pancreatitis is found are, as a rule, of middle age and inclined to stoutness. As regards the age, this would hold true in my cases, with the exception of Case V. In Cases II, V, and VI the patients were, however, very well developed, and not given to excess of adipose tissue. In Case VI, there was the history of a diabetic parent, perhaps only a coincidence, but nevertheless rather striking.

Opie[2] has called attention to the association of various forms of pancreatitis with gallstones, especially those situated in the choledochus. In Cases II, III, and V, I found gallstones, but in no case obstructing the choledochus. In Case IV the gall-bladder was not found; it was so bound down by adhesions that the search for it in the presence of so much free pus, would have been dangerous. In Case VI the bile ducts naturally could not be inspected; but it seems reasonable to assume that stones were present, in view of the many attacks of pain resembling biliary colic.

Thus, in six cases of acute pancreatitis, four (67 per cent.) had gallstones. In two additional cases personally communicated to me, which have recently occurred in this city, gallstones were not present. It is noteworthy that in the three cases in which gallstones were actually found there was no stone in the choledochus. I have a number of times found chronic pancreatitis associated with a stone in the common duct.

My experience, as regards biliary calculi, coincides with that of most authors. The investigations of Opie have already been mentioned. The only investigator of prominence who disagrees with these conclusions is Truhart,[3] who, in the analysis of a large number of cases, found that a smaller percentage of patients with acute pancreatitis had gallstones than those who died from other causes.

Fitz, Opie, and most American and British authors have inclined to the belief that the causative factor in acute pancreatitis is most often some form of infection, while Chiari and his school incline to

[2] Diseases of the Pancreas, 1905.

[3] Pankreas Pathologie, Wiesbaden, 1902.

the theory of a chemical autodigestive process. The experimental results of various investigators have differed greatly on this subject. While Opie, Fitz, and others, as mentioned, give great prominence to the factor of infection, Truhart, in an analysis of 74 cases of acute pancreatitis, found that in only 14 could microörganisms be demonstrated. Naturally this refers to the pancreatitis itself, for when pus has once formed and an abscess is present, this is likely to become secondarily infected. Then again, the fact that microorganisms cannot be shown by culture does not eliminate them as causative factors. In one case (V) Bacillus coli as demonstrated in the pus.

It seems reasonable to assume, however, when gallstones are present and not in the choledochus, that whatever causative action they may have had has been exerted by the concomitant infection. A curious feature in this connection is that in the case mentioned the apparently purulent fluid contained in the gall-bladder, showed no bacteria, while the colon bacillus was found in the pancreatic abscess.

Case III is an example of those in which commonly no bacteria are found, and seems to be a case of primary hemorrhage into the pancreas.

Pathology. The pathology of acute pancreatitis has been the subject of much discussion, and bears an intimate relationship to the question of etiology. So far as my records go, there was nothing to throw any light on the presence of an autodigestive processes, which so many German authors, notably Doberauer[4] and Guleke[5] have brought into the foreground as the initial process in acute pancreatitis. On the whole, I incline to the theory of infection rather than that of primary chemical change in the pancreas. So many substances act as stimuli to the disintegration of the pancreas by so-called autodigestion that it seems quite likely that bacteria can also do this. And, indeed, Hekma[6] furnished experimental proof of this fact.

Sailer and Speese[7] found that a toxic substance was present in the blood of dogs suffering from acute pancreatitis experimentally produced by the injection of sterile oil into the pancreatic duct followed by ligation of the duct. Williams and Busch[8] found "anatomical and experimental evidence that made it seem probable that some cases of acute pancreatitis may be caused by regurgitation of duodenal contents into the diverticulum of Vater, the way having been opened by the passage of gallstones."

Diagnosis. The diagnosis of acute pancreatitis is the most important question that confronts us. Even a cursory examination of

[4] Verhand. Deut. Ges. f. Chir., 1906, xxxv, 280.
[5] Arb. aus d. chir. Klin. d. K. Univ., Berlin, 1906.
[6] Quoted by Doberauer, Arch. f. Phys., 1904, p. 334.
[7] Trans. Assoc. Amer. Phys., 1908, xxiii, 540.
[8] Jour. Med. Research, 1907.

recorded cases will show that in 90 per cent. the correct diagnosis has not been made, except at operation or autopsy. Indeed, even at operation the condition has been overlooked in not a few instances (as in a case lately reported as one of a series by Noetzel[9]).

The symptomatology, therefore, must first be considered. Fitz[10] gives us the best concise statement of the classical symptoms: "Acute pancreatitis is to be suspected when a previously healthy person, or sufferer from occasional attacks of indigestion, is suddenly seized with violent pain in the epigastrium, followed by vomiting and collapse, and in the course of twenty-four hours, by a circumscribed epigastric swelling, tympanitic or resistant, with slight rise of temperature."

The pain, as a rule, is severe, and generally referred to the epigastric region. It was so in all of my cases (II, III, V, VI) in which this is recorded. It has been asserted by some that it is more often to the left of the median line, and while this may be a point of some value when it is present, the absence of localization of the pain to the left side should not influence us in the least in estimating the importance of this symptom. The existence of severe epigastric pain was noted in several cases reported by Mayo Robson, in the two reported by Bornhaupt,[11] in a case reported to me in a personal communication by Dr. Chase, of Philadelphia, and in several of those reported by Doberauer and Noetzel, and those of Murray[12] and Bell.[13] On the other hand, in several of the cases reported by Doberauer, the pain was referred to in a more general way as being abdominal. Indeed, in one case operated by Noetzell, the patient spoke of the pain being in the lower abdomen to such an extent that the operator made his first incision for a supposed pelvic condition.

There is never any doubt concerning the severity of the pain wherever located. It practically always calls for the guarded use of opiates, although these are often without avail. This was true in Case VI, of my series in which the pain was agonizing, and but little affected by several hypodermic injections of morphine. Associated with the pain, but a far less constant symptom, is epigastric tenderness. In many cases it is but slightly marked, although careful examination will usually elicit it. It becomes more marked if the case progresses to suppuration, especially with the formation of a mass, which is practically always tender. In the same way epigastric muscular spasticity may occur as the result of the local condition.

Soon after the onset of the pain there is vomiting—a practically constant symptom. The vomiting, in some instances, lasts but a few hours or perhaps a day—as in Case V; and in others it becomes almost uncontrollable and progressively worse. This is a diagnostic

[9] Beitr. z. klin. Chir., 1908, lxxv, 735.
[10] Quoted by Robson and Cammidge, Diseases of Pancreas, 1908.
[11] Deut. med. Woch., 1908, xxxiv, 1306.
[12] Trans. Amer. Surg. Assoc., 1902, xx, 219.
[13] Ibid., 1904, xxii, 103.

sign of but little value, as it is common to most acute intra-abdominal conditions. Attention has also been called to the fact that it is, as a rule, biliary in character. Indeed, vomiting, especially when persistent, is one of the most misleading symptoms of the disease under consideration, as it often serves to make a diagnosis of intestinal obstruction seem the correct one. I have, however, never noticed fecal vomiting, which is always present in the late stages of intestinal obstruction.

Persistent and uncontrollable belching and hiccoughing are marked symptoms of acute pancreatitis. They are often sufficiently severe to draw the physician's attention to the possibility of some trouble near the diaphragm, and to help to a correct diagnosis. They were especially noted in Case VI of my series, and were far more troublesome than the vomiting itself.

Collapse in the acute onset of pancreatitis is often very marked. In some of the cases, operated and unoperated upon, it may pass very quickly on to a fatal termination. In others, the patient recovers from it sufficiently to last for a few days, in very rapid cases, or even recovers therefrom entirely when a localized pancreatic suppuration supervenes. Fitz and others have called attention to the marked cyanosis which occasionally accompanies this collapse, and it has been referred to as a diagnostic feature. Its cause is still unexplained. It was not noted in any of my cases, but was a feature in the case personally reported to me by Dr. Chase.

A possibly important symptom in the onset of acute pancreatitis is the frequent presence of a slow pulse even during the collapse, and until a septic condition supervenes. Indeed, it may even persist under these circumstances. I have had several instances of this fact sufficient to convince me of its *occasional* significance. Thus, in Case II, the pulse was never very rapid and dropped even to below 100 when the other symptoms abated. In Case III, an ultra-acute one, the pulse on admission was 76. References to the suppurative and less acute cases, Nos. IV and V, show a comparatively low pulse rate. In Case VI, during the primary agonizing pain and collapse the pulse was about 80 to 90 per minute, even when the temperature dropped below 96° F. In this instance, however, the pulse became very rapid and feeble as soon as the marked pancreatic toxemia had a chance to manifest itself.

So far I have been discussing practically only the symptoms immediately associated with the very onset of the disease. If the patient survives the initial shock or hemorrhages, as the case may be, certain other conditions make themselves manifest. There are evidences of intestinal paresis. Besides the vomiting and belching already spoken of, there is frequently in the first two or three days, interference with the passage of gas and feces. After several days repeated enemas will usually give quite copious and bulky stools. Associated with these conditions there is intestinal and abdominal

distention, with corresponding distress. Very often this seems to be largely colonic, affecting principally the transverse and descending colon, the small intestine being involved to a lesser extent.

Slight jaundice, as noted in Case VI for forty-eight hours, and in Case II and Case V (with cholelithiasis) is of frequent occurrence in pancreatitis. As a diagnostic sign it would serve only the purpose of calling our attention to some lesion in the upper abdomen.

The formation of a more or less well-defined epigastric mass, apparently deep, usually somewhat tender, and often to the left of the median line, is very suggestive of pancreatitis, when found in conjunction with some of the other significant symptoms. Formerly, when cases were kept under observation longer, because of failure to diagnosticate them and reluctance to operate, except in the face of very definite indications, this was more often seen than it is now, when the tendency is to operate somewhat earlier. Case VI showed this feature most markedly, that is, the presence of a deep indefinite resistant area in the left epigastrium. Some suggestion of it was noted in Case IV. In the case reported to me by Dr. Chase this feature was most marked.

Naturally the presence of a tumor, such as this, can hardly be expected in those instances of acute pancreatitis which quickly terminate fatally. The swollen, engorged, or hemorrhagic pancreas itself is hardly ever palpable. The hemorrhage in and about the pancreas itself scarcely ever gives us such a considerable mass that palpation is possible, yet even this must be considered within the range of possibility.

Finally, there is to be mentioned that occasional occurrence of dulness or of impaired resonance and breath sounds over the lower lobe of either lung, usually the left. This occurred in Case VI of my series, and led to a diagnosis of pleurisy as a complication. The condition was present at the time of operation, but then was not interfered with. I believe it was due to infection through the diaphragm.

While the diagnostic points in acute pancreatitis are not uniform, there are enough, in at least a certain percentage of cases, to enable us to diagnosticate the condition.

Differential Diagnosis. The differential diagnosis is a matter of some difficulty in most cases. In the majority of instances in which an incorrect diagnosis has been made, it has been that of acute intestinal obstruction. It is not difficult to see why this is so. In both conditions we have the sudden onset, the violent, cramp-like pain, a slow pulse in the beginning, some distention which gradually increases, and vomiting following the pain, with stoppage of gas and feces. The similarity is heightened by the fact that in pancreatitis the distention is largely colonic, and this often leads to a suspicion of an obstruction low down. At the onset of the disease it may be impossible to differentiate the conditions, yet the uncertainty should not

last very long. In the first place, it is rare to find shock, with occasional slow pulse and cyanosis in intestinal obstruction. The pain rather, where extremely severe, causes the patient to look blanched and exhausted. Moreover, it is, as a rule, localized to some point in the lower abdomen, rather than in the epigastrium, as in pancreatitis. Yet, as I have mentioned in discussing the symptom of pain, atypical locations thereof in pancreatitis are not entirely unknown. As the case progresses for some hours the distinction becomes slightly plainer. The vomiting in obstruction is one of the cardinal symptoms. It is persistent, severe, and becomes progressively worse. The vomitus becomes foul in a short time. In pancreatitis the vomiting is bilious in practically every instance; after a short time it becomes less frequent. Far more distressing is the hiccoughing and belching associated with pain. These seem to be much dwelt upon in the case histories of pancreatitis, and were illustrated to a marked degree in Case VI of my series. They are not so prominent or striking in cases of obstruction. In pancreatitis the distention is generally colonic and not of high grade. In obstruction it may or may not be localized to the colon, is more marked and more quickly progressive. In pancreatitis the ileus may be absolute at first, yet often high enemas result in passage of gas and true bowel movements. In acute obstruction we can, by the same means, obtain only emptying of the lower part of the larger bowel, and the passage of flatus, if it occurs at all, is very limited.

In pancreatitis the development of epigastric pain occurs frequently and often early, while in obstruction we have practically never a localization of these signs to the epigastrium. Slight jaundice often develops in pancreatitis; in obstruction it is absent. The rapid development of free fluid in the abdominal cavity would point to pancreatitis. If the case should progress for some days, we have in pancreatitis the gradual appearance of signs of omental bursitis, an upper abdominal lesion, associated with a severe toxemia, but with subsidence of the signs of ileus, vomiting, etc. In obstruction there is also a toxemia from the absorption of stagnated bowel contents, and the signs of intestinal stasis plainly manifest themselves if continued; finally there are fecal vomiting, increasing signs of peritonism, marked distention, and tympany.

The mistake of diagnosticating a case of acute pancreatitis as obstruction was made in Case III. With the increased understanding of the subject which we now have, and perhaps a slightly more extended study of the case (which was operated upon at once on admission), the diagnosis, I believe, could perhaps have been properly made. It is a most noticeable feature of all the more extended series of cases of pancreatitis reported, that in the earlier ones the diagnosis was but rarely made, while in the later ones seen by any one man it was practically always strongly suspected, if not definitely decided upon.

Acute gastroduodenitis has been mentioned as possibly to be con-

fused with pancreatitis. It must, indeed, be a severe type of this illness which would simulate the graver lesion. Indeed, it seems hardly more than necessary to consider the symptoms of each to arrive at a diagnosis, should this be the differential question.

Biliary colic, with or without slight jaundice, would give us many points of similarity to the initial pain of pancreatitis. At the time of the onset of the pain a distinction might be impossible. Gall-stone colic is far more common than acute pancreatic lesions are, and they may be associated, as in Case V, so that the physician would think of that condition first. As a rule, the location of the pain to the right of the median line, its radiation, and the failure of other pancreatic symptoms to supervene should guide us correctly. Yet it is to be remembered that in pancreatitis the location of the pain is variable, and radiation to the back and shoulders has been mentioned as at times occurring.

The diagnosis would be more difficult when we have a perforative peritonitis as the result of some lesion in the biliary passages. This condition would more closely resemble other varieties of perforative peritonitis of the upper abdomen, and will be grouped with these in the discussion thereof.

Other conditions referred to as possibly simulating acute pancreatitis have been poisoning, impacted ureteral calculus, etc. In these the course of events, the other and distinctive signs, and in the former the history, should soon serve to put us on the right track

Thrombosis of the mesenteric vessels, practically never diagnosticated except at operation, may give us symptoms of ileus and peritonism, very likely to simulate acute pancreatitis.

Acute appendicitis has been the diagnosis of several cases of pancreatitis. It does not seem to me that these conditions are sufficiently similar to cause the mistake to be made often. Appendicitis is distinctly an inflammatory condition of the lower abdomen; its symptomatology is well marked and typical in most cases, and its signs with few exceptions are not those of an upper abdominal lesion.

Next to a diagnosis of acute obstruction in those cases of pancreatitis not recognized, there is that of an acute perforative peritonitis of the upper abdomen, be it of stomach, duodenum, or bile ducts. In the first two conditions we can generally get a history which would be something of a guide to us. When the biliary system is the seat of perforation, the history may be misleading rather than a guide to us. In all these conditions the sudden agonizing pain in the epigastrium would at once point to a serious lesion of the upper abdomen.

Treatment. The treatment of acute pancreatitis is one of the most important subjects which confronts the surgeon. The comparative rarity of the condition, the different phases it may assume, and the varying degrees of its severity, make the treatment of first importance.

But a few years ago pancreatitis, except when progressing to

suppuration, or the formation of a palpable mass, was considered to belong to the field of medical treatment alone. Surgical intervention was considered hazardous and almost useless. Several cases have been reported as recovering without operation, in which there were apparently the symptoms of an acute pancreatic lesion. I have seen this occur in acute pancreatitis and in acute exacerbation of chronic pancreatitis. Needless to say, recovery under these circumstances, is rare; indeed, it may truthfully be stated that the chance of such a non-operative recovery is comparatively slight.

The medical treatment of acute pancreatitis has been aptly compared to a similar procedure in acute appendicitis. The latter affection was beyond the range of surgery many years after its pathology was well known. It may be admitted then that the treatment of an acute pancreatitis is by surgery only. This being so, there are three important features to be considered as regards operation:

1. At what stage of the illness shall we operate?
2. What method of approach to the seat of disease shall be adopted?
3. The technique after reaching the pancreas or the pancreatic abscess.

1. *The stage at which operation should be undertaken* in acute pancreatitis varies with the variety of the disease. In a fulminating ultra-acute case, the rapid progress of the patient from bad to worse may make early operation necessary. Operation should not be undertaken in the state of primary shock, following the onset of the distinctive symptoms of pancreatitis. Cases in which this has been done have almost invariably come to a fatal termination. The tendency among surgeons who have investigated the question, and have had the opportunity of seeing a number of cases, seems to be to operate comparatively early.

Nevertheless, I am convinced that this rule does not always hold good. Thus, in Case VI, which terminated as a gangrenous pancreatitis, with sloughing of almost the whole organ, it is almost certain that surgical intervention in the first few days would have ended in the patient's death. Our delay, with stimulation, allowed us to operate upon a patient in somewhat improved condition, and to make use of localizing signs to approach the pancreas by a much more favorable route than it would have been possible to adopt had immediate operation been undertaken. It must be understood, then, that while in cases becoming rapidly and progressively worse operation may be imperative, it is generally of advantage to give the organism an opportunity to prepare itself, so to speak, for a still greater tax upon it. Moreover, when we delay until an abscess extending into the loin has formed, we are able to approach it through the loin. Had operation been deferred in Case III, the result could not have been more unfortunate. The formation of a mass in the upper abdomen, or the occurrence of dulness and tenderness toward the

flank, would not only make our diagnosis more certain, but give us a safer route by which to approach the diseased area than through the peritoneum.

2. *The Method of Approach to the Seat of Disease.* The routes to the pancreas may be grouped under two main heads: (1) Those which involve the traversing of the general peritoneal cavity, and (2) the extraperitoneal route by the loin incision.

1. When there is a beginning acute pancreatitis, when the localizing symptoms are all epigastric, when there is a tumor palpable anteriorly, giving a tympanitic note on percussion and a sense of resiliency on palpation, and when there is doubt concerning the diagnosis, we are compelled to make our incision upon the anterior aspect of the abdomen, and to traverse the general peritoneal cavity in order to finally reach the pancreas. Once in the general peritoneal cavity, we may approach the pancreas itself either (*a*) through the lesser omentum, (*b*) through the gastrocolic omentum, or (*c*) through the transverse mesocolon. As a general rule, the anterior or intraperitoneal incision, if I may so call it, has been adopted by almost all operators. It has one serious disadvantage, that is, the risk of infecting the general peritoneal cavity. Its advantages are the free exposure of the operative field, opportunity for radical surgery, and the opportunities which it offers for the establishment of adequate drainage. The choice of a path above or below the stomach must depend upon circumstances. When we wish to secure an extra path for drainage, tampons and drains must be carried both above and below the stomach.

2. The extraperitoneal route, practically, as for exposure of the kidney, allows us to approach the pancreas, and especially the tail thereof, without entering the general peritoneal cavity. The chief advantages of the extraperitoneal route when feasible cannot be questioned, except possibly by a few enthusiasts who, by preference, open appendiceal abscesses and remove ureteral stones through the peritoneal cavity. Naturally it is only applicable in those instances in which we have a distinct indication for it, that is, the appearance of symptoms pointing to the localization of inflammatory exudate or pus in the loin. By it we get good drainage for the pancreas and the omental bursa. Its disadvantages are, however, many. We get no free exposure of the parts and radical surgery is impossible. We can drain, that is all. Gallstones or concomitant gastric lesions, etc., are entirely inaccessible by this method. Cases III and IV of my series were operated upon by the anterior incision, and correctly so, as in each case the localizing symptoms demanded the opening of the greater peritoneal cavity. In Case V, after the operation for gallstones had been carried out, the postpancreatic collection was evacuated by the loin. Thus, there was avoided the danger of peritonitis, and at the same time, the underlying biliary condition was adequately dealt with.

In this connection I may mention the fact that in some of the

reported instances of acute gangrenous or suppurative pancreatitis, drainage has been instituted by both the anterior and posterior routes. In Case VI we had an omental bursitis with distinct localizing signs in the left loin. Here it was deemed better to undertake the unmistakably less serious extraperitoneal operation, rather than to ignore the symptom which clearly indicated our correct avenue of approach to the seat of the lesion. I do not believe that we should have achieved an equally favorable outcome by any other operative procedure. The wound drained freely, the pancreas, as it sloughed, was discharged without difficulty, and our only unsatisfactory feature was the impossibility of obtaining a full exposure of the wound when dressing it.

3. *The Technique after Reaching the Pancreas.* Whether we have to deal with a pancreatitis still in the presuppurative or hemorrhagic stage, or whether pus has already formed, we have but one cardinal indication for treatment—the establishment of adequate drainage.

Should we find an acute hemorrhagic pancreatitis, or an acute gangrenous pancreatitis, having approached the organ by the intraperitoneal route, our only possible procedure locally is to apply tampons and drains freely to the organ itself, going either above or below the stomach. I believe that tube drainage is always essential. Gauze alone will do more to prevent than to establish drainage.

In a previous article I have stated it as my belief that, when we find an engorged and inflamed pancreas, it is expedient to incise the capsule and to apply our drains to this exposed surface. I have since then had no further experience with a case requiring this treatment. Certain recent investigators, however, have come to the conclusion that this incision into the organ itself is unnecessary. Noetzel, especially, who has written the most recent resume on the subject takes this stand. He brings strong support for his argument from his own experience and that of others. In the present state of our knowledge it must be admitted that the question is still open.

It is practically never possible satisfactorily to resect the pancreas for inflammatory lesions, and this is, indeed, unnecessary. Drainage gives free exit to the debris.

Should there be any free fluid in the peritoneal cavity it will be necessary to see to its removal. It should not be forgotten that in this pancreatic exudate we have toxic material often sufficient to cause death. In this connection it is interesting to note that in one of the first cases of this kind reported, that of Halsted, the evacuation of the free fluid in the peritoneal cavity was the only thing attempted. The patient recovered, and while it may be true, as Körte has reremarked concerning this case, that the patient recovered in spite of and not because of the operation, yet it is doubtful if recovery would have taken place with any added toxins for him to overcome.

The peritoneal fluid may gravitate toward the pelvis. In several cases in which this was marked, tube drainage of the pelvic cavity,

with the semi-erect position of the patient, has been successfully adopted.

It is almost unnecessary to mention the fact that gauze drainage in all these cases under discussion must be supplemented by the use of tube drains, preferably rubber ones of large caliber. Drainage by gauze tampons alone often defeats its own object, the saturated gauze acting only as a plug and not as an outlet.

When there is a pancreatic abscess, or an omental bursitis and peripancreatitis, free drainage either by the anterior or the posterior route is the prime essential. When pus is present it is of still more importance than under any other circumstances to use free drainage by tubes, and not to rely on tampons alone.

Some question has arisen as to the treatment of an accompanying cholelithiasis found at the time of an operation for acute pancreatitis. I believe that when the patient's condition warrants it, we should correct also the underlying and really primary biliary condition, and so avoid any probability of again having to perform a serious laparotomy. Often, as Noetzel has pointed out, the gravity of the pancreatic condition and the weakness of the patient will prevent us from thus completing our operation. And in several reported cases, such as that of Stieda, the leaving of biliary calculi has apparently been no hindrance to the complete recovery of the patient from the pancreatic condition. If there is any doubt in the operator's mind as to the advisability of dealing with the accompanying cholelithiasis, he had better defer. Two operations and recovery are preferable to one operative procedure with a fatality.

The preparatory treatment for operation must evidently be very brief in most cases of acute pancreatitis. It does not offer us great possibilities. We can do little to help the patient to stand the shock of surgical intervention beyond the use of the ordinary stimulants, etc. Speed and the avoidance of shock and chilling on the table play a far more important role in helping the patient to ultimate recovery.

Postoperative treatment is the same as in all other serious laparotomy cases, with but few added features. Drainage should be left in until it has very evidently fulfilled its functions—until there is nothing more to drain.

Recently, Wohlgemuth[14] has, by experiment and also clinically, shown that by placing patients who have been operated upon for pancreatitis upon a typical antidiabetic diet we can so alter the pancreatic secretion that it becomes less active and irritating. This fact seems to be abundantly confirmed by other observers. Not only this, but by the same use of diet, the healing of both recent and old pancreatic fistulæ is accelerated. The results in this direction have been truly remarkable. This is perhaps the only postoperative treatment that differs in any way from that of the other suppurating intra-abdominal lesions and abdominal sinuses.

[14] Berl. klin. Woch., 1907 and 1908.

THE PATHOLOGICAL RELATIONSHIPS OF GASTRIC ULCER AND GASTRIC CARCINOMA.

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The following report is based on the study of specimens from gastric and duodenal resections and excisions for ulcer and carcinoma, by Drs. W. J. and C. H. Mayo, from January 1, 1905, to April 1, 1909. In five of the cases the material was obtained at autopsy from patients on whom gastro-enterostomies had been done for ulcer or carcinoma, and from whom no material had been removed at the operation. These autopsies, however, were all made within one hour after the death of the patient; consequently all the material was quite fresh when placed in fixatives. The routine examination consisted of the study of fresh material, sectioned and stained by the method of one of the writers[1]. Blocks of tissue were then fixed in Zenker's fluid and Flemming's chromo-osmo-acetic fluid and in 10 per cent. formaldehyde. In some instances additional blocks were fixed also in absolute alcohol. The gross specimen was then prepared by Melnikow's modification of Kaiserling's method. The gross specimens were photographed by the method of Wilson and Andrews,[2] either fresh or after fixation. The photomicrographs of sections herewith shown were made from hematoxylin-stained specimens. In these no attempt has been made to show fine detail, which has been sacrificed to a study of the distribution of the cells.

The total amount of material studied comprised specimens from 218 cases. Eight of these were from the duodenum, and were all simple ulcers. The remaining 210 were from the stomach. Of these, 47 were ulcers without suspicion of carcinoma; 2 were sarcomas, 2 adenomas, and 1 a diverticulum. Of the remaining 158 cases from the stomach, 5 were ulcers with enough microscopic appearance of aberrant epithelial proliferation to place them in the doubtful class as possible transition cases. Of the remaining 153 cases, which were undoubted carcinoma, 109 (71 per cent.) presented sufficient gross and microscopic evidence of previous ulcer to warrant placing them in a group labelled "carcinoma developing on preceding ulcer." Eleven other cases (7 per cent.) showed considerable evi-

[1] Wilson, L. B., A Method for the Rapid Preparation of Fresh Tissues for the Microscope, Jour. Amer. Med. Assoc., 1905, xlv, 1737.

[2] Stereophotography of Pathological Specimens: Some Improvements in Technique and New Apparatus, Jour. Med. Research, 1908, xvii, 487 to 494.

dence of precedent ulcer, but not sufficient to warrant placing them in the previous group. In 33 cases (22 per cent.) there was relatively small or no pathological evidence of precedent ulcer.

It is unnecessary to review the enormous literature of this much discussed subject. For years the pendulum of opinion swung back and forth, and it has been only within the last decade that sufficient material from early cases has been collected to give a clear understanding of the facts. The reports on specimens removed at operation during that period have practically settled the question as to the very frequent occurrence of gastric carcinoma on the site of previous ulcer. Our excuse for offering these cases at present is to place them on record as one more bit of evidence to clear up a misconception which has done much harm in the past, and which still exists, as is shown by the attitude of the author of the most exhaustive recent work on cancer.[3]

Protocols. Case No. 22,826 (Fig. 1). This specimen is from a woman, aged twenty-six years, who for nine years had had some stomach distress, some gas, occasional vomiting, and eructations. For the last eight weeks she had had some loss of strength, had lost in weight (20 pounds), and had vomited occasionally. Stomach analysis showed a total acidity of 65, free hydrochloric acid 50, lactic acid absent, and blood absent. Operation revealed a chronic ulcer of the lesser curvature. Fig. 2 shows the scar tissue and the eroding base of the ulcer within which are no epithelial inclusions. The character of the lesion deep down below the overhanging border, just where the mucosa comes in contact with the basement membrane, is shown in Fig. 3. Here are numerous groups of epithelial cells cut off by the products of inflammation.

Case No. 22,020. The specimen in this case is through the pylorus of a man, aged fifty-one years, who for three years had had some stomach distress with gas, vomiting, etc. For three months he had had some loss of strength and loss of weight (15 pounds), with severe pain. The stomach analysis showed a total acidity of 56, free hydrocloric acid 30, lactic acid absent, blood absent. This case much resembled the preceding one. Fig. 5 shows the eroding mucosa with swollen epithelial cells in the overhanging border of the ulcer. Fig. 6 from near the base of the mucosa shows several small groups of epithelial cells which are segregated from the rest of the mucosa, as in Case 1.

These two cases show how in chronic gastric ulcers in which no carcinoma is demonstrable there already exist isolated areas of epithelium which is under conditions favorable to its aberrant proliferation.

Case No. 18,088 (Fig. 7). This specimen is from the pyloric half of the stomach from a man, aged forty-five years, who had only

[3] Williams' Natural History of Cancer, Wm. Wood & Co., 1908, pp. 279 to 280.

mild symptoms of so-called dyspepsia until nine months ago, when he began to have marked gastric distress, vomiting blood, gas, eructations, loss of appetite, loss of strength, and loss in weight (60 pounds). The stomach analysis showed a total acidity of 50, free hydrochloric acid 12, lactic acid absent, blood present. Operation showed multiple ulcers of the lesser curvature. Three of these were carcinomatous. Fig. 8 is a section from the overhanging border of the ulcer showing the least amount of carcinoma. Fig. 9 is a section from deeper down in the tissues showing the isolated groups of epithelial cells proliferating and infiltrating.

Case No. 18,867 (Fig. 10). This specimen is from a woman, aged sixty years, who for twenty-five years had had more or less severe stomach symptoms, distress, vomiting, gas, etc. For the last six months she had had considerable loss of strength, loss of weight, and severe persistent pain. Analysis of the stomach contents showed a total acidity of 60, free hydrocloric acid 45, lactic acid absent, blood absent. Operation revealed a carcinoma on an ulcer of the lesser curvature. Fig. 11 is from the ulcerating portion of the stomach lesion. Fig. 12 is of a section showing the proliferation of the epithelium without infiltration. Fig. 13 is from a section showing the true carcinomatous character of the lesion.

Case No. 16,525 (Fig. 14). This specimen is from a male, aged forty-six years, who for seven years had had considerable stomach distress with vomiting and eructations of gas. For the last seven months he had had loss of strength and appetite and had lost 45 pounds in weight, being now quite emaciated. The stomach analysis showed a total acidity of 42, with a free hydrocloric acid content of 37, lactic acid present, and blood present. On operation there was found a carcinoma on the border of an ulcer covering a greater portion of the lesser curvature. Fig. 15 is of a section from the ulcerating border showing cross sections of distended glands with round cells between. Fig. 16 shows the bases of glands clipped off by scar tissue. Fig. 17 shows active proliferation in segregated epithelium. Though strongly suggestive of carcinoma, one would hesitate to diagnosticate this section, since the field is obscured by the round cell infiltration. Fig. 18, however, shows typical scirrhous cancer, that is, the inflammation has here subsided and the fibrous tissue has increased around the islands of proliferating epithelium. These four sections are all from the border of the ulcer, but in successive microscopic steps away from its centre.

Case No. 15,681 (Fig. 19). This specimen is from a man, aged thirty years, who has suffered from gastric distress, nausea, vomiting, and gaseous eructations for five years. During the last seven months he had had marked loss of strength, with a loss of thirty pounds in weight. Stomach analysis showed a total acidity of 62, free hydrochloric acid 32, lactic acid absent, and blood present. Operation revealed a carcinoma on an extensive ulcer of the lesser curvature,

Fig. 1

Fig. 2

Fig. 1 (Case No. 22,826).—*Stereogram of a chronic ulcer of the lesser curvature of the stomach. Note the overhanging mucosa, the perforation of the muscularis, and the large mass of adhesions.

Fig. 2 (Case No. 22,826).—Photomicrograph of the base of the ulcer. (× 100)

* These pictures were originally shown as stereograms but are here reproduced as single pictures only.

Fig. 3

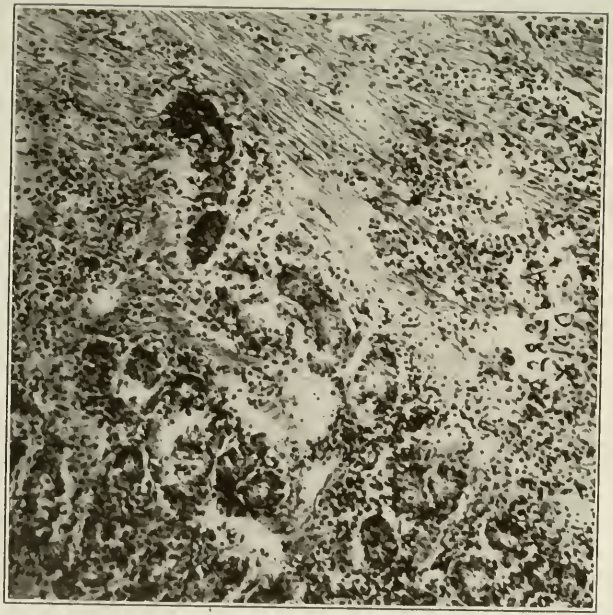

Fig. 4

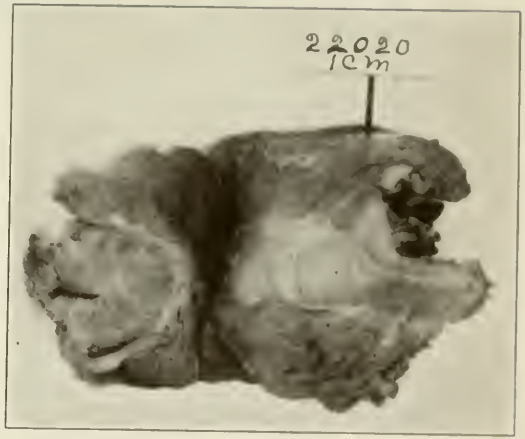

Fig. 3 (Case No. 22,826).—Photomicrograph showing groups of normal epithelium (bases of glands) cut off by products of inflammation deep under the overhanging border of the ulcer. (× 100)

Fig. 4 (Case No. 22,020).—Stereogram of a section through the pylorus, showing the eroded mucosa of a chronic ulcer.

Fig. 5

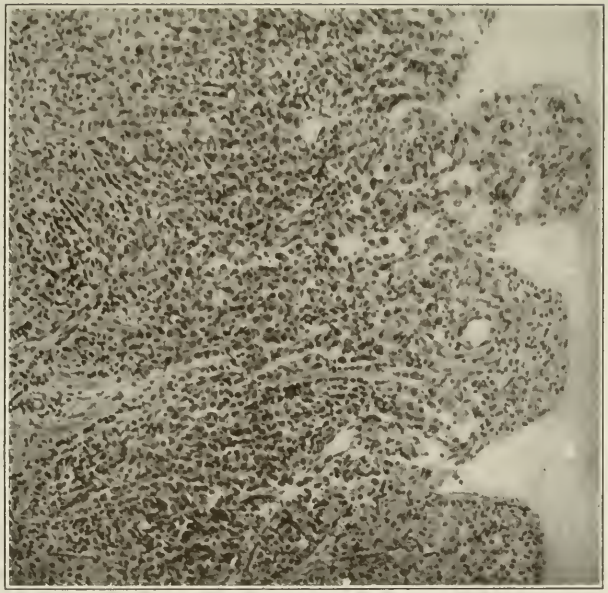

Fig. 6

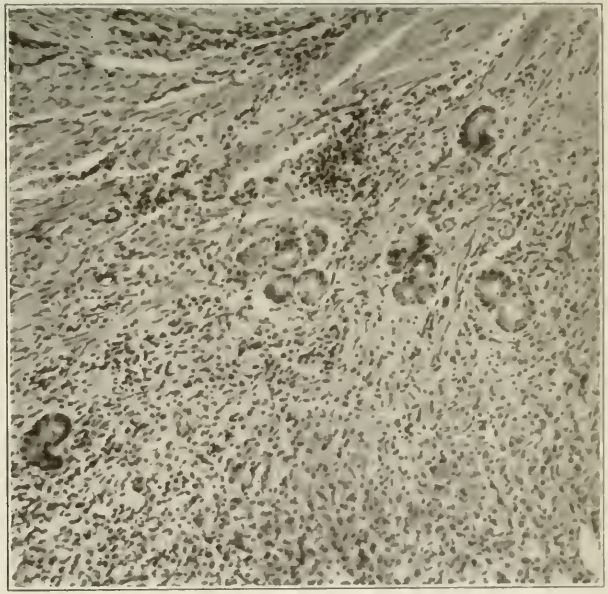

Fig. 5 (Case No. 22,020).—Photomicrograph of the eroding border of the ulcer. (× 100)
Fig. 6 (Case No. 22,020).—Photomicrograph of the base of the ulcerating mucosa, showing isolated groups of epithelium. (× 100)

Fig. 7

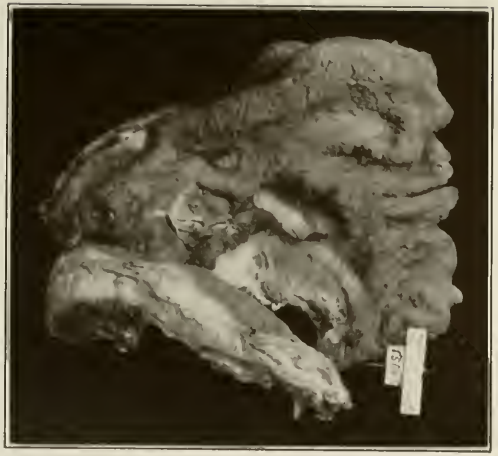

Fig. 8

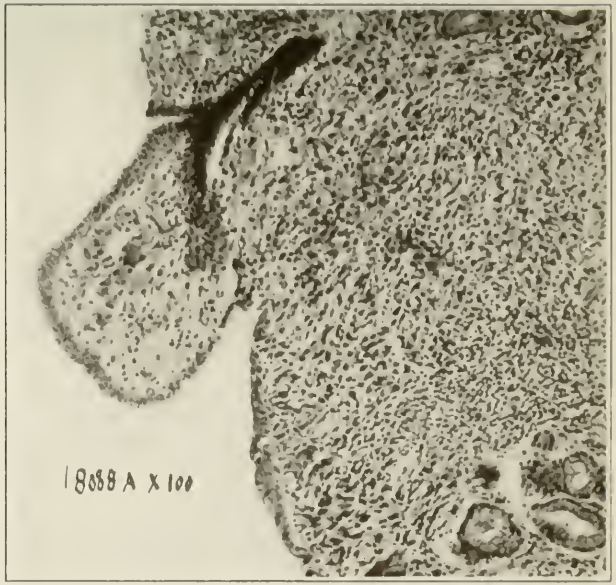

Fig. 7 (Case No. 18,088).—Stereogram of the pyloric one-third of the stomach, looking at the lesser curvature; multiple ulcers; a mass of cancerous tissue was removed from the area near the number needle.

Fig. 8 (Case No. 18,088). Photomicrograph from the overhanging border of the ulcer. (× 100)

Fig. 9

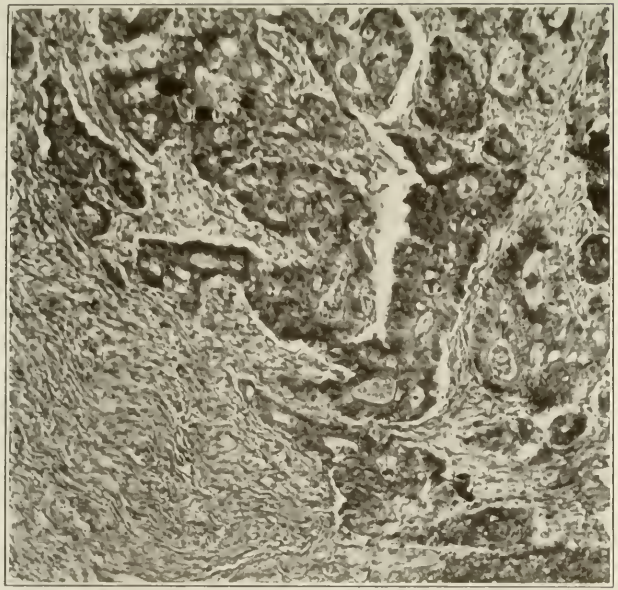

Fig. 10

Fig. 9 (Case No. 18,088).—Photomicrograph from an area deeper than that shown in Fig. 8. The epithelium shows aberrant proliferation and infiltration. (× 100)
Fig. 10 (Case No. 18,867).—Stereogram of the lesser curvature, showing proliferation of the muscularis by an ulcer with carcinoma in the border.

Fig. 11

Fig. 12

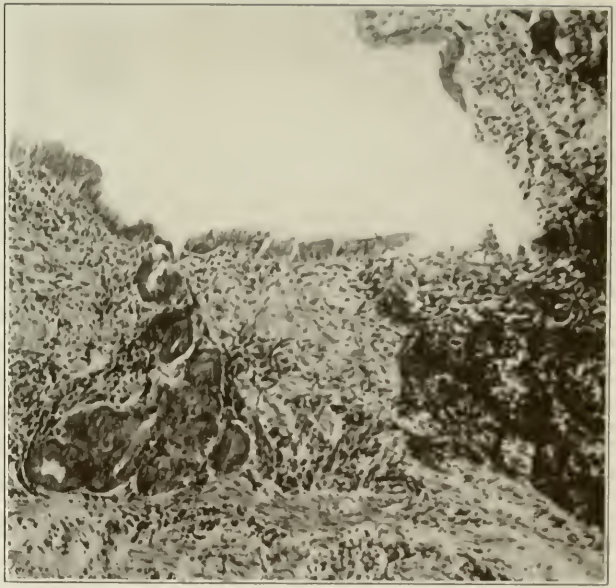

Fig. 11 (Case No. 18,867).—Photomicrograph of the base of the ulcer. (× 100)

Fig. 12 (Case No. 18,867). Photomicrograph showing groups of epithelial cells partially cut off from the surface, actively proliferating but not infiltrating the surrounding tissues. (× 100)

Fig. 13

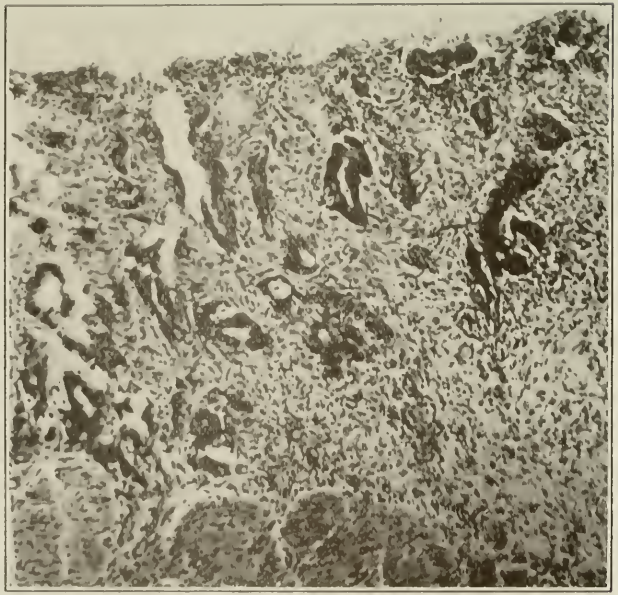

Fig. 14

Fig. 13 (Case No. 18,867). Photomicrograph showing typical carcinoma. (× 100)
Fig. 14 (Case No. 16,525). Stereogram of the pyloric two-thirds of the stomach; carcinoma on a large ulcer beginning in the lesser curvature.

Fig. 15

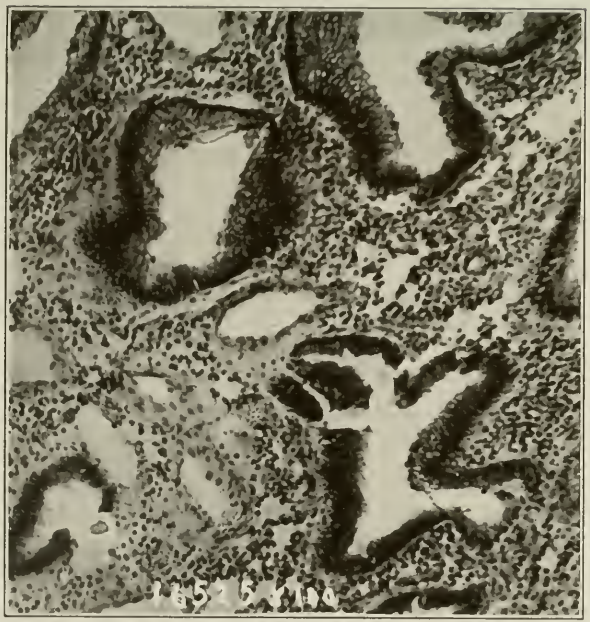

Fig. 16

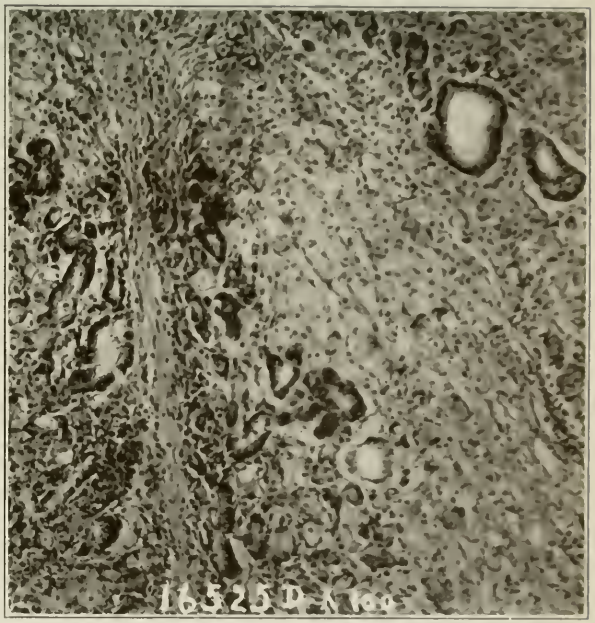

Fig. 15 (Case No. 16,525).—Photomicrograph showing a cross-section of the swollen glands with round-cell infiltration between the glands. (× 100)
Fig. 16 (Case No. 16,525).—Photomicrograph showing the bases of the glands clipped off by scar tissue. (× 100)

Fig. 17

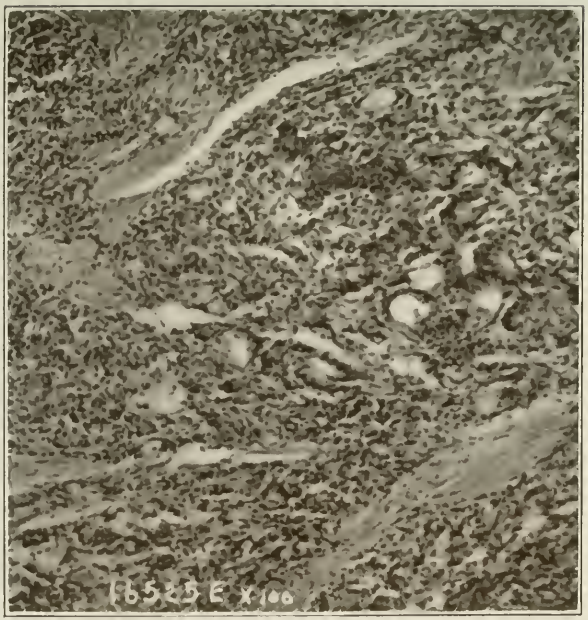

Fig. 18

Fig. 17 (Case No. 16,525).—Photomicrograph showing active proliferation and much round-cell infiltration. (× 100)

Fig. 18 (Case No. 16,525).—Photomicrograph showing typical scirrhous cancer. Figs. 15 18 are from the border of the ulcer, but in successive microscopic steps away from its centre. (× 100)

Fig. 19

Fig. 20

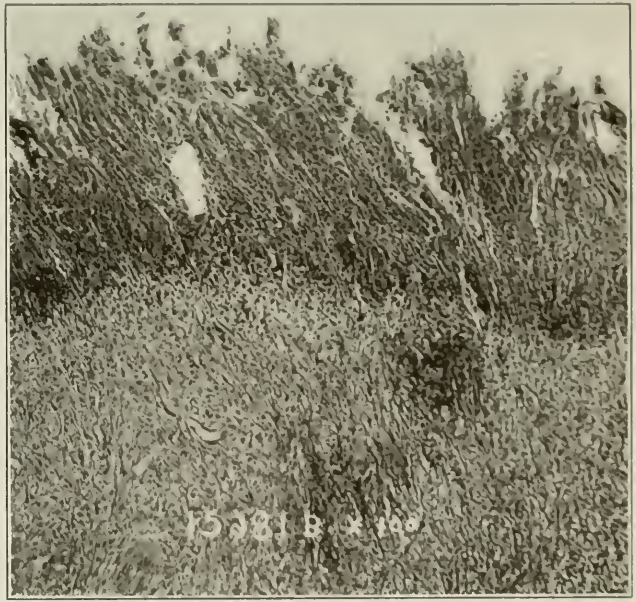

Fig. 19 (Case No. 15,681).—Stereogram of a portion of the pyloric one-half of the stomach, showing carcinoma of the lesser curvature involving also the pylorus.
Fig. 20 (Case No. 15,681).—Photomicrograph from the base of the ulcer. (× 100)

Fig. 21

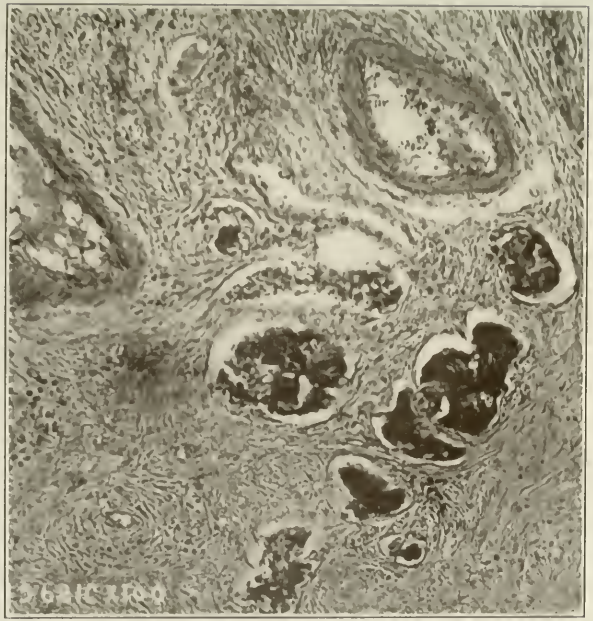

Fig. 22

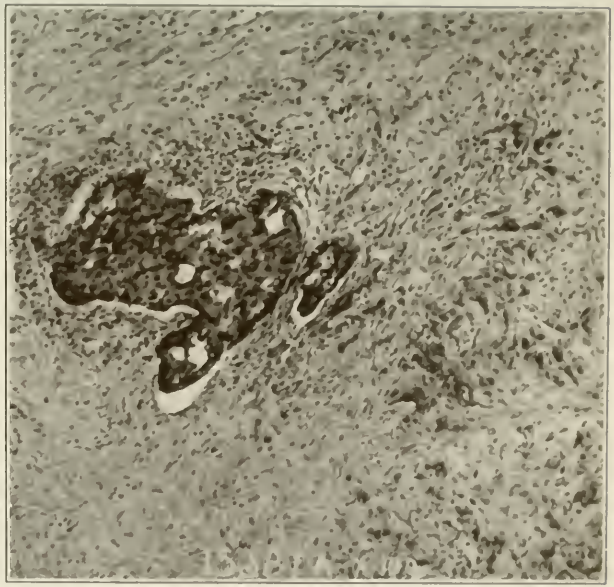

Fig. 21 (Case No. 15,681).—Photomicrograph showing proliferating epithelial masses at the base of the swollen mucosa; edge of the ulcer. (× 100)
Fig. 22 (Case No. 15,681).—Photomicrograph showing an area a little farther removed from the ulcerating area than the preceding section (Fig. 21). (× 100)

Fig. 23

Fig. 24

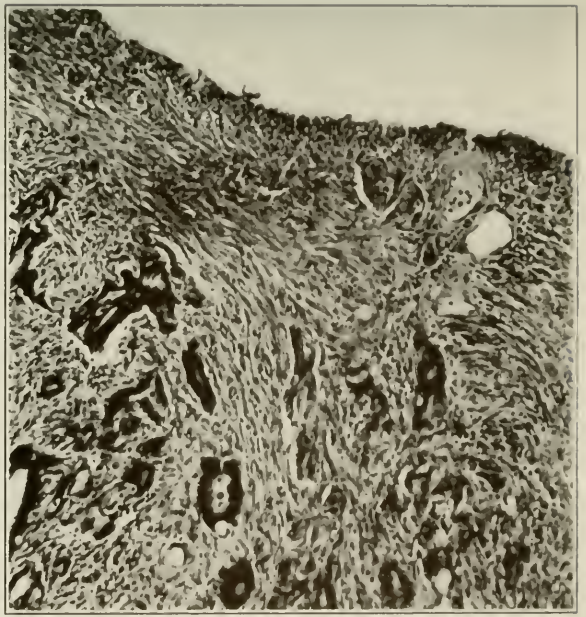

Fig. 23 (Case No. 16,651).—Stereogram of a portion of the pyloric one-third of the stomach showing carcinoma involving the lower lesser curvature and pylorus.

Fig. 24 (Case No. 16,651).—Photomicrograph of a section from the ulcer border, showing at the right side the ulcer base and at the left the developing carcinoma. (× 100)

Fig. 25

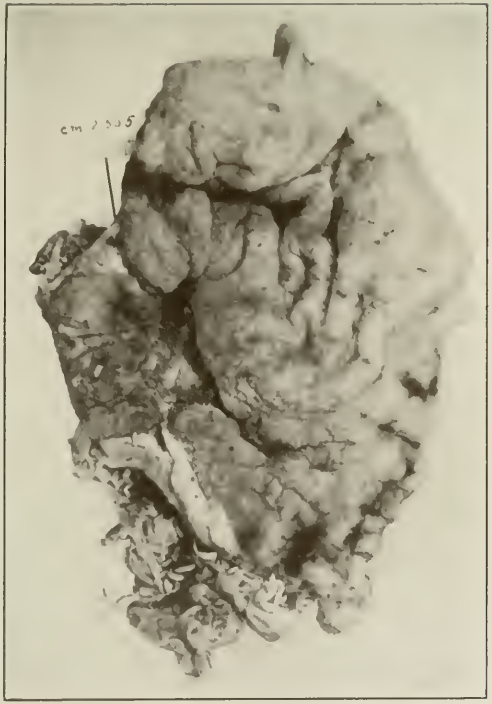

Fig. 26

Fig. 25 (Case No. 21,555).—Stereogram showing carcinoma on a large ulcer of the lesser curvature of the stomach.
Fig. 26 (Case No. 21,555).—Stereogram of a gross section through a carcinoma and ulcer.

Fig. 27

Fig. 28

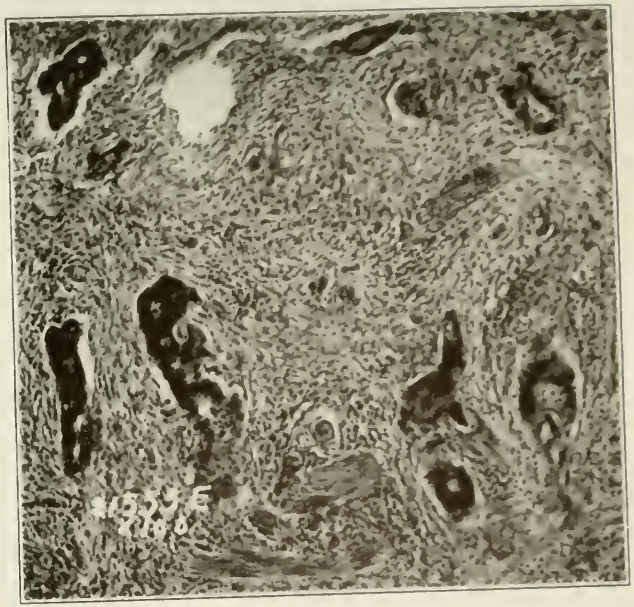

Fig. 27 (Case No. 21,555). Photomicrograph from the edge of an ulcer; base of the ulcer at the right; separated bases of tubular glands showing early carcinomatous changes at the left. (× 100)

Fig. 28 (Case No. 21,555). Photomicrograph of a section from under the base of the overhanging edge, showing scirrhous cancer. (× 100)

Fig. 29

Fig. 30

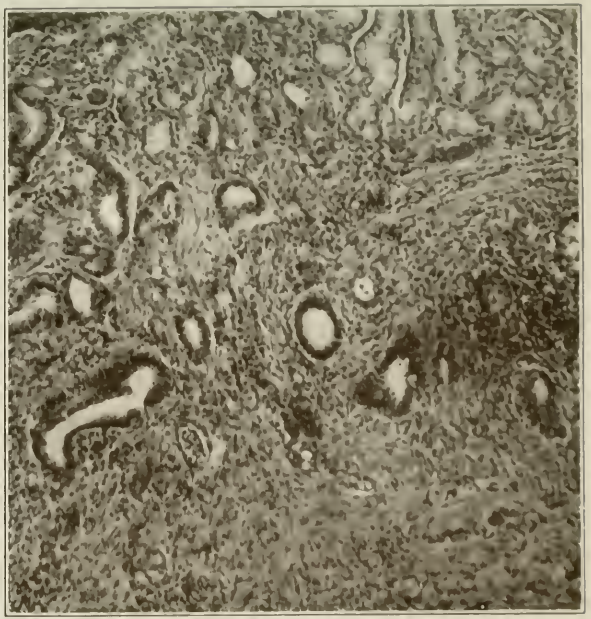

Fig. 29 (Case No. 19,322).—Stereogram showing a "ring cancer" of the pylorus (originated in an ulcer of the lesser curvature).
Fig. 30 (Case No. 19,322).—Photomicrograph showing islands of proliferating epithelium at the base of the mucosa of the overhanging border of an ulcer. (×100)

Fig. 31

Fig. 32

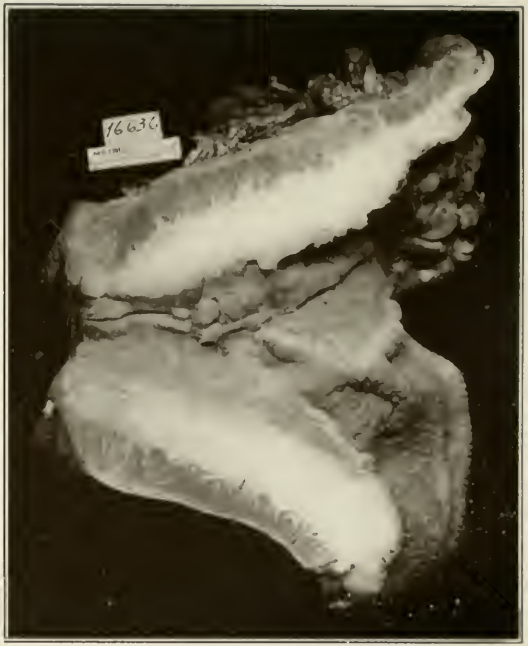

Fig. 31 (Case No. 19,322).—Photomicrograph showing the base of the ulcer at the right, and carcinoma in the border at the left. (× 100)
Fig. 32 (Case No. 16,636).—Stereogram showing a large ulcer of the lesser curvature with carcinoma in the border.

Fig. 33

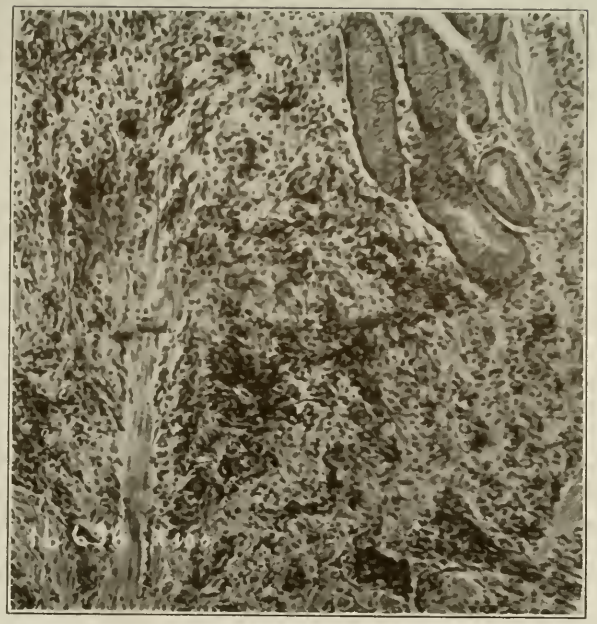

Fig. 34

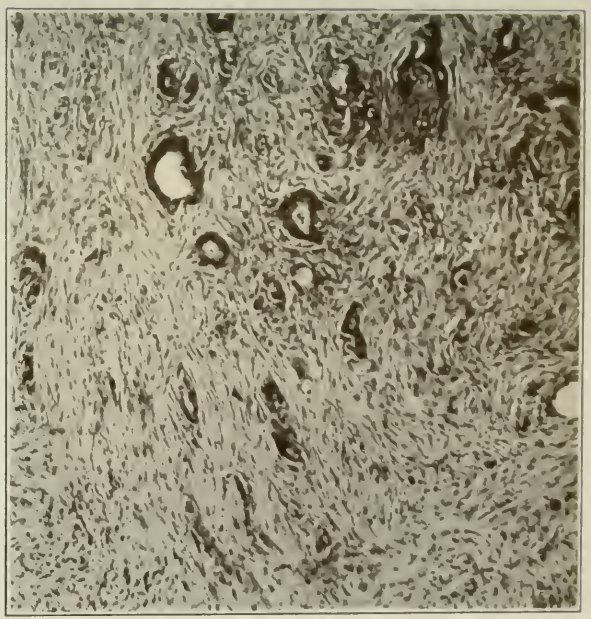

Fig. 33 (Case No. 16,636).—Photomicrograph showing a group of partially segregated tubular glands, and, at the lower right hand corner, one nest of aberrant proliferating epithelium. (× 100)

Fig. 34 (Case No. 16,636).—Photomicrograph showing the development of carcinoma in the scar tissue in the edge of an ulcer. (× 100)

Fig. 35

Fig. 36

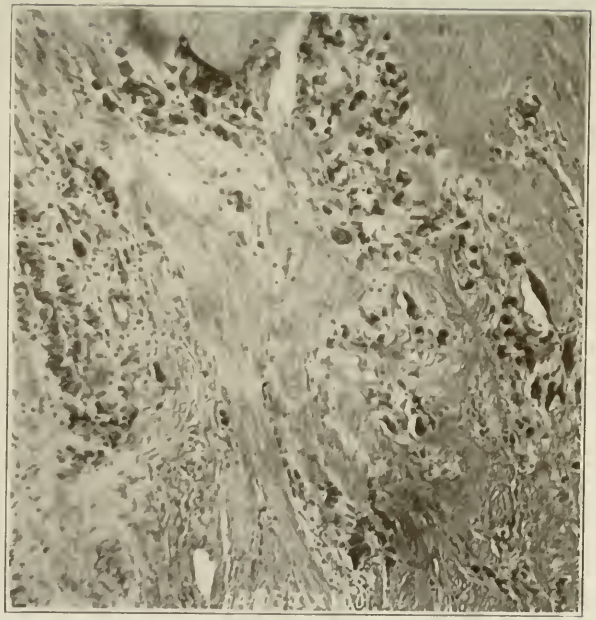

Fig. 37

Fig. 35 (Case No. 14,857).—Stereogram showing a carcinoma filling the site of an old perforating ulcer of the stomach.
Fig. 36 (Case No. 14,857).—Photomicrograph showing a carcinoma with scar tissue. (× 100)
Fig. 37 (Case No. 15,351).—Stereogram showing a carcinoma in a small raised island of tissue in the base but near the edge of a large ulcer of the lesser curvature.

Fig. 38

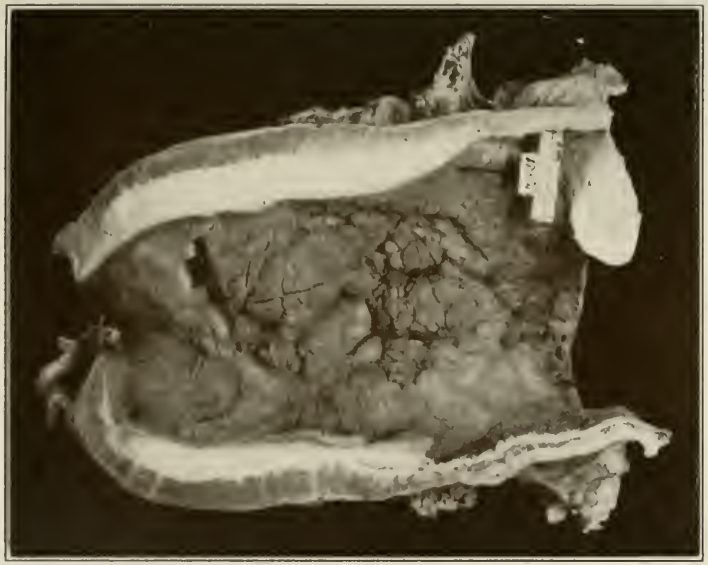

Fig. 39

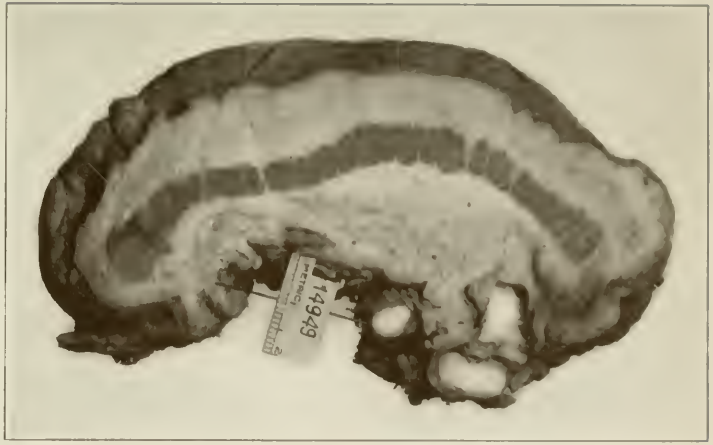

Fig. 38 (Case No. 18,514).—Stereogram showing an extensive carcinoma on a large ulcer of the lesser curvature.

Fig. 39 (Case No. 14,949).—Stereogram of a gross section through the pylorus and the lesser curvature showing an ulcer of the lower 8 cm. of the lesser curvature and carcinoma about 2 cm. above; metastasis in the glands.

Fig. 40

Fig. 41

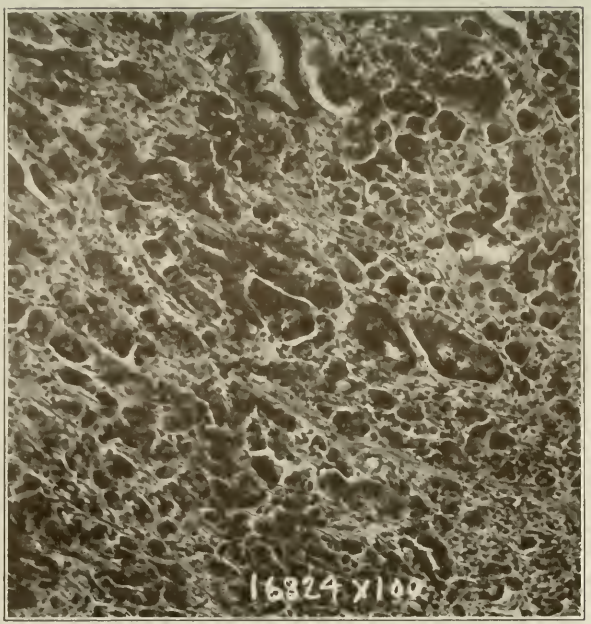

Fig. 42

Fig. 40 (Case No. 16,824).—Stereogram showing a small early carcinoma of the pylorus without pathological evidence of previous ulcer.
Fig. 41 (Case No. 16,824).—Photomierograph showing an early adenocarcinoma. (× 100)
Fig. 42 (Case No. 16,806).—Stereogram of a portion of the stomach wall showing carcinoma with colloid degeneration and deposition of lime salts; no pathological evidence of previous ulcer.

involving also the pylorus. Fig. 20 is from a section from the base of the ulcer. Fig. 21 is of the overhanging border nearest the ulcerating area, showing segregated proliferating epithelial masses. Fig. 22 is of an area a little farther removed from the ulcerating area than the preceding section.

Case No. 16,651 (Fig. 23). This is a specimen from a woman, aged forty-two years, who for sixteen years had had intermittent stomach distress with vomiting, etc. For the last four months she had had considerable loss of strength, weight, and appetite. Stomach analysis showed a total acidity of 50, with free hydrochloric acid absent, lactic acid absent, and blood present. The gross specimen shows a lesion of the lesser curvature. Fig. 24 is of a section from the edge of the ulcer showing at the right side the old scar tissue in the border of the ulcer, and at the left side the carcinoma advancing into the scar.

Case No. 21,555 (Fig. 25). This specimen is from a man, aged sixty-seven years, who for twelve years had had considerable stomach distress, including vomiting, eructations, g etc. During the last few months he had grown considerably w , had lost strength, appetite, and 35 pounds in weight. The stomach analysis showed a total acidity of 30, free hydrochloric acid 15, lactic acid absent, and blood present. The gross specimen showed a large ulcer of the lesser curvature, which is further shown in gross section in Fig. 26, in which the character of the ulcer can be more clearly made out. Fig. 27 shows the carcinoma advancing into the scar tissue at the edge of the ulcer. Fig. 28 is from material at the base of the overhanging mass, and shows scirrhous carcinoma change.

Case No. 19,322 (Fig. 29). This specimen is from a man aged thirty-three years, who for ten years had had symptoms of gastric ulcer, that is, distress, vomiting, gas, eructations, etc. During the last eight months he had had considerable loss of appetite with a loss of 50 pounds in weight. The stomach analysis showed a total acidity of 35, free hydrochloric acid 14, lactic acid absent, and blood absent. The gross specimen closely resembles a simple ulcerated pylorus. In one area, near the greater curvature, however, a nodular mass may be seen which suggests carcinoma. The character of the isolated islands of mucosa at this point is shown in Fig. 30; in Fig. 31 is shown the carcinomatous invasion of the scar tissue in the edge of the ulcer.

Case No. 16,636 (Fig. 32). This specimen is from a man, aged forty-six years, who for ten years had shown marked stomach symptoms, distress, gas, eructations, etc., and for the past eight months had loss of strength, appetite, and 70 pounds in weight. The stomach analysis showed a total acidity of 35, free hydrochloric acid absent, lactic acid present, and blood present. The specimen shows an enormous thickening of the muscularis and submucosa. On this mass of scar tissue is developing a carcinoma. Fig. 33 is of a

section from the lesser curvature area, showing a group of partially segregated tubular glands and near the lower edge of the figure a nest of aberrant proliferating epithelium. Fig. 34 shows the development of the carcinoma in the scar tissue.

Case No. 14,857 (Fig. 35). This specimen is from a man, aged fifty-one years, with a history of chronic stomach trouble for ten years, nausea, vomiting, gas, distress, etc. During the last six months these symptoms have been markedly increased and the patient has suffered a loss of 30 pounds in weight. The gross specimen shows the site of an old perforating ulcer, the muscularis being completely broken through, and the adhesions from the old perforation being quite evident. The carcinoma fills the site of the ulcer. Fig. 36 shows a carcinoma developing within the scar tissue. Judging from the microscopic appearance alone, one might hesitate to decide that this was a case of precedent ulcer, although the bands of scar tissue with masses of epithelium included are quite suggestive. The gross specimen with the history, however, is sufficient to warrant a positive diagnosis.

Case No. 15,351 (Fig. 37). This specimen is from a man, aged forty-one years, who for many years (fifteen or more) had had gastric distress, vomiting, and eructations of gas. In the last six months he had had a loss of strength, and a loss in weight of 30 pounds. The stomach analysis showed a total acidity of 25, free hydrochloric acid absent, lactic acid present, and blood present. The gross specimen shows a large ulcer of the lesser curvature and a small carcinoma developing in a raised island of tissue in the base of the ulcer near one edge. It is the only instance of the kind in our series.[4]

Case No. 18,514 (Fig. 38). This specimen is from a female, aged forty-one years, who had for three years suffered gastric distress, vomiting, gas, eructations, etc. In the last six months she had had marked loss of appetite, loss of strength, and loss in weight (60 pounds). The stomach analysis showed a total acidity of 100, free hydrochloric acid absent, lactic acid present in large amounts, and blood present. The specimen showed a widely diffused cancer on a large ulcer of the lesser curvature.

Case No. 14,949 (Fig. 39). This specimen is from a man, aged thirty-one years, from whom no history of any stomach trouble could be elicited prior to that beginning one year ago, when he began to have nausea, vomiting, loss of strength, and loss of 65 pounds in weight. Stomach analysis showed a total acidity of 10, free hydrochloric acid absent, lactic acid present, and blood present. A gross section through the lesser curvature of the stomach is here shown. The stomach wall at this point was from 2.5 to 4 cm. thick. The lesion was purely of an ulcerous character for the lower 8 cm. of the

[4] The microscopic details in this and most of the succeeding cases are omitted for lack of space. They were closely parallel with those already shown.

lesser curvature; about 2 cm. of the upper portion of the lesser curvature showed a rapidly growing carcinoma, which had formed metastases in the adjacent glands.

This case is presented to show the unreliability of ever so well taken clinical histories, particularly in the young male, who is not accustomed to give much attention to slight stomach trouble. There can be no question that this patient had stomach ulcer for years preceding the onset of cancer.

The preceding nine cases are fair representatives of those gastric resections for carcinoma in which we consider the pathological evidence of preceding ulcer sufficient to warrant such a diagnosis.

Case No. 16,824 (Fig. 40). This specimen is from a man, aged forty-three years, who was apparently well until one year ago, when he began to show stomach distress, with gas, loss of strength, and loss of weight (40 pounds). The stomach analysis showed a total acidity of 25, with free hydrochloric acid 8, lactic acid present, and blood present. The specimen shows a small carcinoma of the pylorus, without gross evidence of previous ulcer. Fig. 41 is from a deep level of a section and shows adenocarcinoma. We would not seem to be warranted in making any diagnosis of preceding ulcer in this case of carcinoma, either from the history or the pathological evidence.

Case No. 16,806 (Fig. 42). This is a specimen from a man, aged sixty-eight years, who during the last thirty years had had three prolonged attacks of stomach trouble, marked distress, gas, vomiting, eructations, etc. During the last eight months he had had loss of strength, loss of appetite, and a loss of 30 pounds in weight, and there was also severe persistent pain. Stomach analysis showed a total acidity of 30, free hydrochloric acid absent, lactic acid present, and blood present. Two-thirds of the stomach was removed. Only a small portion of the specimen is here shown—a section through the wall of the lesser curvature, which was about 3 cm. thick. Colloid degeneration with deposition of lime salts had occurred throughout the wall of the removed portion of the stomach. In this case there is absolutely no pathological evidence of the occurrence of previous ulcer, although the thirty-year history is very strong clinical evidence.

These last two cases represent the group of 33 (22 per cent.) of our cases, in which we could find insufficient pathological evidence to warrant a diagnosis of preceding ulcer. The latter case also is a fair example of the type of case which frequently comes to autopsy and shows no evidence of preceding ulcer.

The preceding cases are fair representatives of our series. The 109 cases (71 per cent.) which present pathological evidence, gross and microscopic, parallel with that shown in detail herewith, that is, large ulcers with scar tissue centres and overhanging borders, deep in the bases of which cancer is present, in almost every instance have unmistakably originated on the lesser curvature of the stomach, the

usual site of gastric ulcer. Further, almost every case gives a clinical history suggesting gastric ulcer for a long period of years preceding the relatively short period when the history became that of gastric cancer.

That carcinomas should develop in the edges of gastric ulcers is only what we should expect; the wonder is that the facts should have been so long in being recognized. This has been due to: (*a*) Failure to recognize clinically the frequency of gastric ulcer; (*b*) failure to recognize that gastric cancers are not initially pyloric tumors, but extensions thereto from the lesser curvature; and (*c*) giving undue weight to observations at autopsies. When the patient has died of gastric cancer, the neoplasm has usually obliterated all gross and microscopic evidence of previous ulcer.

As the pathologist examines stomach specimens from the surgical clinic he constantly observes the various steps in the following sequence:

1. Chronic ulcers from the centres of which the mucosa has disappeared leaving a scar tissue base.
2. In the overhanging broders of the ulcers the mucosa is proliferating.
3. Deep in the borders many groups of epithelial cells have been nipped off by scar tissue and are exhibiting all stages of aberrant proliferation with infiltration of the surrounding tissues.
4. Metastases are forming in the lymphatics of the stomach wall and adnexa.

A small percentage of cases operated upon are too far advanced to show these steps, and a very small percentage—probably not over 2 per cent.—give evidence of rapid aberrant epithelial proliferation and infiltration without any sign of previous ulcer.

Adopting Adami's[5] classification we may therefore correctly desig-designate most gastric carcinomas as "blastomas originating from unipotential cells of postnatal displacement," although it is probable that a very small number are "blastomas originating from unipotential cells that assume neoplastic characters without displacement and rapidly assume malignancy."

[5] Principles of Pathology, 1908 i, 770.

THE ETIOLOGY AND PATHOLOGY OF INGUINAL HERNIA.

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In order to understand the mode of occurrence of inguinal hernia and employ intelligent operative measures for its cure, the surgeon must be familiar with those structural conditions which make it possible for a portion of the intra-abdominal contents to protrude through the inguinal triangle, a space bounded above by a line drawn horizontally inward from the junction of the middle and outer thirds of Poupart's ligament to the outer margin of the rectus muscle, below by the inner two-thirds of Poupart's ligament, and internally by the outer margin of the rectus muscle. The structures to be considered are the inguinal fossæ, Hesselbach's triangle, the internal abdominal ring, the inguinal canal, and the external abdominal ring.

When the anterior abdominal wall is viewed from behind the following structures are seen: Extending from the apex of the bladder to the umbilicus is the urachus, the degenerated intra-embryonic portion of the allantois, also known as the median vesico-umbilical ligament. On either side of this median cord there is another, extending obliquely upward and inward to join it at the umbilicus. These are the remains of the hypogastric arteries, which are patent only during intra-uterine life. They are also called the lateral vesico-umbilical ligaments. Lying external to these lateral cords are the deep epigastric arteries. These five structures are covered with peritoneum, arranged in more or less well-defined folds, that covering each obliterated hypogastric and deep epigastric artery being known as the plica hypogastrica and plica epigastrica respectively.

In relation with these cords three depressions may be distinguished—one between the urachus and the obliterated hypogastric artery, another between the hypogastric and the epigastric arteries, and a third external to the epigastric artery. These depressions are known as the internal, middle, and external inguinal fossæ respectively, and with the exception of the internal one are very important with reference to the occurrence of inguinal hernia (Fig. 1). They can be examined better after the peritoneum has been carefully stripped off. When this has been done it will be found that the internal fossa is bounded in front, with the exception of a small area above its inferior external angle, by the rectus abdominis muscle, which is strong and unyielding, and well adapted

to withstand any unusual intra-abdominal pressure which may be exerted upon it. Therefore, only the small area of this space which is not covered by the rectus muscle can be considered as a weak spot in the abdominal wall. It is quite different, however, with the middle fossa, the one between the obliterated hypogastric and the deep epigastric arteries. This one is bounded in front only by the transversalis fascia and the attenuated lower fibers of

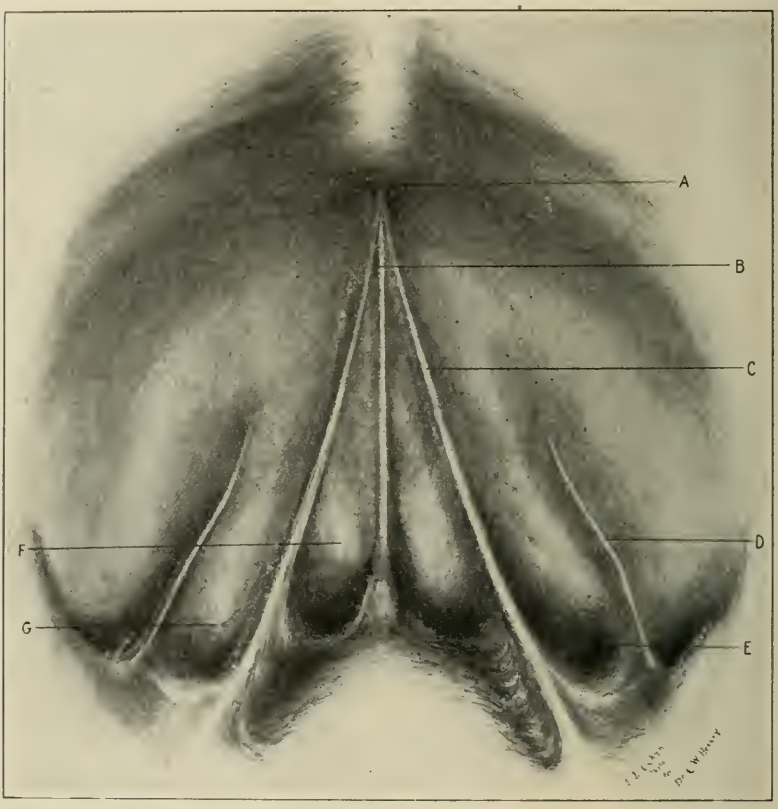

FIG. 1.—The anterior abdominal wall viewed from behind, showing the inguinal fossæ: *A*, umbilicus; *B*, urachus; *C*, obliterated hypogastric artery; *D*, deep epigastric artery; *E*, external inguinal fossa; *F*, internal inguinal fossa; *G*, middle inguinal fossa. (Drawing made from a dissection prepared by Dr. Bonney, Department of General Anatomy, Jefferson Medical College.)

the internal oblique muscle. An examination of several hundred cadavers has shown that there is not a decided blending of the lower fibers of the internal oblique muscle with those of the transversalis to form a well-developed conjoined tendon in more than one-fourth of all cases. That portion of the internal oblique arising from Poupart's ligament has often been found to be aponeurotic instead of muscular, and to fuse almost imperceptibly with the

aponeurosis of the transversalis; whether it stops short or is continued downward with the latter to help form the deep crural arch cannot positively be stated, for the reason that it has not been possible satisfactorily to dissect the one from the other so homogeneously are they united. Even when a decided tendinous union takes place there is always a considerable area below it which is filled in only by fascia. Thus, it is seen that the middle fossa is a decidedly weak area in the anterior abdominal wall, ill adapted to resist the force of any unusual intra-abdominal pressure which may be brought to bear upon it. In fact, it is through this area and the contiguous small uncovered portion of the internal inguinal fossa already described that a certain percentage of inguinal herniæ occur. The space thus formed, which is bounded externally by the deep epigastric artery, internally by the outer margin of the rectus muscle, and inferiorly by Poupart's ligament, is known as Hesselbach's triangle.

The external inguinal fossa, lying external to the deep epigastric artery, constitutes another weak spot in the anterior abdominal wall. This fossa really owes its existence to the internal abdominal ring, a spot in the transversalis fascia through which the testicle pushes its way on its course downward from the abdominal cavity to the scrotum. If the transversalis fascia be examined carefully at this spot after the peritoneum has been stripped away, the following arrangement will be found. Internally it is strengthened by tendinous fibers passing upward from the inner end of Poupart's ligament, and externally and superiorly it presents a somewhat annular arrangement of its fibers, which extend downward along the spermatic cord. To this funnel-shaped process, the term infundibuliform fascia is applied, and the internal stronger margin is known as the interfoveolar ligament of Hesselbach. The latter really forms a line of demarcation between the external and middle fossæ just as the deep epigastric artery does (Fig. 2).

The formation of the internal ring and the inguinal canal is best made clear by tracing the descent of the testicle from the abdomen to the scrotum. The scrotum is formed from the genital swellings, into which a little sac of peritoneum is invaginated comparatively early in fœtal life. The inguinal ligaments, or gubernacula, are also attached to the bottom of this peritoneal pouch. Now as the fœtus develops, this pouch (which in the male is called the vaginal process and in the female the canal of Nuck) becomes more and more elongated, keeping pace with the growth of the genital swellings, until toward the termination of intra-uterine life, probably at about the end of the eighth month, it has formed a sac extending well down into the scrotum. It is to be borne in mind that this sac is placed in front of the testicle before that organ emerges from the abdomen, that it always bears such a relation to the testicle, and that it is not pushed from the anterior abdominal

wall by the testicle in the way in which the other layers enveloping that gland and the spermatic cord are displaced. From these considerations it becomes evident that at a certain stage of fœtal life there is a free communication between the peritoneal cavity and the scrotum. Shortly after birth—probably between the tenth and twentieth days—the vaginal process normally becomes obliterated except as to its lower portion, which surrounds the testicle

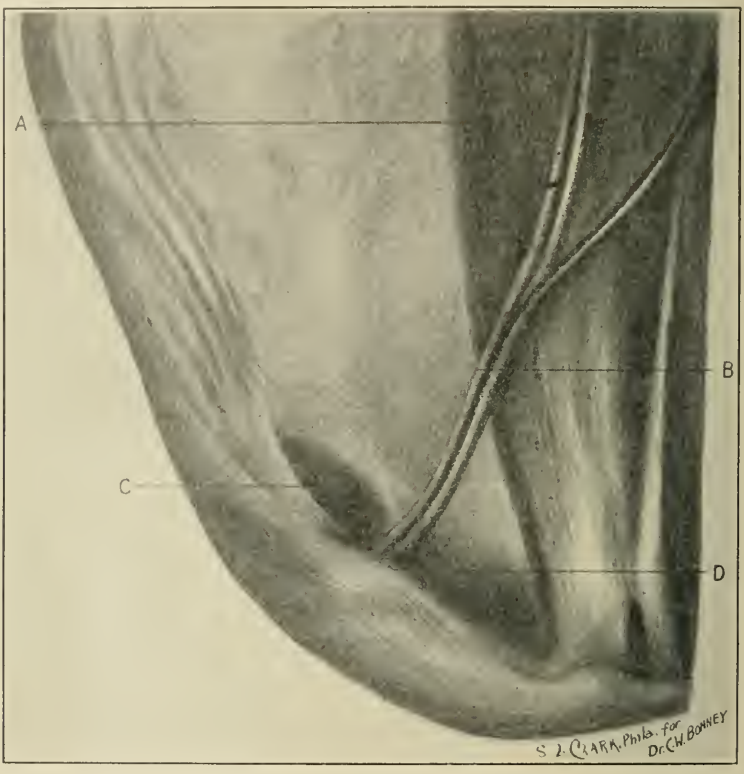

FIG. 2.—The anterior abdominal wall viewed from behind after the peritoneum has been stripped up: *A*, rectus muscle; *B*, deep epigastric vessels; *C*, external inguinal fossa; *D*, middle inguinal fossa. Observe that the internal inguinal fossa is almost entirely covered by the lower portion of the rectus. (Drawing made from a dissection prepared by Dr. Bonney, Department of General Anatomy, Jefferson Medical College.)

and forms its tunica vaginalis. Failure of the upper portion of this peritoneal process to become obliterated constitutes an important factor in the production of congenital hernia. Reference to it will be made again.

By what force or forces the testicle is made to change its position from abdomen to scrotum has been a subject of considerable discussion. In the light of our present knowledge it may be stated that the conditions operative in bringing about this change are

the disparity of growth between the inguinal ligaments and the abdominal wall and the elongation of the vaginal process of peritoneum. Modern embryologists are agreed that the former factor is a very important one. As the abdominal walls develop more rapidly than the inguinal ligaments, there is a tendency for the testicles to be drawn down toward the inguinal region where the ligaments are attached. As this attachment is into the little sac of peritoneum in the genital swellings which develops into the vaginal process, it seems reasonable to suppose that the testicle will be carried still further downward as the process becomes elongated. The theory that the ligament itself draws the testicle downward by contracting is not tenable in view of our present embryological knowledge.

As the testicle passes through the abdominal wall it carries in front of it a layer from each of the muscular or fascial planes which it perforates. Thus, as it comes out of the abdomen at the spot known as the internal ring, it carries a portion of transversalis fascia along, drawing it down like a cone or funnel. Hence the application of the term infundibuliform fascia to this portion of the transversalis fascia. From the next stratum of the belly wall, the internal oblique muscle, it takes up a covering known as the cremaster, and as it passes through the succeeding layer, the aponeurosis of the external oblique muscle, it pushes a fascial investment called the external spermatic fascia in front of it.

The spermatic cord occupies and fills up the channel thus made by the testicle in the substance of the abdominal wall. This channel is known as the inguinal canal. It extends from the internal to the external abdominal ring, and in the adult is about an inch and a half long. It is bounded anteriorly by the aponeurosis of the external oblique muscle and in its outer third by the internal oblique, posteriorly by the transversalia fascia, superiorly by the arched fibers of the internal oblique and transversalis muscles, and inferiorly by Poupart's ligament. A study of hundreds of dissections has convinced us that these structures form the true boundaries of the canal, and that any additional ones which may be given are artificial, inaccurate, and confusing.

It must always be borne in mind that the term "canal" is a misnomer. There was a canal in fœtal life, but under normal conditions there is none in extra-uterine life except for a very short time. In the adult the inguinal canal is nothing but a slight gap in the abdominal wall well filled in by the spermatic cord or round ligament. From the description of the internal abdominal ring it will be readily understood that the term "ring" is also inaccurate (Fig. 3).

The external abdominal ring likewise is not a ring in the true sense of the word. It is a cleft in the aponeurosis of the external oblique muscle, situated just above and external to the spine of the

pubes, having its base below and its apex above. It transmits the spermatic cord in the male and the round ligament in the female. The margins of this cleft are called pillars, of which the outer is the stronger, being formed by the inner end of Poupart's ligament. The inner pillar is attached to the symphysis pubis, its fibers interdigitating with those of the internal pillar on the opposite side. Stretching across the ring from one pillar to the other are fibers likewise derived from the aponeurosis of the external oblique, known as the intercolumnar fascia, and continuous with the external spermatic fascia which envelopes the cord. Thus it is seen that there is no distinct opening in the aponeurosis of the external oblique muscle, the cord and intercolumnar fascia effectually closing in the breach between its fasciculi. It is only after the intercolumnar fascia has been removed and the cord displaced that the gap becomes apparent (Figs. 4 and 5).

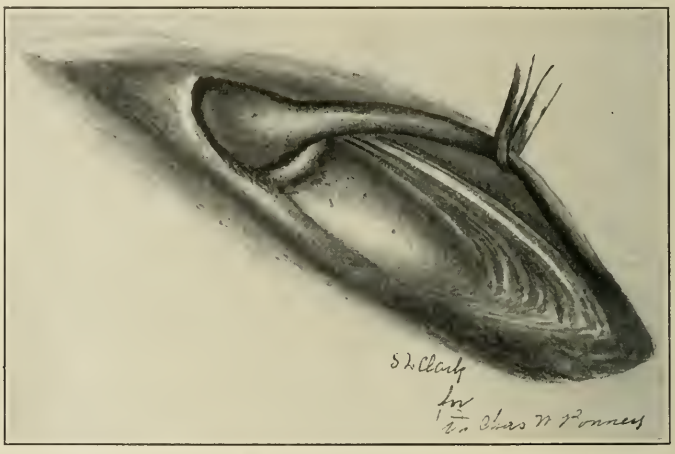

Fig. 3.—The inguinal canal and the internal abdominal ring. The spermatic cord has been lifted out of the canal, and traction is being made upon it so as to render the infundibuliform fascia tense. Observe the deep epigastric artery internal to the ring. (Drawing made from a dissection prepared by Dr. Bonney, Department of General Anatomy, Jefferson Medical College.)

As has previously been stated, a certain number of all inguinal herniæ, certainly not more than four or five out of every hundred, protrude from the abdomen through Hesselbach's triangle. The remainder come out through the internal abdominal ring. They traverse the inguinal canal, break through the external ring, and either present above and external to the pubes or pass downward into the scrotum. A hernia of this kind is known as an indirect, oblique, or external hernia: indirect, in contradistinction to the one which comes out through Hesselbach's triangle; oblique, because of the direction in which it passes downward; external, because it lies to the outer side of the deep epigastric artery. A hernia

which passes through the external ring is called a complete inguinal hernia. The coverings of an indirect hernia are derived from the various layers of the abdominal parietes, just as are the coverings of the spermatic cord. From within outward they are peritoneum, preperitoneal fat, infundibuliform fascia, cremaster muscle or fascia, intercolumnar fascia, superficial fascia, and skin. Two points in reference to these coverings are worthy of mention: (1) The peritoneal covering, or sac, of an acquired hernia is derived from the parietal peritoneum, so that when the hernia extends down into the scrotum, its peritoneal covering has nothing in com-

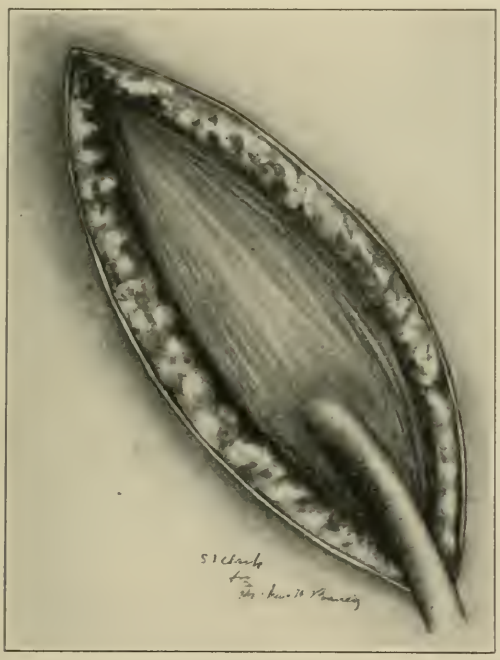

FIG. 4.—Showing the spermatic cord emerging from the external abdominal ring. Observe that there is no distinct opening in the aponeurosis of the external oblique muscle. (Drawing made from a dissection prepared by Dr. Bonney, Department of General Anatomy, Jefferson Medical College.)

mon with the tunica vaginalis testis. This is not true, however, of congenital herniæ, presently to be considered. (2) In enumerating the coverings of a scrotal hernia, the term dartos may be substituted for superficial fascia, the dartos being continuous with the superficial fascia in the lower third of the abdomen, but containing some muscle fibers. Although this point may seem trivial it has often been puzzling to students. Possibly this may be the most appropriate place to state that a scrotal hernia is merely a complete inguinal hernia, either indirect or direct, which has reached the scrotum.

A hernia coming through Hesselbach's triangle is called a direct or internal hernia: direct, for the reason that it bulges forward into the lower part of the inguinal canal without following the course of the cord obliquely downward from the internal ring; internal, because it lies to the inner side of the deep epigastric artery. As has already been stated, such a hernia almost always comes out through the middle inguinal fossa. We cannot agree with those writers who state that it more commonly emerges from the internal fossa. The latter is well protected by the rectus muscle in the manner previously described. That a loop of gut may work its

FIG. 5.—Showing the external abdominal ring after the intercolumnar fascia has been removed and the spermatic cord freed. Observe the opening in the aponeurosis of the external oblique muscle. (Drawing made from a dissection prepared by Dr. Bonney, Department of General Anatomy, Jefferson Medical College.)

way through the unprotected lower external corner of this fossa is not to be doubted, but it is much more likely to come out through the middle fossa, both lateral boundaries of which are less resistant than the strong tough margin of the rectus muscle.

A hernia escaping through the internal fossa has been termed an internal oblique or vesicopubic hernia. When the sac attains a considerable size it would be difficult to trace its origin unless the urachus could be readily distinguished. It is interesting to note that isolated cases are on record in which a hernia has separated

the fibers of the rectus muscle, and even pushed its way between the two recti, presenting at the linea alba.

The coverings of a direct hernia are, from within outward, peritoneum, preperitoneal fat, transversalis fascia, intercolumnar fascia, superficial fascia, and skin. The conjoined tendon is usually enumerated among the coverings of a direct hernia, but, as already stated, it is only in about 25 per cent. of all subjects that such a structure is actually present. When it does exist it is not pushed forward by the hernia, as has generally been taught, but the hernia slips around it, leaving it undisturbed. The tendon, when present, is firmly fixed to the crest of the pubes and iliopectineal line, and above is continuous with the substance of the internal oblique and transversalis muscles. That it could be pushed forward by a hernia is not at all probable, and, moreover, we have never found it displaced in those cases which we have observed at the operating table. In those cases in which there is no conjoined tendon it is possible that some of the attenuated lower fibers of the internal oblique may be carried forward by the hernia, especially if the latter is large. In about forty operations for direct hernia, however, one of us has never observed any muscle fibers in the coverings of the bowel.

It has been frequently stated that about one-fifth of all inguinal herniæ are direct. We are of the opinion that this estimate is much too high. We believe, as we have already stated, that not more than four or five out of every hundred are direct.

Etiology. The causes of inguinal hernia may be considered under three headings: (1) Congenital defects; (2) natural weakness of the abdominal wall in the inguinal triangle, and forces which increase this weakness; and (3) conditions which increase intra-abdominal pressure.

Congenital defects relate chiefly to faulty obliteration of the vaginal process of peritoneum. As previously stated, that portion extending from the internal ring to the top of the testicle usually becomes obliterated between the tenth and twentieth days after birth. The exact manner in which closure takes place is not known. It is generally conceded that obliteration begins in the middle part of the process, and some who have studied the subject also think it commences simultaneously near the internal ring. At all events there are a certain number of individuals in whom closure partly or entirely fails to take place. These persons are particularly predisposed to hernia. When the vaginal process remains open throughout its whole extent there is an uninterrupted passage from the peritoneal cavity to the scrotum into which the intestine can readily find its way, with the result that a hernia is produced. Such a hernia is called congenital, its sac being preformed. The contents of the sac are in contact with the testicle, but the testicle is not suspended by its cord within the sac in the manner that a

chandelier is suspended from the ceiling into the middle of a room, although we have found that undergraduates and practitioners of medicine alike sometimes entertain this erroneous conception of the relation of the one to the other. The inguinal portion of the cord lies in the retroperitoneal connective tissue, the scrotal portion is also posterior to the vaginal process, and one layer of peritoneum covers the testicle, namely, the visceral layer of the tunica vaginalis, which is identical with the posterior wall of the hernial sac. Thus, the testicle really lies behind the sac, being separated from the gut by a single layer of peritoneum instead of a double one, as is the case with acquired indirect inguinal hernia. These relations are well illustrated in Fig. 6.

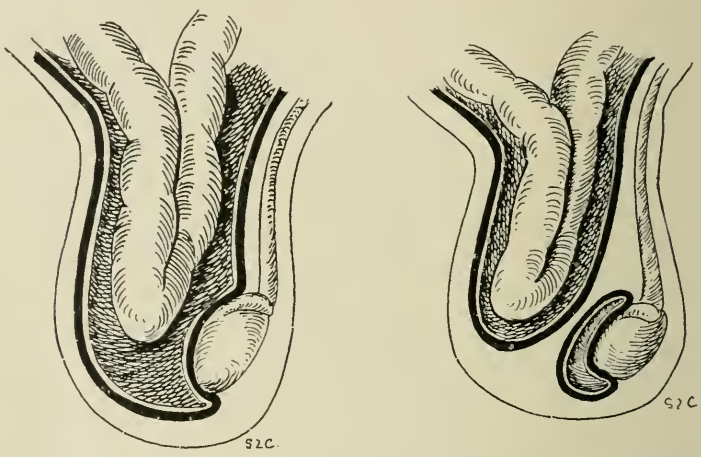

FIG. 6.—The left-hand illustration shows the relation of the testicle to the sac of a congenital scrotal hernia; the right-hand illustration shows the relation of the testicle to the sac of an acquired scrotal hernia. (Adapted from Gray.)

If the vaginal process has become obliterated below, but not above, there will be a channel extending from the abdominal cavity down to the site of obliteration. This channel is called the funicular process, and a projection of intestine into it is known as hernia into the funicular process. This variety differs from congenital hernia only in the extent to which it is possible for the gut to descend.

If the vaginal process is closed above, at or near the internal ring, but remains open below, two kinds of hernia may be formed. In some cases the septum between the abdominal cavity and the unobliterated vaginal process gives way slowly and allows the intestine to pass down behind the vaginal process. Such a hernia is known as an infantile hernia. Its sac is formed by the displaced septum together with the contiguous parietal peritoneum. The bowel has three peritoneal coverings, namely, the two walls of the vaginal process and the true sac of the hernia.

In other cases the septum gives way in such a manner that it and the contiguous parietal peritoneum are carried down into the unobliterated vaginal process. A hernia thus formed is termed an encysted infantile hernia. It has two peritoneal coverings, namely, the anterior wall of the vaginal process and its proper sac.[1]

Finally, a remnant of the vaginal process may persist above at the abdominal ostium, projecting slightly downward into the inguinal canal or appearing merely as a bulging at the internal abdominal ring. That such remnants predispose greatly to the formation of hernia is not to be doubted. In reality they constitute a preformed sac which becomes stretched and elongated by the pressure of the gut which impinges upon them from above. It is no doubt true that a considerable percentage of herniæ which are thought to be acquired originate in these remnants of the vaginal process, and therefore are originally due to a congenital defect. To maintain that all indirect herniæ are dependent upon such a defect, however, as has recently been done by some writers, is to ignore a very important structural condition normally present in the inguinal triangle which may rationally be considered to predispose to the escape of the intra-abdominal contents, namely, the arrangement of the transversalis fascia at the internal ring. This ring, although only a potential one, can be proved both embryologically and anatomically to constitute a weak spot in the parietes; and to deny the possibility and probability of this spot yielding to intra-abdominal pressure is, we think, unwarranted. Another point to be considered is the following: when a mere bulging of peritoneum is found at the internal ring in the adult subject, can it be positively determined whether this bulging represents a remnant of the vaginal process or the beginning of an acquired hernial sac? Certainly not. No one can prove it to be the one, no one can demonstrate it to be the other. The only way to decide the matter beyond question would be to cut a large number of children open in such a manner as to permit an examination for remnants of the vaginal process, sew them up again, and have them watched throughout life for the development of hernia. Until it shall have been determined in this manner that only those persons who possess remnants of the vaginal process develop hernia in adult life, we are unwilling to admit that all indirect inguinal herniæ are of congenital origin. We admit the probability of a considerable percentage being due to the presence of remnants of the vaginal pro-

[1] We believe that it would be better to abolish the old classification, according to which these herniæ are divided into congenital, infantile, etc., and call them all congenital. One is just as much dependent upon a defect of development as is another, and it certainly would be more accurate, and also probably less confusing to students, to speak of them as different forms of congenital hernia.

cess, but also believe that others develop irrespective of any defect in obliteration of this process.

Malposition of the testicle is a defect which is frequently associated with inguinal hernia. When the gland is retained high in the inguinal canal it has a tendency to dilate the internal ring and thus facilitate the escape of omentum and bowel. As the bowel passes downward its course may be obstructed by the testicle, so that it rises above the latter, with the result that the sac becomes curved. In cases in which the testicle has passed through the external ring, but has not descended into the lower part of the scrotum, the hernia pushes its way in front of the testicle, but usually does not descend below it. Non-descent of the testicle is also thought to be the chief cause of the rare pre-inguinal hernia, in which the sac escapes from the inguinal canal by pushing its way into the cellular tissue between the aponeurosis of the external oblique muscle and the skin. In this form the sac is divided into two or even three parts—one inguinal, one abdominal, and occasionally also a scrotal portion. The abdominal or superficial sac is the more important. It may be situated between the external abdominal ring and the anterior superior spine of the ilium, just in front of the ring, between the scrotum and the thigh, as in a case reported by Busch; or it may extend up toward the umbilicus, as it did in Broca's case.

Another variety most likely dependent upon a congenital defect is the inguino-interstitial, which develops in the inguinal canal, but works its way into the substance of the abdominal wall instead of emerging through the external ring, which is much smaller than usual or even absent. In the male such herniæ are associated with malposition of the testicle, and the narrowness of the ring was supposed by Tillaux and others to be due to the fact that the testicle had not passed through the external oblique muscle. Some cases, however, have been observed in the female, and, moreover, in others an accessory sac has been found in the scrotum, and the external ring has been observed to be wider than normal. Berger believes that the location of the sac in the substance of the abdominal wall is due to the presence of a congenital diverticulum in the vaginal process of peritoneum, which becomes enlarged by intra-abdominal pressure after the intestine engages in it. Such a hernia usually lies beneath the three lateral muscles of the abdomen, although some have been found between the internal and external oblique, extending high up into the abdominal wall.

Finally, there remains to be described the properitoneal hernia, for the pathogenesis of which a number of explanations have been offered. This variety of hernia also has two sacs, one deep and one superficial. The deep, or properitoneal sac is situated between the peritoneum and the transversalis fascia, and generally extends outward toward the anterior superior spine of the ilium. It may,

however, extend downward toward the pubes. The superficial sac occupies the inguinal canal and sometimes extends down into the scrotum, just as any other hernia which follows the cord may do. An angle of varying degree marks the divergence of one sac from the other and there is also a space, forming a vestibule, between them. Although some difference of opinion has existed as to whether properitoneal hernia is congenital or acquired, it has come to be pretty generally admitted that it owes its origin to a congenital defect. Thus, some have taught that the properitoneal sac is formed from a peritoneal diverticulum situated near the inguinal fossæ and that the superficial one is merely a spur carried out from the deep one by a process of dilatation as more and more of the intra-abdominal contents force themselves into it. Others maintain that both sacs are formed from the vaginal process, the deep from the iliac portion, which often presents a retro-inguinal fold well adapted for distension, and the superficial from the remainder of the process. The latter theory is the simpler and, moreover, is the one which those who have recently studied the subject most carefully are inclined to accept.

Perineal ectopy of the testicle may also be associated with hernia. This is infrequent, and is mentioned merely for the sake of completeness.

With regard to direct hernia it may be stated that this variety is never of congenital origin. The middle inguinal fossa is structurally a weak spot, through which the abdominal contents can be pushed by increased pressure from above. It has already been so well described under anatomy that further allusion to its structural peculiarities and relations are unnecessary.

Among conditions which increase the natural weakness of the abdominal wall may be mentioned diseases which impair the nutrition of the muscles, such as rickets, for instance; systemic maladies which produce great emaciation; abdominal tumors which impinge upon the anterior belly wall and distend it; pregnancy, which acts in the same way; accumulation of fat in the omentum and preperitoneal tissue, also tending to stretch the peritoneum; relaxation of the parietes incident to old age; and, finally, repeated efforts in lifting or dragging heavy weights.

It will be readily understood that some of these causes, such as pregnancy and accumulation of fat in the omentum, also increase intra-abdominal pressure. Other potent factors in augmenting pressure are cough, vomiting, and straining to empty the rectum or bladder. Bronchitis, emphysema, asthma, and whooping cough are not uncommonly the immediate exciting cause of hernia. In Berger's series of cases cough was considered an etiological factor more than eight hundred times. Elongation of the mesentery has also been considered a factor in the production of hernia. Although the mesentery is relatively longer in infancy than it is

in adult life, it is probably never so short as to prevent the intestines from coming out of the abdomen. This opinion is confirmed by the investigations of Mr. C. B. Lockwood, of London.

Age, sex, and heredity have also to be considered from the standpoint of etiology. In regard to age, it may be stated that hernia is most common in childhood and in middle adult life. Berger, who has studied the age incidence carefully, shows that by far the larger percentage of cases occur in children, that there is a constant decline until the thirtieth year, after which a steady increase takes place until the fifty-fifth year. It should not be forgotten, however, that hernia is relatively frequent in old persons on account of the relaxation of their abdominal wall and their susceptibility to bronchitis, asthma, etc. Were it not for the fact that they lead a shielded life and do not follow laborious occupations, they would be more liable to hernia than those in middle life. Berger's statistics also show that double hernia is rare in infancy and frequent in old age, reaching its maximum at sixty-five years.

All will admit that inguinal hernia is more common in males than it is in females. It is our belief, however, that it is more common in females than it has been thought to be, as the statistics of those hospitals and dispensaries in which female physicians are employed show a greater percentage of cases in women and girls than those institutions where only male physicians are in attendance. In over 800 operations for inguinal hernia which one of us has performed, not more than 50 were upon women.

The role of heredity is not thoroughly understood, but in our opinion it is of unmistakable etiological influence, as we know of many families in which several members have hernia.

Pathology. In studying the pathology of hernia the sac should receive consideration first. In shape it may be round, cylindrical, conical, or pyriform. In size it varies greatly. That portion just below the internal ring is called the neck, the remaining part the body. It differs in appearance according to the age of the hernia and the complications to which the hernia is subjected, being thin, smooth, and of the color of normal peritoneum in very recent cases; but thick, tough, corrugated, and white, yellow, or grayish in color, much resembling fibrous tissue, in those of long duration. In strangulation, it becomes dark red, purple, or black. In herniæ which have existed for any length of time it becomes blended with the other coverings of the bowel, frequently to such an extent that its separation is very difficult or even impossible. This is particularly true in regard to its relation with the transversalis fascia. In old herniæ there has usually been sufficient inflammation to blend the two layers inseparably together. One of us has had the opportunity of making postmortem dissection of a number of large scrotal herniæ, evidently of many years' duration, and has found that it is impossible to separate the coverings into more than two

layers after the herniated mass has been freed from the scrotum. One of these layers has invariably been traced to the aponeurosis of the external oblique muscle, the other has always disappeared within the internal hernial orifice. It is our opinion that the internal layer is composed of both peritoneum and transversalis fascia. In no case has it been possible to find any traces of cremaster muscle fibers. The external layer has always been adherent to the internal surface of the scrotum.

As the result of inflammation, which is bound to occur in the course of time, adhesions form which prevent the reduction of the sac with its contents. In the dissections just mentioned, the sac, together with other coverings, has been firmly attached to the pillars of the external ring, so that considerable cutting was necessary to free it.

Adhesions also not uncommonly form between the sac and its contents, a circumstance which still further renders reduction difficult or imperfect. Some of these inflammed areas may undergo calcareous change. Finally the inflammatory process may cause partial obliteration of the sac. It commences at the neck and may be confined to this part, but in some cases it extends downward and causes a variable degree of contraction. If obliteration at the neck be complete, a cyst may be formed in the lower part of the sac, owing to retention of the secretion which is produced. If a zone of obliteration occurs below the neck, the sac will be divided into two parts. These multiple sacs have been found both in congenital and acquired hernia. Longitudinal fibrous bands have also been observed. We have seen a few of these ourselves, and they have been mentioned by Okinczyc. No doubt others have also noticed them.

Hernia always produces changes in the abdominal parietes, the alterations depending upon the size of the hernia. The inguinal canal is always dilated, and the two abdominal rings are stretched and more or less approximated. The deep epigastric vessels may be displaced inward. Large herniæ cause pressure atrophy of the lower fibers of the abdominal muscles, and when they descend into the scrotum may likewise produce atrophy of the testicle. The extent to which the inguinal region may be weakened is well illustrated by the conditions which one of us recently observed in the case of a dissecting room subject who presented a large irreducible scrotal hernia which showed every evidence of having existed for years. The lateral muscles on the affected side in the lower part of the abdomen had become converted into a thin homogeneous layer, looking much like a piece of sheepskin; the aponeurosis of the external oblique had lost its firmness, presenting a number of small loose folds for a considerable distance above the anterior superior spine of the ilium, and the lower margin of the external ring had been pushed down below Poupart's ligament

almost to the saphenous opening. There was hardly any space between the superior margin of this greatly dilated external ring and the internal hernial orifice.

In reference to the contents of inguinal hernia, it may be stated that they are usually composed of omentum and small intestine. The large intestine, the stomach, the uterus and ovaries, the bladder, in fact any of the intra-abdominal contents but the pancreas may occasionally escape through the inguinal region. It is stated that a floating kidney has been found in the sac of an inguinal hernia.

The contents are subject to morbid changes. Thus the omentum is never normal except in very recent cases. Its adherence to the sac has already been mentioned. Its folds also become adherent to one another and sometimes portions of it become attached to the intestine. At the neck of the hernia the omentum is also often found converted into a fibrous band, this condition being the result of inflammation.

From a practical point of view the most important change which takes place in hernia is strangulation, by which is meant an occlusion of the lumen of the intestine together with arrest of its blood supply, produced by a constriction, which also renders the hernia irreducible if it was not already so before the said constriction occurred. As a result of strangulation the passage of both feces and gas is stopped and gangrene supervenes in the herniated bowel.

The most common site of strangulation in inguinal hernia we believe to be at the external abdominal ring, and not at the internal ring or within the sac, as has frequently been taught. Certainly this has been our experience, and we believe also that of most modern surgeons. In recent herniæ it may occur in the inguinal canal, between the two rings, being due probably to inflammatory changes in the transversalis fascia. In congenital hernia strangulation may be produced by strands or valves within the sac, or by the neck of the sac itself. In like manner it may result from the action of the inflammatory bands present in a partly obliterated sac, or be caused by the omentum, which, becoming adherent to the sac, forms a band that constricts the intestine. Rarely the bowel has perforated the omentum and become strangulated. Finally the possibility of torsion of the mesentery within the hernial sac must be borne in mind as a possible cause of strangulation.

The appearance of the strangulated intestine depends upon the length of time which the constriction has lasted. At first deep red, it gradually becomes darker and darker, until it may be almost or quite black. Its lustre disappears, patches of fibrinous deposit become visible, and here and there breaches in the continuity of the serous covering take place. These changes are the forerunners of gangrene, being shortly superseded by sloughs, which in turn may lead to perforation of the bowel. If the strangulation be relieved, the constriction of the intestine is plainly discernible.

The sac of a strangulated hernia is usually round in shape and almost always contains a considerable quantity of fluid. The sac, like the intestine, is much congested and may even share in the gangrenous process with which the latter becomes affected. We have often found the sac of a strangulated hernia to be of a dull slate color, with violaceous or dark brown patches scattered over its surface. The liquid in the sac is at first thin, clear, and of a light yellow color; but it soon becomes thick, turbid, and of a reddish or brown tinge, and contains flakes of fibrin. It may soon assume a fecal odor, caused by the presence of the colon bacillus, which migrates easily through the walls of the damaged bowel. Occasionally there is no fluid, the bowel being in direct contact with the internal surface of the sac. It is important to remember this fact and not to depend upon an outflow of fluid as a necessary sign that the bowel has been reached. Rarely it happens that only a segment of the bowel becomes constricted. To this condition the term lateral strangulation may be applied (Richter's hernia). Although the caliber of the bowel is diminished, it may nevertheless be possible for gas and fecal matter to work their way through. The bowel immediately below the constriction is usually found dilated, so that it may present the appearance of a diverticulum.

By an obstructed hernia is meant one in which the intestinal contents have accumulated to such an extent as to block the fecal current. The term incarcerated hernia has also been applied to this condition, but we believe it to be an inaccurate one, as it does not convey the idea of the condition which actually exists. If the term incarcerated is to be used at all, we believe it should be applied to those herniæ which are temporarily irreducible. We insist that this would be the correct use of the term. Obstructed hernia is probably much rarer than it was formerly supposed to be. Its existence has even been denied, but without sufficient reason, we are sure. We have seen the condition a number of times, and Nau has recently collected 23 authentic cases. It occurs almost always in herniæ of the large intestine, which have become irreducible as the result of inflammation. Intestine thus imprisoned loses its peristaltic power more or less, and this functional impairment, together with the mechanical conditions present, permit the occurrence of a gradual fecal obstruction.

A hernia may be attacked by tuberculosis or malignant disease. Cotte[2] has recently collected 136 cases of tuberculosis—the most thorough study of the subject with which we are familiar. The sac of the hernia alone, or both sac and contents, may be affected. Jonnesco states that the intestine may be diseased and the sac healthy, but his statement has not been generally accepted. In children the sac is usually the site of the tuberculosis; in adults

[2] Revue de Gynecologie, November and December, 1906.

both sac and contents are usually affected. Three forms of tuberculosis have been observed in the sac, namely, the miliary, the caseous, and the fibrous. Of these, the miliary is the most common. It is evidently the same lesion as that of miliary tuberculous peritonitis. There is generally considerable fluid in the hernial sac. Next in order of frequency is the caseous form, the fibrous being the rarest of all. The lesions of the intestines are no different from those found when the bowel becomes tuberculous in its normal habitat.

The neoplasms which have been found associated with hernia are carcinoma, sarcoma, and lipoma. They may affect the sac, the intestine, the omentum, or whatever organ is contained within the sac. The lipomas naturally develop in the omentum.

In another paper the diagnosis and treatment of inguinal hernia will be discussed.

THE ACTION OF THE SHORT ROTATORS ON THE NORMAL ABDUCTION OF THE ARM, WITH A CONSIDERATION OF THEIR ACTION IN SOME CASES OF SUBACROMIAL BURSITIS AND ALLIED CONDITIONS.

By James H. Stevens, M.D.,

of Boston, Mass.

Codman, of Boston, in a recent monograph on lesions of the shoulder-joint due, in his opinion, to trauma or to primary inflammation of the subacromial bursa, has done more to clear up the interesting subject of painful shoulders, and to explain certain definite restrictions of motion between the humerus and the scapula than has been accomplished by any other investigator, and his masterly interpretation of the mechanics of the shoulder-joint is so simple, clear, and conclusive (as far as he goes) as to establish almost beyond question his contention as to the primary action of the supraspinatus in abduction of the humerus preliminary to the action of the deltoid, which is thus permitted to come into action and exert its force in the proper direction to complete the arc of abduction. He has followed his theory through the operating room and into the dissecting room, and has conclusively established his contention by anatomical specimens showing the pathological changes following rupture of the supraspinatus, with the subsequent loss of the power of abduction in these cases. He has applied his researches into the normal mechanics of the scapulohumeral joint to the practical treatment of a class of shoulder lesions showing restriction of motion and tenderness below the acromion, which, as he says, constitute by far the largest class of patients applying to any

surgical clinic for the relief of trouble referable to the shoulder-joint. For this class of cases he has adopted Kuster's term of subacromial bursitis in place of the subdeltoid bursitis used by him in his first paper, although the latter term would seem to be by far the better, since more of the bursa is subdeltoid than subacromial, and since Piersol, in his recent anatomy, used the term subdeltoid in his description of the bursa. Whatever term is used, the credit must go to Codman for the most convincing description of the mechanics of this complicated joint. And in the light of his investigation the real action of the joint does not appear to be so complicated after all. A study of normal shoulder action is necessary for the proper understanding of his cases, and the few which have come under observation which we hope to demonstrate are departures from the regular types which he has described. If one considers anatomically the origin and insertion of the various muscles surrounding the shoulder-joint, and if one bears in mind the fact that the real capsule of the joint is formed by these surrounding muscles, and not by what is known as the capsule, with its ligamentous bands, which is a lax structure neither aiding nor opposing the normal motions of the joint, it would seem to be easy to assume, and equally easy to prove from a study of the anatomical structure, that the first few degrees of motion in abduction are due to the supraspinatus contraction, and is preliminary or preparatory, as it were, to the action of the deltoid.

Piersol says that the capsule of the shoulder-joint, among other uses, by means of its tense, firm under surface helps to prevent the arm from being raised beyond a certain point in abduction, but it is extremely doubtful if the capsule has any such action, although this one is by far the more probable of all the uses ascribed to it by the various anatomists, and it is probable that the capsule per se has very little retarding action on the movements of the joint itself. As Codman says, it is necessary to disregard our previous teaching and conception of the structure which has been called the capsule. The real capsule of the joint is made up of the muscles which surround it. Codman's contentions, which are, I think, accepted for the most part or will be so accepted by all who carefully examine the anatomical structure of the shoulder-joint, bearing in mind the laxity of the capsule, are, briefly, that the first few degrees of motion in abduction of the humerus are always due to the contraction of the supraspinatus acting on the short arm of the lever, the fulcrum of which is on the glenoid, the long arm of the lever extending downward through the arm (Fig. 1).

It will be readily seen that the fulcrum of such a lever must be a constantly changing point, and that the action of such a lever would be comparatively slight, and such seems to be the case. From its insertion high up on the greater tuberosity of the humerus the action of the supraspinatus must be limited in power, because the power in that case would be applied so near the fulcrum, while the weight

would be far removed (see Fig. 1: *A*, power, *C*, weight, of the lever *A*, *B*, *C*), and it is only sufficient to move the arm outward in abduction sufficiently to permit the line of pull of the deltoid (the mean line of the pull of all the fibers of the deltoid, assuming that these muscle fibers work together, which would be the line *H-E*, in Fig. 2) to fall superiorly to the fulcrum (*B*, Fig. 2) of the lever, in which case the deltoid could expend its force in abducting the arm still more, instead

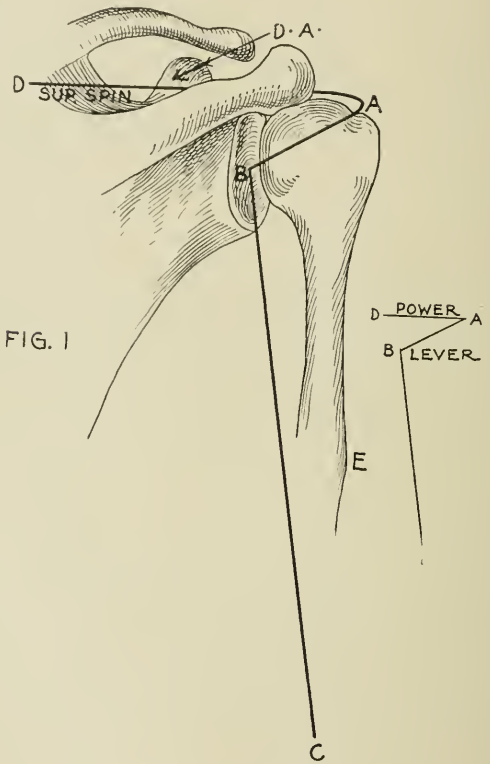

FIG. 1

Fig. 1.—*D A*, the line of pull of the supraspinatus; *A*, the power applied to the short arm of the lever; *A*, *B*, *C*, the lever; *B*, the fulcrum on the glenoid; *C*, the weight to be lifted. The contraction of the supraspinatus can swing the arm, obviously, in but one direction—abduction.

of uselessly forcing the humerus upward, as it would do acting in the line (*H*, *E*., Fig. 2) of its pull when inside (toward the body side) of the fulcrum on the glenoid, thus forcing the head of the bone against the lower surface of the acromion. It will be seen, therefore, from the origin and insertion of the deltoid (from the spine of the scapula, the acromion, and the outer third of the clavicle into the deltoid tubercle midway on the external surface of the shaft of the humerus) that its action alone, provided its fibers contracted as a single muscle,

would not be expended in the right direction to abduct the humerus, but rather would act uselessly in forcing the head of the bone against the under surface of the acromion, or if it worked too soon after

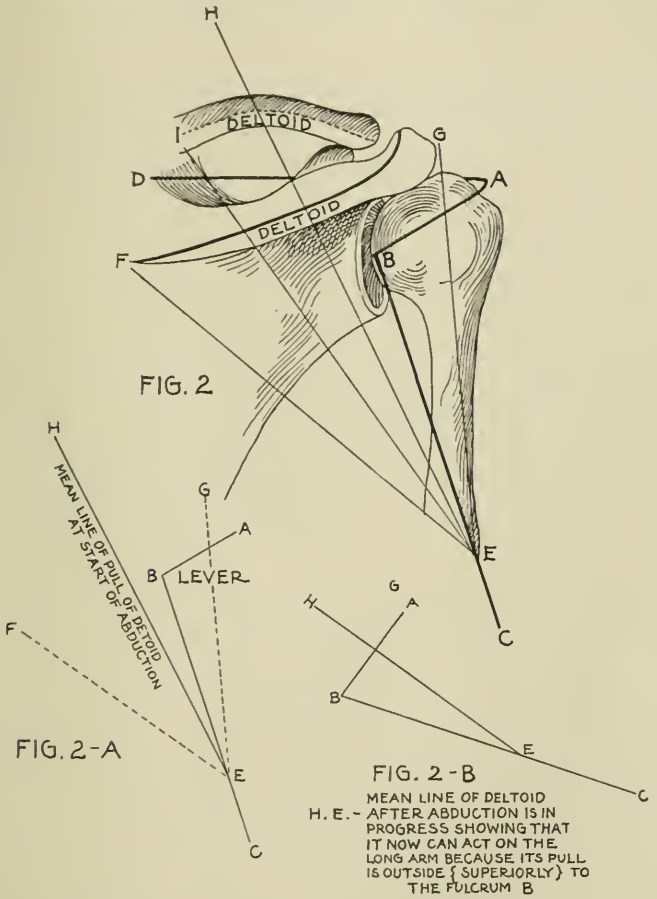

Fig. 2.—Showing the lines of pull of the fibers of the deltoid, providing these fibers acted together, H, E, being approximately the line of such pull, showing that in this case it could not act on the lever A, B, C, to abduct it. Assuming that the acromion portion of the deltoid acted as a separate muscle or as a separate contraction, then it could (consider the line G, E, and its effect acting at E on the long arm of the lever A, B, C) aid in starting the humerus upward in abduction, but should its fibers act simultaneously the line of pull would be inside the fulcrum, B (Fig. 2, A), and provided it was in contraction it would act in opposition to the line of pull of the supraspinatus, D, A. In order to act on the long arm of the lever the line H, E, must fall outside the point B (fulcrum), and this is possible only after the long arm of the lever (the humerus) has been partially abducted (Fig. 2, B).

primary action by the supraspinatus would force the greater tuberosity to take its fulcrum from the under surface of the acromion process (Fig. 2). If two lines of pull are at right angles to each

other, and equal force is applied in both directions, the body acted upon would always feel the force thus expended in a line midway between the two equally acting forces, and if one considers the origin and insertion of the deltoid it will be clearly demonstrated that if this muscle were acting alone it would not raise the arm in abduction (Fig. 2).

The normal mechanism of the shoulder-joint is, therefore, up to this point probably as follows: The supraspinatus contracting starts the motion of the humerus in abduction, but the supraspinatus from its insertion high up on the upper facet of the greater tuberosity of the humerus, is acting at a considerable disadvantage. It is the power applied to the short arm of a lever whose fulcrum is on the glenoid (Fig. 1, *B*, fulcrum, *D*, *A*, power). In other words, the lever is arranged, power, fulcrum, weight, the short arm of the lever being joined to the long arm at the fulcrum, at an angle which is constantly changing, and constantly becoming a more acute one as the arm rises in abduction, but which is at the beginning of motion usually a little over 45 degrees, and such a lever cannot, as said before, be one of great power. It is, however, sufficient to start the humerus outward, and begin the arc necessary to abduction, probably even up to 30 degrees, and during this time, unless the acromial portion of the deltoid acts as a separate and individual muscle from the rest of the deltoid, it can assist the action of the supraspinatus in no way (Fig. 2).

When the lever (humerus) has swung beyond a certain point the great deltoid contracting takes up the work, but it cannot do this until the supraspinatus has abducted the long arm of the lever (the humerus) sufficiently so that the deltoid contracting (the mean line of its pull when the muscles contract falling superiorly to the point of fulcrum on the glenoid) can act on the long arm of the lever, thus converting it into a more powerful one, where the power is applied as fulcrum, power, weight, and which is still aided by the power of the supraspinatus still acting on the short arm of the same lever. Should the deltoid contract before this point is reached, then the supraspinatus tendon is caught between the head of the bone and the acromion process, and either torn across or injured. As the arm reaches a certain degree of abduction the power of the supraspinatus, from its anatomical consideration, would seem to be slight, its work having been accomplished, but it would seem that its anatomical position is such as to render it of value as a tractor, possibly assisted by the lower tense part of the capsule (Piersol) to prevent slipping downward of the fulcrum (the articular surface of the head on the glenoid), thus preventing destroying the efficiency of the lever. If one looks at Fig. 3, he will see that this is the only part of the joint which is not surrounded by powerful tractors (the inferior portion), and motion here is prevented by the supraspinatus, tense superiorly, and possibly by the under surface of the capsule. If this should be

so, it would be the only value as far as aiding or retarding motion in the joint rendered by the so-called capsule. Another feature to be considered is the subacromial bursa which comes into play, as Codman clearly demonstrates, when the arm is abducted, and the tuberosity of the humerus is approaching the under surface of the acromion by the interposition of its well-lubricated surface between these two bony points (preventing the tuberosity from taking its fulcrum on the under surface of the acromion, and thus restricting further motion in abduction), permitting (the force of the deltoid continuing) the articular surface of the humerus to turn in but one direction, namely, towards the glenoid.

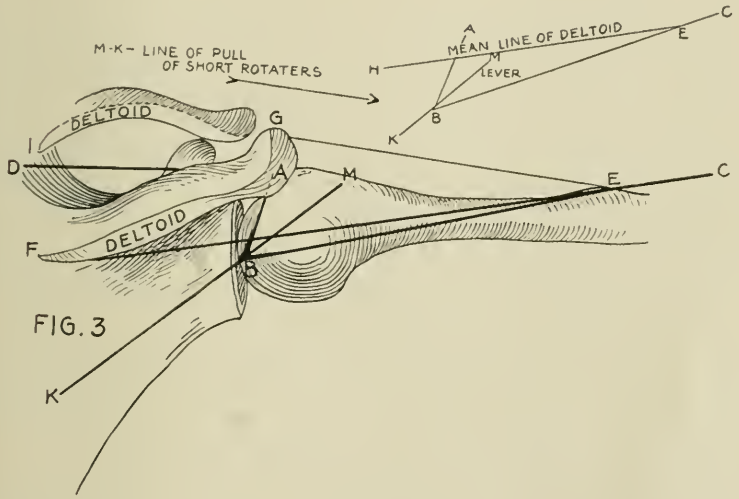

FIG. 3.—This shows the lines of pull of the deltoid when the arm is partly abducted, *G, E*, *F, E*, and *I, E*, and the line of the pull of the supraspinatus, *D, A*, acting on the short arm of the lever *A, B, C*, while the deltoid acts on the long arm of the same lever, its power being applied to *E*, a point between the weight *C* and the fulcrum *B*. Note also that both lines of pull, deltoid and supraspinatus, are above the fulcrum *B*, and unless there is another force applied, the fulcrum of the lever would be unstable and render worthless any amount of power applied. That force, *K, M* (the line of pull of the infraspinatus and teres minor behind and of the subscapularis in front) is supplied by these three short rotators, without which the head of the humerus would slip upward, answering the pull of the supraspinatus and deltoid. Note that the line of pull of the short rotators passes through the fulcrum, therefore it acts merely as a tractor and exerts no force either on the long or short arm of the lever, consequently offering no impediment to the abduction, while at the same time rendering such action possible by keeping the fulcrum firmly in its place.

Codman's contention is that the subacromial bursa accomplishes this purpose, and he goes no further. If we consider now the origin and insertion of these two muscles together, both acting and the humerus partly raised in abduction, we come to the consideration of the third part of the mechanics of the shoulder-joint in abduction, a very important part, indeed, and one which has not been considered

(so far as I know), but which I believe absolutely essential to the normal action of the joint, and which action is especially to be remembered in the class of cases classified by Codman, and also in those cases the consideration of which I shall take up, and which are closely allied to the condition known now as subacromial bursitis. As the arm continues to abduct, the line of pull of the supraspinatus and the line of the deltoid pull are coming nearer and nearer into line (Fig. 3, *D*, *A*, and *F*, *E*), and they are both upward and inward, and both forces are superior now to the point of the fulcrum (Fig. 3, *B*). Unless there were another force to offset this upward and inward pull of these two muscles, the tendency would still be to force the tuberosity against the under surface of the acromion process, and it is inconceivable that nature should leave such a task to the slippery surface of any interposed bursa. While, undoubtedly, this interposition of the bursa is necessary, and acts to direct the articular surface of the humerus against the glenoid, and also permits the tuberosity to pass smoothly under the acromion, still, without the presence of another force acting at an entirely different angle than either the supraspinatus or the deltoid, the articular surface, while it might and probably would be directed to the proper fulcrum, would not be held firmly enough to permit of that efficiency which is present in the normal joint, and it would permit riding up of the tuberosity against the acromion with unnecessary force.

Consider the lines of pull of these two muscles inward and upward, and we can see that the tendency would still be to pull the tuberosity upward either with or without the bursa (Fig. 3). Another force applied here is an absolute necessity. Such a force is supplied when we consider the origin and insertion of the short rotators.

Following the anatomical law that the strength of opposing muscles, or the strength of the sum of opposing muscles, is equal or nearly equal, then the subscapularis and the infraspinatus, plus the teres minor, must be equal, the one an inward rotator, the others outward rotators.

But consider the insertion of these muscles so near the head of the bone, and it becomes more probable that the simultaneous contraction of these three muscles, following the primary pull of the supraspinatus and simultaneous with the contraction of the deltoid, is necessary to pull the head of the humerus into the glenoid, and fix it firmly there forming the fulcrum, and that without this additional pull the action of the deltoid would still be to force the tuberosity to take its fulcrum from the under surface of the acromian, and that this action is equally important with their well-recognized action as rotators of the arm. These three short rotators or tractors, as they should be called, form a practical sling around the anatomical neck of the humerus, and their lines of pull, when acting together, would not only enable the articular surface to find its proper fulcrum, but would also counteract the tendency of the combined action of the

supraspinatus and the deltoid to pull upward the tuberosity (Fig. 3). Consider these muscles in their relation to the arms of the lever, and we see (Fig. 3) that the line of pull of these short rotators passes practically through the fulcrum of the lever, and, therefore, while exerting power sufficient to keep the articular surface constantly and firmly against the glenoid, and the tuberosity away from the acromion, thus permitting abduction of the humerus, the line of pull, passing as it does practically through the fulcrum, it could not in any way act on either arm of the lever to hinder elevation or depression of the humerus. For a demonstration of this contraction of the subscapularis, infraspinatus, and teres minor, it is only necessary to stand behind a model who is abducting his arm, and note with the hand the tenseness of these muscles, especially after the humerus has swung past the horizontal line, in contrast with the flabby relaxed belly and tendon of the pectoralis major in the same position. Beyond a certain point, however, the infraspinatus and teres minor behind and the subscapularis in front, which have acted together up to this point solely as tractors to hold in place the articular surface of the humerus, may possibly take on an added action, although it is probably to a very limited extent, inasmuch as abduction of the humerus is by this time a nearly completed act, namely, that of abductors also, as aids of the deltoid.

The action of the supraspinatus is practically little by this time, and the short arm of the lever has ceased to be of much advantage. Its force is probably expended more as a tractor to help hold firmly the articular surface of the humerus from slipping on its fulcrum, and it is in this possibly aided by the tense, firm inferior surface of the capsule, as suggested by Piersol. A new lever, therefore, takes the place of the old (Fig. 4). Considering the articular surface of the humerus as the segment of a circle, turning constantly as the arm abducts and forming a constantly changing fulcrum or point on the glenoid, the moment that the lines of pull of the short rotators have swung upward through the fulcrum of the old lever they cease to act simply as tractors, and a new lever, or a new short arm, is formed instantly. Or, whenever the line of pull of these short rotators transects the segment of the circle above the fulcrum they become the power on the short arm of a new lever, the long arm of which remains the same (Fig. 4, *M*, *B*, *C*), and their action would be to raise the arm to a still higher level, assisting the deltoid.

The reverse of this is also true to a limited extent. Thus, the short rotators act in abduction of the humerus when the arm has passed a certain level, and act to pull it downward, adduct, after it has fallen below another fixed level on its downward course, depending on the relation of the line of pull to the fulcrum of the lever at *B*.

To recapitulate, it would seem that from a theoretical and practical consideration of the shoulder-joint, always taking into consideration the origin and insertion of its various muscles, and considering

them as the power applied to the lever (the humerus), whose fulcrum in the normal shoulder would be a constantly changing point, it would seem that the necessary movements would be as follows: (1) Supraspinatus contraction would abduct the arm and tend to tip the articular surface of the humerus, so as to bring it against the glenoid, but its action and its strength are slight (30 degrees). The powerful deltoid, contracting, forces the tuberosity against the under surface of the acromion, where interposes the well-lubricated subacromial bursa, which, the force of the pull continuing, permits the articular surface to turn in but one direction, namely, toward the glenoid.

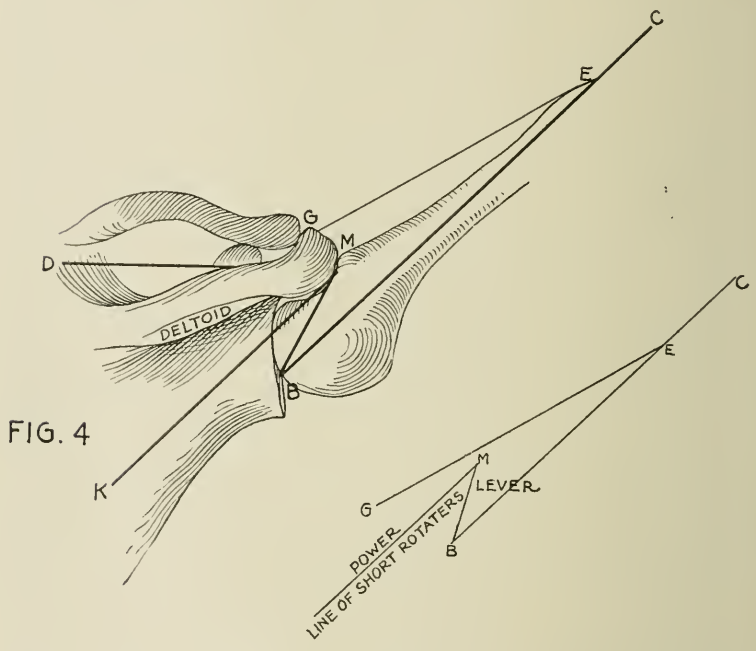

FIG. 4

FIG. 4.—*G, E*, the power applied by the deltoid at *E* to the lever *M, B, C; M*, the point where the power is applied to the short arm of the new lever by the short rotators; *B*, the fulcrum on the glenoid; *K, M*, the line of pull of the short rotators. Note that the line of pull of the short rotators (subscapular, infraspinatus, and teres minor) is in this position of the arm superior to the point of fulcrum *B*, therefore it may act slightly as an additional abductor.

To complete this action and in order to form a theoretically perfect mechanical entity, we must invoke another force or pull, fixing the head of the bone in the glenoid, yet having no restriction on the abduction of the arm. Such a force we have seen that we have in the combined and simultaneous action of the subscapularis on the one side, and the infraspinatus and teres minor on the other. In other words, a narrow, firm sling pressing the head of the bone into

the glenoid, yet not preventing abduction, but even acting above a certain point as an aid, and varying in forward and backward swinging exactly as the muscles of one side over or under acted in consideration with the opposing muscles, always, however, exerting sufficient pull to keep the articular surface firmly against its fulcrum on the glenoid cavity of the scapula.

To come now to a consideration of the subject of shoulder lesions involving the bursa, or of lesions presenting symptoms which closely ally them to lesions of the subacromial bursa. Codman has investigated this subject thoroughly, and has written a monograph on the subject of subacromial bursitis which will become a classic. He has divided his cases into three classifications:

Type I, the acute or spasmodic form with localized tenderness just below the acromion process of the scapula, over the bursa, and to the outside of the bicipital groove. This tender point disappears under the acromion on abduction because no adhesions are present in the bursa to prevent, and this sign is considered pathognomonic of this type of bursal lesions by him (Dawbarn's sign). In abducting or in external rotation, after a certain point is reached the scapula is locked by spasm, and moves with the humerus. In mild cases, with but little spasm, the patient thinks he cannot abduct his arm, but will allow passive motion, and is usually perfectly able to abduct once he has overcome his fear of pain. Codman thinks this is explained by the unwillingness of the supraspinatus to start the pull on its sensitive tendon.

In *Type II* (the subacute or adherent type) actual adhesions exist in the bursa, and there is an actual mechanical limitation to abduction and external rotation. Localized tenderness may or may not be present, according to the degree of existing inflammation. Dawbarn's sign is absent—the tuberosity cannot pass under the acromion because of adhesions. Abduction and external rotation of the humerus on the scapula are limited; 10 degrees of free motion in abduction exist because the bursa is not brought into play before this point is reached. Beyond this arc of 10 degrees the scapula accompanies the humerus in all its motions, active or passive. Pain is in the same distribution as Type I, and in severe cases it may resemble a brachial neuritis.

Type III. The chronic type; the essential characteristics are painful motion, but the full arc exists. The trouble is due in this class to slight irregularities of contour of the base of the bursa usually external to the bicipital groove. Motion, instead of being smooth, is jerky and interrupted in its arc. Localized tenderness may or may not be present. If present, then Dawbarn's sign is present. Abduction and external rotation are but slightly interferred with, but at some point in abduction acute tenderness is experienced, which disappears as soon as the tuberosity is under the acromion. The scapula does not accompany the humerus. There may or

may not be pain, and if present it is often felt at the insertion of the deltoid.

While forced to agree with all which Codman says in his paper, as far as he goes, and also that nearly all of the shoulder cases will fall into one or the other of his classification, it has been my fortune recently to come in contact with two cases, out of a total of seven, which it seems to me impossible to class with any one of the three types which he has given, and it has seemed that there was possibly a class of cases, limited in number perhaps, and usually or always the result of trauma, the symptoms of which are practically those of subacromial bursitis, and which may be, and perhaps always are, accompanied by some inflammatory changes of the bursa, but which show distinctive symptoms sufficient to call attention to the involvement of the short rotators, the action of which Codman does not take up in his article.

These two cases, one following a dislocation unreduced for twelve hours, and the other a fall with the arm in extension, presented the same symptoms: No tender point over the bursa beneath the acromion; the greater tuberosity disappeared under the acromion; a normal active arc without pain up to 23 to 25 degrees, and then restriction, but not on account of pain; passive motion, practically throughout the entire arc; external rotation lost, internal rotation preserved; no paralysis of the deltoid.

From an examination of these cases, I am forced to the conclusion that the greater number will readily fall into Codman's classification, and that they will also show a subacromial bursitis with or without adhesions; that at autopsy evidence of tearing or fraying of the tendon of the supraspinatus will be more often observed than evidence of injury to the short rotators, but it is my contention that there is a limited number of cases which, with or without a well-defined bursitis, present certain varieties of symptoms which are not due entirely to inflammation or adhesion of the bursa, but which are due to either a tearing or more frequently an inflammatory involvement of these short rotators, more particularly the infraspinatus and the teres minor. There is no tender point over the bursa. In the acute cases of Codman there is a tender point. The greater tuberosity disappears under the acromion without pain. In his cases of acute type the tuberosity disappears under the acromion, but with pain, and in the adherent type it does not disappear at all. His cases show 10 degrees of abduction before pain. The cases under observation show 23 to 25 degrees of active abduction, and then motion was not stopped on account of pain; it was inability to lift the arm, the deltoid visibly acting. Inward rotation was preserved, and this would apply equally well to bursitis alone, the preservation being due to less involvement of the subscapularis, because it comes to a less degree into contact with the bursa. External rotation was lost, and Codman, contradicting Kuster, says that it is also true of subacromial bursitis,

probably accounted for by the proximity of the posterior short rotators to the floor of the tender bursa.

In the two cases cited there is, however, no tender bursa, at least not on pressure, passive motion is practically preserved, which would not be the case in adherent bursitis, and would be painful in acute bursitis, while in his chronic type external rotation is preserved. The full arc of passive motion, the absence of tender points over the bursa, the preservation of internal rotation, the loss of external rotation, 23 to 25 degress of painless, active abduction, and no deltoid paralysis, together with tenderness over the bellies of the infraspinatus and teres minor, and especially marked and referred outward to their insertion on using faradization, would seem sufficient to warrant us in recognizing a lesion of the shoulder-joint usually following trauma, which may be accompanied by a low grade of bursal inflammation, and may always be so accompanied, but whose chief pathological change is either a complete or incomplete rupture, or, more frequently, the involvement in an inflammatory lesion of the tendons of the infraspinatus and teres minor, and less frequently of the susbcapularis.

There is also a class of fractures involving the greater tuberosity of the humerus, not so rare as has been assumed, which invariably lead to a set of symptoms resembling bursitis. Some of these cases undoubtedly develop a bursitis, and to that condition their symptoms should direct attention, but some of them present symptoms identical with those described in the two cases reported above, and it has seemed to me that involvement of the short rotators, which can be proved by the *x*-rays, is more of a factor in these particular cases than an inflammation of the bursa. In the two cases which have presented themselves to me, in which the accompanying *x*-ray prints show the nature of the lesion, there has been some question both among the clinicians and the *x*-ray men as to the exact anatomical injury, but it will be plain to anyone, without entering into a discussion of that point, that the greater tuberosity has been, at least, torn off.

In Fig. 5 it shows as an absolute loss of substance, and for the purposes of this paper it is sufficient to deal with it simply as a lesion of the tuberosity. The loss of motion to have been expected here was absolute inability to abduct and loss of external rotation with the preservation of internal rotation. This was exactly what this patient showed. He could move his arm in the frontal plane because his biceps was not involved, but he could not start his arm from the side in abduction because his supraspinatus was torn loose, and external rotation was lost because the same thing had happened to the infraspinatus and teres minor. The deltoid was not involved, and could be seen to contract vigorously both voluntarily and by faradization, but the arm was not abducted, and could not be held when passively abducted. Internal rotation was preserved because the position of the subscapularis was not involved in the injury and its belly and

insertion were both intact. In this class of cases when passive motion is free and full, and the only restriction is in active motion, should there exist the 10 degrees of normal supraspinatus abduction

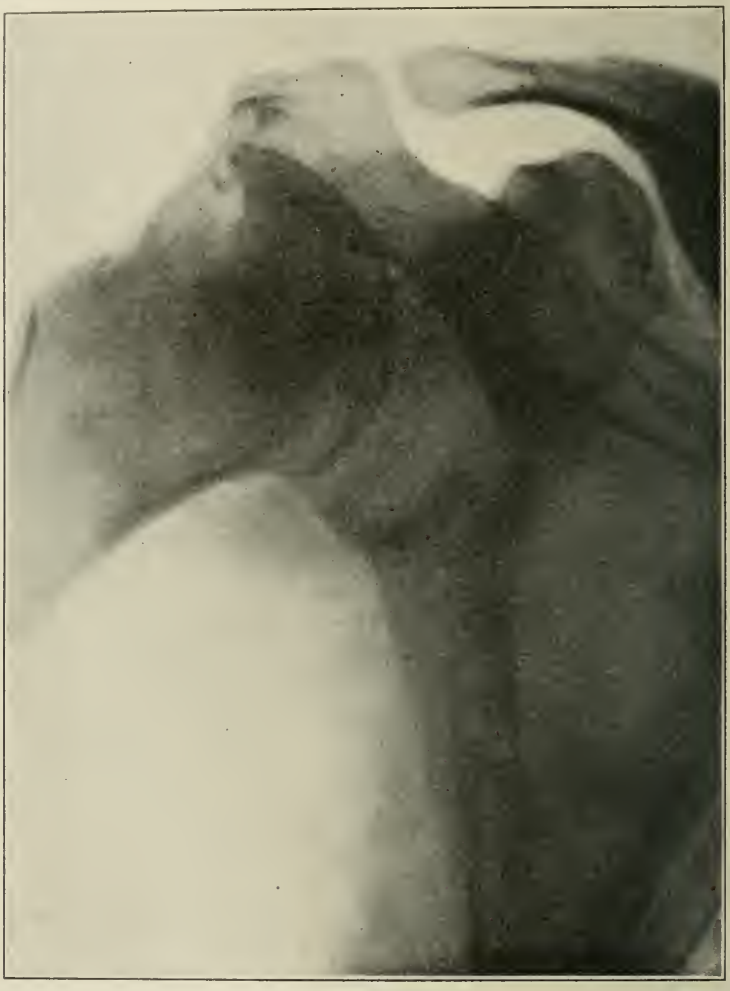

FIG. 5.—Fracture of the great tuberosity of the humerus, involving the insertions of the supraspinatus, infraspinatus, and teres minor, with displacement of the fragment. There was entire loss of abduction; external rotation was lost permanently, but the power to abduct was recovered; internal rotation was preserved; forward and backward motions were possible with slight pain. (*X*-ray by A. W. George, M.D.)

mentioned by Codman, then it is quite clear that that tendon has not been torn away, and if the motion is painless, it is equally certain that it has escaped injury.

If the humerus is abducted painlessly to 23 or 25 degrees and the deltoid contracts and still active abduction is impossible beyond that point, with the full arc on passive motion, and the disappearance of the tuberosity under the acromion, then the cause of the impairment of motion is, I am convinced, to be looked for in a lesion of the short rotators. If a painful point exists (Dawbarn's sign) it is possible that a bursitis is the cause of the loss of motion, but I am con-

FIG. 6.—Fracture of the great tuberosity of the humerus, involving the tendons of insertion of the supraspinatus, infraspinatus, and teres minor; radiogram two and one-half months after the injury. Six months later external rotation was still lost. Internal rotation was preserved at all times. Some restriction in abduction still persists when the arm swings beyond the horizontal position. There is no pain; note callus. (*X*-ray by A. W. George, M.D.)

vinced that there is a certain number of cases with the symptoms mentioned in which the lesion is one involving the short rotators, more frequently the infraspinatus and teres minor, and that this is the principal lesion, whether or not associated with a bursitis. Among the

cases which Codman cites is one in which passive motion was free and the tuberosity disappeared under the acromion, but with tenderness. The deltoid, to quote his own words, was as big as a ham, and yet when abducted passively he could hold his arm only by tremendous effort, and then only for a few seconds. The weight of a finger would send it downward. It seems as though this might be equally characteristic of his first or third type, or of the class of cases to which I have called attention, and that the mechanism involved here is that either from injury or from their proximity to the base of the tender bursa the short rotators refused to do their work, therefore not holding the fulcrum firmly in place and permitting it to slip on the

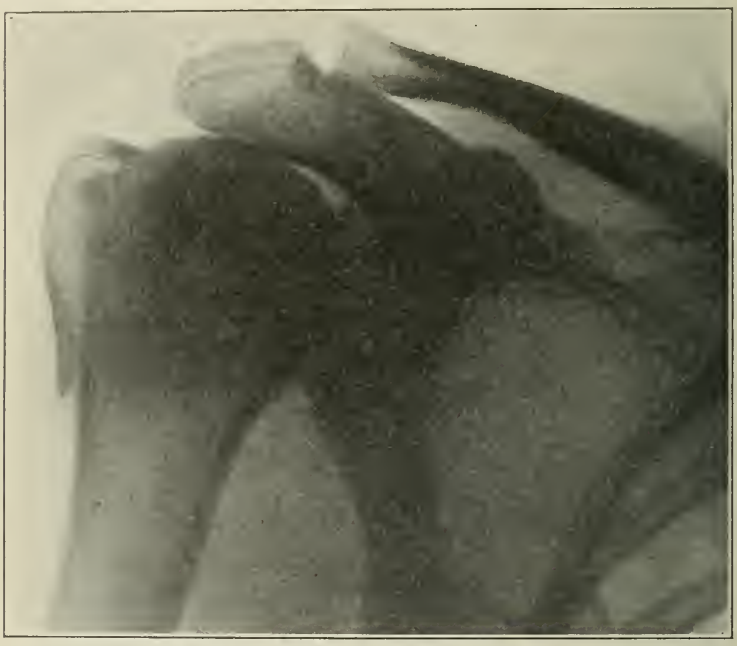

FIG. 7.—Fracture of the great tuberosity of the humerus, involving the insertions of the supraspinatus, infraspinatus, and teres minor. The patient could start the arm in abduction only a few degrees, however, and with pain; motion forward and backward was preserved but restricted; external rotation was lost (involvement of the infraspinatus and teres minor); internal rotation was preserved (subscapularis intact). All motions were finally recovered. (*X*-ray by A. W. George, M.D.)

glenoid, in which case, a very large deltoid would be of as little value as one of microscopic size. A lever to be of any value must have a fixed fulcrum, and it must be, for the time being, at least, a firm and solid one. Disturb it in the slightest degree or render it unstable, even for the infinitesimal part of a second, and unless one instantly substitutes another point for the one disturbed, the greater the force at the long arm of the lever the quicker and more surely will that lever fail.

SPASTIC PARAPLEGIA DATING FROM CHILDHOOD (LITTLE'S DISEASE?), WITH LITTLE OR NO DEMONSTRABLE LESION IN THE PYRAMIDAL TRACTS.

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It is not the object of this paper to discuss the classification of cases of cerebral spastic palsies, or the relation which Little's disease bears to these cases; but rather to call attention to the remarkable and interesting fact, already discussed by Spiller[1] and others, that spastic paraplegia may occur with little or no demonstrable degeneration in the pyramidal tracts of the cord, and to add a case in point.

While a few cases have been reported bearing on this subject, the literature is not very extensive, and the last word has not yet been said. Although in some cases degeneration of the pyramidal tracts has been described, this has been caused either by a spinal lesion (Dejerine,[2] Spiller), a meningo-encephalitis (Sachs[3]), a sclerosis of the motor cortical areas (McMutt[4]), or a venous effusion into the meninges, causing destruction of the cortex (Muratow[5]). The cases of porencephalon and atrophy of the motor and other convolutions, without degeneration in the pyramidal tracts (Railton,[6] Ross,[7] Phillip and Cestan[8]), present a different problem; and yet more difficult of explanation are those cases in which there is little or no demonstrable lesion, either in the cortex or the motor tracts of the spinal cord, as in the cases of Spiller, Rolly,[9] Dejerine, Haushalter and Collins,[10] and in my own case to be reported herewith.

In some of the cases no true degeneration of the pyramidal tracts is found, and in these have been described: a fineness of the fibers of the pyramidal tracts (Spiller, Biswanger[11]), a scarcity of fibers (Ganghofner,[12] Gerlich[13]), incomplete myelization without diminution in the number or size of the fibers (Haushalter and Collins), agenesia or imperfect development of the pyramidal tracts (Dejerine, Spiller, Anglade, Jacquin,[14] Ganghofner, Cestan,[15] Steinlechner-Gretschisch-

[1] University of Pennsylvania Medical Bulletin, January, 1905; Jour. Nerv. and Ment. Dis., 1898, 81.
[2] Revue neurol., 1903, xi, 601. Comp.-rend de la soc. de biol., 1897, p. 261.
[3] New York Med. Jour., 1891, v, 1.
[4] Amer. Jour. Med. Sci., 1885, 59.
[5] Deut. Ztschr. f. Nerv., 1896-97, x, 272.
[6] Brit. Med. Jour., 1892, i, 441.
[7] Brain, 1882, 473.
[8] Comp.-rend. de la soc. de biol., 1897, p. 1080.
[9] Deut. Ztschr. f. Nerv., 1901, xx, 152.
[10] Comp.-rend. de la soc. de biol., 1905, lix, 223.
[11] Virchow's Archiv, 1882, 427.
[12] Ztschr. f. Heilk., 1896, xvii, 203.
[13] Arch. f. Psych., 1891-92, xxiii, 201.
[14] L'encephale, 1909, 252.
[15] Prog. Med., 1899, x, 102.

nikoff,[16] and Oddo[17]) or atrophy of the pyramidal tracts (Ganghofner, Steinlechner-Gretschischnikoff, Oppenheim[18]); while an effort has been made to connect some of these conditions with degeneration of the cortical ganglion cells, or diminished number, or absence of these (Biswanger, Rolly, Otto,[19] Köster,[20] Sachs).

This brief and hasty review of the pathology of the cases of cerebral spastic palsies dating from infancy or birth, does not explain satisfactorily at once the cause of the spasticity, and the paralysis in all the cases. Marie's theory that the contractures are due to absence of the pyramidal tracts, which normally exercise an inhibitory influence on the cells in the anterior horns, is not applicable in those cases in which little or no change can be demonstrated in the pyramidal tracts. Mya and Levi[21] believed also that the contractures in Little's disease were entirely of spinal origin, resulting from the predominance of the action of the cells of the anterior horns in agenesia of the pyramidal tracts. They believed that in the newborn the radicular cells of the spinal cord have an autonomous function, independent of the cortical cells. The physiological hypertonicity at this age is accentuated when the motor centres of the cord are not properly connected with the higher centres. Van Gehuchten,[22] however opposed this view, claiming that the question of age is of little importance, as the same spasticity is found in adults in affections of the pyramidal tracts. He believed that the contractures are an expression of an exaggeration of normal muscular tone, due to an interruption of the corticospinal fibers, with persistence of the corticopontocerebellospinal fibers, maintaining the cells of the cord under the influence of the motor cells of the cortex.

It is not difficult to understand that sparseness of fibers, or fineness of fibers, may have the effect of lowering conductibility, and in this way interfere with the inhibitory control normally exercised by the cortex over the spinal centres, the persistent action of the cells of the anterior horns thus giving rise to spastic conditions of the extremities.

One word as to the cause of the agenesia of the pyramidal tracts, the presence of poorly developed, thin fibers, and the sparseness of fibers in the pyramidal tracts, described in some of the cases. That these conditions result from a lack of the usual number of cortical ganglion cells is plausible, although extremely difficult to demonstrate satisfactorily. The view held by Van Gehuchten is also acceptable in this connection: that spasticity is due to the arrest of growth of the fibers in the pyramidal tracts, he having found, in fœtuses of seven months, that the axis cylinders are absent in the cord, but present above the anterior pyramids.

[16] Arch. f. Psych., 1886, xvii, 649.
[17] Arch. d. méd. d'enf., Paris, 1899, ii.
[18] Neurol. Centralbl., 1895, p. 130.
[19] Arch. f. Psych., 1891-92, xxiii, p. 53.
[20] Neurol. Centralbl., 1887, No. 10.
[21] Cited by Oddo.
[22] Revue neurol., 1897, 558.

How can the spasticity be explained when the pyramidal tracts are not degenerated? The presence of fibers in the pyramidal tracts presupposes the presence of intact cells in the cortex, or at least in the cerebrum. The character of the fibers and cells, then, must have some bearing upon the development of this symptom. When fibers of small caliber are called upon to conduct vigorous motor impulses, do we not meet with the same condition as when large currents of electricity are forced through wires of small caliber, that is, increased resistance, and imperfect conduction? The increased resistance offered by the small fibers to the motor impulses may prevent the impulses from reaching the cells in the spinal cord in toto, and thereby there is a cutting off, partial at least, of the influence of the higher inhibitory cortical centres. While, of course, this cannot be demonstrated, it seems a possible explanation of the spasticity in some cases.

The following case is interesting in this connection on account of the absence of gross and microscopic changes in the nervous system to account for the symptoms presented during life:

J. C., aged sixty-four years, was admitted to the Philadelphia Home for Incurables September 28, 1899. His father was killed in an accident, and his mother died as a result of a "broken blood-vessel." Three brothers are dead, two as the result of "kidney trouble;" the cause of death of the third is unknown. When a child the patient had measles, chickenpox, and "spasms." No history could be obtained as to the conditions at birth or during early infancy.

He stated that the present disease began at about five years of age, although he said, on a subsequent occasion, that the spasms in infancy had left him in his present condition. Physical examination showed a spastic condition of both legs, which remained stationary from the time of his admission (September 28, 1899) until his death (May 9, 1906). His gait was that of spastic diplegia, and he assumed a stooping attitude when walking. He assumed a perfectly erect position only with considerable difficulty, and could not maintain it for any length of time. The knee-jerks were much increased and equal. The Babinski phenomenon was marked on both sides, as was also the plantar reflex. There was no clonus present. His grasp in the right hand was good, but was slightly weaker in the left hand. He used his hands readily, and there seemed to be no loss of power or spasticity on either side. He spoke with difficulty, but this was due to stuttering, and there was no evidence of spastic speech. Mentally he was considerably below the average intelligence.

The autopsy was performed a few hours after death; the brain and spinal cord were removed and placed in formalin.

Macroscopically the brain showed an extensive effusion of blood in the pia arachnoid, extending over a large part of both hemi-

spheres, and also at the base. There was no evidence of fracture of the skull or local trauma. Otherwise the macroscopic examination was negative.

Sections of the brain and spinal cord revealed absolutely nothing abnormal macroscopically. Portions of both paracentral lobules and the left frontal region were studied microscopically, as well as the pons and medulla and various portions of the spinal cord. The pia of the cortex showed a sparse cellular infiltration composed of connective tissue cells. The bloodvessels were somewhat thickened, and the cellular elements in the walls somewhat increased.

The cells of the cortex, stained by thionin, revealed no abnormality, although no Betz cells were found. The pons, medulla, and spinal cord, stained by the Weigert method, showed no evidence of degeneration. Sections of the spinal cord stained with hematoxylin and acid fuchsin, showed that the crossed pyramidal tracts on both sides took the stain slightly more intensely than the remaining white matter. Study of the fibers in the crossed pyramidal tracts by this stain demonstrated the presence of a number of small fibers closely packed together, and a sparseness of large well-formed fibers. There was a slight increase in the connective tissue, but at no time did this reach the degree which is seen in spinal cords in which the pyramidal tracts show secondary degeneration. The smallness of the fibers of the crossed pyramidal tracts contrasted clearly with the much larger fibers in the adjoining direct cerebellar tracts. The cross section of the spinal cord itself was normal in size. There were also present in the crossed pyramidal tracts, as well as in the posterior columns at all the levels studied, numerous corpora amylaceæ. The fineness of the nerve fibers was more apparent in the cervical and thoracic regions than in the lumbar region. By the thionin stain the ganglion cells of the anterior horns, at all the levels studied, showed a marked change. While there were present many normal cells, a number of cells were seen which were swollen, some showed marked atrophy of the pigment with excentric nucleus, while most of the cells showed increase in the yellow pigment. At some levels fresh capillary hemorrhages were present in the posterior horns, probably agonal in origin.

Summary. A man, aged seventy-one years, presented spastic paraplegia dating from early childhood. A study of the brain and spinal cord revealed nothing of importance, excepting a fineness of the fibers in the crossed pyramidal tracts.

In my opinion this case antedated the age of five years (as the patient stated), as his intelligence was below normal, and it could not be expected that at the age of sixty-four (when he was admitted) an accurate history of his early infancy could be obtained. The result of the anatomical study makes it possible that this case was one of Little's disease, although, of course, without definite history of the onset, this claim cannot be definitely made. It is certainly

extraordinary that an individual could live to the age of seventy-one years, suffering for certainly the greater part of his life from spastic paraplegia, without its being possible to demonstrate more pathological change in the nervous system than the slight, and perhaps indefinite one which I have described. It seemed to confirm the theory, advanced earlier in this paper, that the fine caliber of the nerve cells offered resistance to the transmission of motor impulses, which, for their perfect conduction, require well-formed and large nerve fibers.

The three cases reported by Spiller are apparently similar to mine. In two of his cases he found practically nothing abnormal in the nervous system except a fineness of the nerve fibers of the crossed pyramidal tracts, and in the third case there was no positive microscopic lesion whatever. He believed there was an agenesia of the pyramidal tracts in the spinal cord in the first two cases cited, while in the third one the explanation offered was that the nerve fibers had not grown downward as far as the lumbar region in normal numbers, although he confessed that this was difficult to prove.

REVIEWS.

THE PRINCIPLES AND PRACTICE OF MEDICINE. By WILLIAM OSLER, M.D., F.R.S., Regius Professor of Medicine in Oxford University, England. Seventh edition; pp. 1143. New York and London: D. Appleton & Co., 1909.

IT is a pleasure to record the publication of a new edition of Osler's *Practice of Medicine*, a book which, in many respects, ever since its original issuance, has not acknowledged a peer. The volume has been notably improved, particularly, as the author points out, in the section relating to the infectious diseases. Herein are incorporated the advances in the etiology of syphilis, the work of the New York Pneumonia Commission, the triumph of the British army and navy surgeons in stamping out Malta fever, the work of Gorgas and his colleagues at Panama in connection with yellow fever, the studies of Strong and his associates in the Philippine Islands in connection with dysentery, recent work in trypanosomiasis, psorosomiasis, tropical splenomegaly, cerebrospinal fever, tuberculosis, epidemic anterior poliomyelitis, Rocky Mountain fever, milk sickness, serum disease, carriers in acute infections, parasitic infections, etc. New sections have been incorporated on diverticulitis, parotitis, pancreatic and adrenal insufficiency, œdema of the lungs, Banti's disease, polycythemia, serum therapy, and the surgical treatment of internal diseases, as well as other matters that need not be mentioned in detail. There is an interesting short paragraph on what the author speaks of as the cult of the day—faith healing—that well repays the reading. The book has already been translated into French and German, and translations into Spanish and Chinese are in preparation.

Assuredly, therefore, in its new edition, the volume maintains and strengthens the commanding position universally accorded it; it remains, as it always has been, an extremely personal book, not only reflecting the personality and knowledge of medicine of its author, but also exemplifying the depth and breadth of his learning aside from medicine; it is a record of illuminating clinical descriptions based upon wide experience and careful and discerning sifting of large numbers of cases; and it is fully abreast of the times, since no important advance has escaped the discriminating pen of its accomplished author. A. K.

A Practical Treatise on Diseases of the Skin, for the Use of Students and Practitioners. By James Nevins Hyde, A.M., M.D., Professor of Dermatology in Rush Medical College, Chicago. Eighth and revised edition, illustrated with 223 engravings and 58 plates in colors and monochrome. Philadelphia and New York: Lea & Febiger, 1909.

The volume in hand is a large, imposing, handsome octavo, numbering over eleven hundred pages, and may be said to cover the field of cutaneous diseases in an exhaustive manner. From the first edition to that now published the growth and improvement have been progressive, a very complete book being the result. A careful perusal of the pages shows that the work of revision has been satisfactorily and conscientiously performed. It is noted that diseases of warm countries and the tropics have been considered in a separate chapter, this grouping, however, bringing together, it need hardly be stated, diseases more or less diverse in nature. New articles have been written on prurigo nodularis, certain forms of erythema, "the fourth disease," paraffin prosthesis, osteoma and calcification of the skin, meralgia paræsthetica, acrodermatitis pustulosa hiemalis, lichen spinulosus, keratolysis exfoliativa congenita, lipoma, Fordyce's disease, causalgia, leukæmia and pseudoleukæmia cutis, tinea ciliorum, and a few other diseases. It may be said that the volume contains such a wealth of material, from whatever standpoint it may be viewed, that it constitutes a most valuable work of reference, to which one may turn with assurance that the subject, no matter how obscure or rare, will be at least touched upon if not fully elaborated. The references throughout are copious and include the literature of many countries, while the illustrations are both numerous and, as a whole, satisfactory. Many of the photographs portray the lesions and other features of the disease admirably (as, for example, "gangosa," pityriasis rubra pilaris, dermatitis venenata, and circinate erythema multiforme), and a number of them, moreover, are new cases. In the chapter devoted to general diagnosis occurs an excellent, concise, and distinctly useful table devised for the investigation of cases, aiding and leading up to diagnosis; and the remarks made on general treatment, internal as well as external, are both sound and to the point. Words of caution and advice are uttered concerning the use of arsenic internally which are worthy of being impressed upon general practitioners, many of whom are too prone to prescribe this drug without the indications existing for its administration. There is no drug that is prescribed more recklessly and with more damage than arsenic for diseases of the skin. The sound views on this subject here and there throughout the book must eventually do much to show that this drug, while of distinct value in some cases, is nevertheless in most diseases to be withheld. The nomenclature in the work is that employed by the most prominent dermatologists and teachers,

although many terms either obsolete or but seldom used, it would seem, might have been relegated to footnotes rather than be made conspicuous in the body of the text. Concerning the volume, so well and favorably known as it has long been, it is unnecessary to say more. It must for many years remain a standard and useful work, and one that the general practitioner especially should possess. As a book for the student, it might perhaps be criticized as being both too voluminous, as well as too elaborate in scope and detail. Author and publisher alike are to be congratulated for such a valuable contribution to literature. L. A. D.

The Principles and Practice of Medicine. By Arthur R. Edwards, A.M., M.D., Professor of the Principles and Practice of Medicine and of Clinical Medicine in the Northwestern University Medical School, Chicago. Second edition; pp. 1257; 121 illustrations. Philadelphia and New York: Lea & Febiger, 1909.

About two years ago, in commenting upon the publication of the first edition of Edwards' *Practice of Medicine*, we took occasion to commend the book to the attention of students, practitioners, and teachers, in the belief that when tried it would not be found wanting, but, on the contrary, always of service. This opinion has found abundant confirmation, which is especially well exemplified in the early demand for a second edition. The book has been really revised, not merely reprinted. As stated by the author, particular attention has been given to therapeutic details, numerous new preparations and modified names and dosages, particularly for children, being explicitly specified. There are practically new chapters on the arrhythmias and other cardiac neuroses, tropical splenomegaly, and various other tropical affections. Due consideration has been given Flexner and Jobling's antimeningococcic serum, Strong's work on amœbic dysentery, blood cultures in typhoid and other bacteremias, the "carriers of infection," epidemic meningitis and poliomyelitis, tuberculosis, the spirochete of syphilis, etc.

Throughout the book, as in the first edition, the causative pathology has been blended with the consecutive clinical features of disease, reasons have been given for facts, exceptions have been subordinated to what is usually found at the bedside, and the allurements of typical clinical pictures and dogmatic generalizations have been avoided, because, as stated by the author, they hold neither in practice nor at the bedside. The volume contains an unusual wealth of well-arranged and well-digested clinical and other facts, perhaps more than may be found in any other book of its kind; much attention has been devoted to diagnosis and differ-

ential diagnosis—which undoubtedly will be appreciated by the harassed busy practitioner; and the details of treatment, based, in large part, upon physiological principles, are unusually full and explicit, enabling the physician to resort to them in practice with assurance of a happy outcome. The book unquestionably is a credit to its author; and in the revised edition is an improvement upon an originally excellent volume. A. K.

Diagnostic Methods. By Ralph W. Webster, M.D., Ph.D., Assistant Professor of Pharmacological Therapeutics and Instructor in Medicine in Rush Medical College, University of Chicago. Pp. 641; 37 colored plates and 164 other illustrations. Philadelphia: P. Blakiston's Son & Co., 1909.

The wide field of work suggested by the title given to Dr. Webster's book has been ably and for the most part thoroughly covered by the author. A glance at the table of contents will show what a systematic and comprehensive compilation one has in his hands. "Compilation" is hardly the correct word, since we learn in the author's preface and throughout the book that many of the methods described have been mentioned by him because of his personal acquaintance with them in his laboratory. Insistence is laid throughout on the macroscopic and unstained examination of a specimen, and on the correlation of the laboratory findings with the clinical aspects of the case. The book comprises laboratory examinations of sputum, of oral, nasal, and conjunctival secretions, of gastric contents, of feces. A special chapter is devoted to parasites, and is admirably illustrated by many good figures. The detail which the author has devoted to his work may be judged of when it is known that 200 pages alone are needed for the urinary examination. The rest of the book describes examinations of secretions of the genital organs, examination of the blood, transudates, and exudates, and secretions of the mammary glands. In a work of this magnitude it is impossible to avoid omissions of minor importance. However, certain well-known tests have been overlooked, or is it because the author has found them valueless? We refer, among others, to Lange's test for acetone, Rivalta's test for differentiating between exudates and transudates, dimethylamidobenzaldehyde test for urobilin, some test for hydrobilirubin in the feces, and Hopkins' method of estimating uric acid. It is, of course, an impossibility to obtain a book dealing with clinical diagnostic methods in which one can find everything he wishes, and for this reason Dr. Webster's sins of omission should not be too severely judged.

Certain tendencies of the author to overload the book with unnecessary facts are shown on page 18, page 45, and page 504. In the first

instance, under the description of the tubercle bacillus, reference is made to the diagnostic use of the tuberculin reaction; in the second instance a histological consideration of the stomach is given, while in the last instance attention is directed to therapeutics and prognosis. A small error is noted on page 49, where an Ewald meal is said to consist of a piece of bread and a cup of tea, and in the next sentence is the statement that this will represent in approximate figures "36 grams of wheat bread and 400 c.c. of water." An ordinary cup is incapable of holding more than 180 c.c. Regret is here expressed that the author's intention to limit the bibliography was adhered to, as some of the descriptions of methods are unclear and too sparse to be of much use. The plates are uniformly good, but exceptions must be taken to three: Plate IV shows no browning of the body of the bacillus as described in the text, while Plates XXI and XXII are so nearly alike that their purpose is not apparent. The above points of criticism will undoubtedly be eliminated in succeeding editions which are sure to follow. The book is to be heartily recommended. E. H. G.

Pain: Its Causation and Diagnostic Significance in Internal Diseases. By Rudolph Schmidt, M.D., Assistant in the Clinic of Hofrath von Neusser in Vienna. Translated and edited by Karl M. Vogel, M.D., Instructor in Pathology, and Hans Zinsser, M.D., Instructor in Bacteriology, in the College of Physicians and Surgeons, Columbia University, New York. Pp. 326. Philadelphia and London: J. B. Lippincott Co., 1908.

The forelying manual professes to be a systematic analysis of pain. Its scope is obvious from the chapter headings, which comprise: the sensation of pain; the functional modification of pain, including the influence of position, motion, pressure, food, drugs, chemicals, and organic function; topography, in its relation to pain; the quality and time occurrence of pain; and painful sensations referable to the nervous system, the organs of locomotion, the digestive system, the urinary system, the respiratory system, and the circulatory system; to which the translators have added a chapter on the results of Head's studies of cutaneous tenderness in visceral disease. It is immediately apparent that to the accomplishment of his task the author has brought not only his long experience, but also a critical mind and a philosophical temperament. The result is a book of much merit and unusual interest, and this, too, in spite of the elusiveness of the ultimate nature of painful sensations and the subjectiveness and psychological aspects of pain as encountered by the physician. The majority of physicians doubtless will read with most intentness the chapter of 125 pages on painful sensations in diseases of the

digestive system, wherein are recounted the varieties and modifications of pain in the gastralgias, gastric ulcer and carcinoma, pyloric spasm, appendicitis, lead colic, intestinal colic, disease of the gall-bladder, etc. Perhaps too much is made of a single subjective symptom, but the author delivers his message rather convincingly. At all events the book well repays the perusal. A. K.

PRACTICAL POINTS IN ANESTHESIA. By FREDERICK-EMIL NEFF, M.D., New York City. Pp. 45.

BLOOD-EXAMINATIONS IN SURGICAL DIAGNOSIS: A PRACTICAL STUDY OF ITS SCOPE AND TECHNIQUE. By IRA S. WILE, M.S., M.D., New York City. Pp. 158; with 35 figures and 2 colored plates.

700 SURGICAL SUGGESTIONS. By WALTER M. BRICKNER, M.D., ELI MOSCHOWITZ, M.D., and HAROLD HAYS, M.D., New York City. Third series; Pp. 150. New York: Surgical Publishing Company, 1909.

DR. NEFF'S *Practical Points in Anesthesia* gives some valuable points in the administration of chloroform and anesthol, the latter being a combination of chloroform, ether, and ethyl chloride. The value of the book would be greatly increased if the practical points in the administration of ether anesthesia were included. From the title of the book one would suppose that they would be given. The routine administration of morphine before all anesthesias is to be generally condemned. The author, however, advises it even before giving chloroform and anesthol. We would suggest that the title of the book be changed to *Practical Points in Chloroform and Anesthol Anesthesia*. The book altogether is a valuable one, and if its reading will make anesthetists, and doctors generally, more careful in the administration of all anesthetics, and particularly chloroform, it will have accomplished a noteworthy object.

Dr. Wile's *Blood Examinations in Surgical Diagnosis* presents in a compact form the technique of blood examinations in general, and treats more particularly of blood changes in surgical and gynecological conditions. The immense literature on hematology has been carefully collected and is presented briefly in reference to its practical importance and value in surgical diagnosis and prognosis. Of special excellence are the chapters on "the blood in obstetric and gynecological conditions," and "blood examinations in surgical operations." The book is very practical, and to the student of general surgery and surgical diagnosis it adds a most valuable chapter to his knowledge of the subject.

700 Surgical Suggestions has reached its third edition, it being first presented in 1906 as *500 Surgical Suggestions*. The text consists

entirely of practical suggestions arranged systematically to the various regions of the body. In scope the work is very complete; and it contains many practical points for the student and practitioner which are difficult to locate in general text-books on surgery. The book may be recommended thoroughly for the purpose the authors had in view in preparing it. J. A. K.

Epoch-making Contributions to Medicine, Surgery, and the Allied Sciences. Collected by C. N. B. Camac, M.D., of New York. Pp. 435. Philadelphia and London: W. B. Saunders Co., 1909.

Dr. Camac has placed us under considerable obligation by collecting in a single volume eight of the important contributions that have significantly influenced medical thought and practice—really epoch-making contributions. These are Antisepsis, by Lord Lister; The Circulation of the Blood, by William Harvey; Percussion of the Chest, by Leopold Auenbrugger; Auscultation and the Stethoscope, by R. T. H. Laënnec; Vaccination against Smallpox, by Edward Jenner; Anesthesia, by William T. G. Morton; and Puerperal Fever, by Oliver Wendell Holmes. Dr. Camac very truthfully points out that we may turn for inspiration and knowledge to these early writings, stating that "in many instances subsequent writers have merely paraphrased the statements of the original observers; indeed, one may go farther and say that some of the errors of today are the result of disregarding or misquoting the facts clearly set forth in these original treatises." The book should be read by every member of the profession, not only by those grown old in the service, but by the youngsters who often need, and assuredly thus will secure, much needed stimulation to careful and painstaking work. A. K.

Progressive Medicine. A Quarterly Digest of Advances, Discoveries, and Improvements in the Medical and Surgical Sciences. Edited by Hobart Amory Hare, M.D., Professor of Materia Medica and Therapeutics in the Jefferson Medical College. Assisted by H. R. M. Landis, M.D., Demonstrator of Clinical Medicine in the Jefferson Medical College, Philadelphia. Vol. III, 1909; pp. 336. Philadelphia and New York: Lea & Febiger, 1909.

Volume III of *Progressive Medicine* for 1909 opens with an instructive account, comprising 106 pages, of diseases of the thorax

and its viscera, including the heart, lungs, and bloodvessels, by William Ewart. Special mention is made of tuberculosis, the respiratory gases and aërotherapeutics, the physical signs and examination of the chest, pleural effusion, common colds, asthma, physical examination of the heart, heart strain and overstrain, arteriosclerosis, the arterial wall and blood-pressure readings, etc. William Gottheil devotes 50 pages to dermatology and syphilis. Edward P. Davis devotes 124 pages to obstetrics, special mention being made of disorders and complications of the pregnant state, the toxemias of pregnancy, placenta prævia, labor and its complications and their management, obstetrical surgery, and the management of the puerperal period. William G. Spiller devotes 49 pages to diseases of the nervous system, noting in particular tumors of the brain, aphasia, apoplexy, meningitis, cerebral syphilis, tabes dorsalis, poliomyelitis, neuritis, epilepsy, etc. The publication continues to merit the professional favor bestowed upon preceding issues.

A. K.

The Principles of Hygiene as Applied to Tropical and Subtropical Climates and the Principles of Personal Hygiene in them as Applied to Europeans. By W J. R. Simpson, M.D., F.R.C.P., D.P.H., formerly Health Officer of Calcutta; Lecturer on Tropical Hygiene at the London School of Tropical Medicine; and Professor of Hygiene, King's College, London. Pp. 396; 98 illustrations. London: John Bale Sons & Daniels Son, 1908.

This book deals essentially with the subject of hygiene as adapted to the requirements of the tropical British possessions. The dangers of the tropics and the differences in conditions there from those of the temperate zone are clearly pointed out. Practical, simple, and efficient advice is given how to escape these dangers and how to meet these differences. The book represents the application of sound, modern hygienic principles to the conditions of tropical countries. Emphasis is laid upon personal and domestic hygiene, while such subjects as the examination of water, communicable diseases, and sewage disposal are adequately treated. Laboratory methods are given in a concise and clear manner, and those that are both practical and useful in the tropics are included. However, there is for the most part a freedom of technical detail, and the clear simple style makes the book of value to a reader not possessing a highly technical training. The paragraphing is well arranged and makes the book suitable for purposes of reference. The many illustrations serve mainly to point out the worse features of the poor sanitation in the tropics, and to give plans for proper sanitary construction, as has been found especially useful in hot climates. The writer has gathered much useful information during his sojourn in the tropics, and the

book contains many practical suggestions. The details of the text seem to be correct and in accord with the latest principles of hygiene. The book should be a valuable possession for either a medical man or a layman visiting a tropical or subtropical country.

G. C. R.

Manual of the Diseases of the Eye for Students and General Practitioners. By Charles H. May, M.D., Attending Ophthalmic Surgeon to the Mt. Sinai Hospital, New York; Consulting Ophthalmologist to the French Hospital, to the Gouverneur Hospital, and to the Italian Hospital, New York. Sixth edition; pp. 600; 362 original illustrations, including 22 plates, with 62 colored figures. New York: William Wood & Co., 1909.

This book has passed through six American editions and the same number of reprints in nine years. Three editions have also appeared in England, two in German, the same number in Italian, Dutch, and Spanish, and a translation also in French and Japanese. This is surely a remarkable history and most convincing evidence that the work has fulfilled the purpose which the author proposed to himself in the preface to the first edition—"to present a concise, practical, and systematic Manual of the Diseases of the Eye, intended for the student and general practitioner of medicine." This sixth edition, while not increased in size, has been brought up to date. Transillumination, the conjunctival tuberculin reaction, decompression, cyclodialysis, exsection of the sympathetic, are among the newer subjects treated. The figures and plates with which the book is richly illustrated are a valuable addition. Some of the colored plates, though generally quite satisfactory, leave something to be desired in fidelity to natural coloring. T. B. S.

The Surgery of the Ear. By Samuel J. Kopetzky, M.D., Attending Otologist to the New York City Children's Hospitals and Schools. Pp. 368; 75 Illustrations. New York: Rebman Company, 1908.

The operative surgery of the ear has advanced so much in recent years that its literature has assumed an enormous bulk. Dr. Kopetzky is entitled to the gratitude of the American profession for the able manner in which he has collated the literature of operative otology and the lucidity with which he describes aural operations. This book presents unmistakable evidence of large experience and good practical work on the part of its author. One of its most useful features is the complete bibliographies which it contains of the various operative procedures. The book is well worthy of a place in the library of any one interested in the subject of otology.

F. R. P.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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Polycystic Disease of the Kidneys.—Coombs (*Quart. Jour. Med.*, 1909, iii, 30) reports a case of cystic disease of the right kidney, absence of the left kidney, multiple hemorrhages, and death from uremia. With a view to determining whether there is any relation between the amount of healthy kidney substance and the onset of symptoms indicating systemic poisoning, such as are associated with chronic nephritis, he has studied the notes of 45 cases of cystic kidney; 32 showed advanced cystic disease, and of these, 18 had fatal symptoms of uremia, while among 12 moderate cases there were 4 such deaths. In 9 others death was due to cardiovascular disease. Circulatory changes due to heightened arterial pressure, œdema, and capillary hemorrhages into the skin or mucous membranes may be present in both cystic kidney and chronic nephritis. There is, thus, clinically, a close analogy between these two conditions which are pathologically marked by destruction of renal tissues.

Rheumatic Arthritis and "Scarlatinal Rheumatism."—Poynton (*Quart. Jour. Med.*, 1909, iii, 15) has traced the after histories of 25 cases of rheumatism in childhood directly associated with scarlet fever, and finds that the clinical symptoms are identical with those of acute rheumatism. One case was fatal from pericarditis, and from it a diplostreptococcus was isolated which showed the same characters as the diplococcus found by Poynton and Paine in acute rheumatism. The portal of entrance in the two diseases is apparently identical, for scarlatinal rheumatism begins either soon after an initial sore throat, or in association with a secondary sore throat. The children who suffered from rheumatism during scarlet fever were liable to relapses, in which

multiple arthritis, chorea, or heart disease might light up again. Nodules, purpura, erythemas, and psoriasis are also found in association with both true and scarlatinal rheumatism. Both diseases are benefited by salicylates. In children, scarlatinal as well as rheumatic arthritis is frequently followed by valvular heart disease. In adults, in both types, the arthritis is a more prominent feature than the cardiac involvement. Poynton is inclined to believe that more thorough bacteriological investigation will show a close relationship between the streptococci isolated in scarlet fever and Micrococcus rheumaticus.

Remissions and Recovery in Tuberculous Meningitis.—Martin (*Brain*, 1909, xxxii, 209) has made a critical study of the reported instances of remissions and of recovery in tuberculous meningitis. Disregarding a number of cases in which the correctness of the diagnosis was probably but not absolutely certain, there were 7 cases in which remissions occurred and in which the clinical condition was proved. In 3 of these instances postmortem examinations showed an old, healed tuberculous meningitis. In 2 the diagnosis was made by the development of tuberculosis in a guinea-pig injected with the spinal fluid. In one the inoculation test was positive, and tubercle bacilli were found in a smear of the spinal fluid. In one case the inoculation test, autopsy, and the presence of a tubercle of the choroid all helped to establish the diagnosis. These patients were all children. The remissions varied from four months to two and one-half years in length. There were 22 cases classed as recoveries in which the diagnosis was certain. In 7 of these tubercle bacilli were demonstrated in the spinal fluid. In 7 cases the inoculation of guinea-pigs was positive. In 3 cases autopsy showed the old tuberculous lesion. In 1 case the bacilli were found in the fluid, and later the old lesion was found at autopsy. In one case both examination of the fluid and inoculation were positive. In three instances the diagnosis was made very certain by the presence of tubercle of the choroid. In none of these cases was there, so far as is known, any recurrence of the meningitis. Martin calls especial attention to the liability of confusion of tuberculous meningitis with "meningism" occurring during the course of infectious diseases and intoxications, and serous meningitis in which there is an exudation of serum into the subarachnoid space, but in which the symptoms are usually less marked. In making the diagnosis of tuberculous meningitis he would rely only on the demonstration or cultivation of tubercle bacilli from the spinal fluid, and on the inoculation test. The presence of a lymphocytosis in the spinal fluid is of less value, for it may occur in other diseases, and some few cases of tuberculous meningitis may show a polynuclear leukocytosis. It is important that a healed lesion of the meninges may form a starting point of a fresh, fatal infection. The treatment in the cases which recovered was so varied that it offers no direct indications.

A Bacteriological Study of Poliomyelitis.—Potpeschnigg (*Wien. klin. Woch.*, 1909, xxi, 1334) reports a bacteriological study of 14 cases of recent poliomyelitis. Particular attention was directed to the cerebrospinal fluid obtained by lumbar puncture. In all of the cases

there was constantly present a Gram-positive coccus, usually arranged as a diplococcus, at times as a tetracoccus. The organism was present in small numbers in the fresh fluid. Bouillon tubes to which 1 to 2 c.c. of fluid was added showed a luxuriant growth after one to three days in the incubator. Round, yellowish-white colonies developed on agar plates which were inoculated with the spinal fluid. In these colonies the Gram-positive coccus was present in pure culture. Only once did the organism grow on blood serum. Whether this organism is identical with the others which have been described, whether it is the cause of poliomyelitis, and what its relations are to the meningococcus have not been determined. In one case of poliomyelitis the same diplococcus was cultivated from the blood one day after the onset. A more detailed account of this microörganism will appear later.

The Prognosis and Treatment of Pellagra.—In a general review of the subject, Lavinder (*Public Health Reports*, 1909, xxiv, No. 37) concludes that the prognosis in all cases is grave as to final and complete recovery. The average mortality, founded on asylum reports is 67 per cent. He believes that the earlier the diagnosis is made and treatment begun, the better the outlook. The chronic type of the disease is the more hopeful. Acute manifestations, fever, mental involvement, nervous disturbances, moist, extensive erythemas, progressive emaciation, especially if accompanied by inveterate diarrhœa, all increase the gravity of the prognosis. The same is true of such complications as malaria, bronchitis, pneumonia, nephritis, intestinal parasites, etc. In considering treatment the most important factor is prophylaxis, and here general hygiene plays the major part. He believes that good corn is wholesome, but points out the difficulty in differentiating it from harmful corn, and urges that it be admitted into the dietary of institutions, such as insane asylums, only with the utmost caution. Among drugs arsenic has given the best results. The newer preparations, atoxyl, first recommended by Babes, arsacitin, and soamin, have been tried, but Lavinder inclines to favor the use of Fowler's solution. He states that the combination of atoxyl with arsenic trioxide, which has been recently advocated by Babes as giving brilliant results, has not proved efficacious in the cases observed by himself.

Tubercle Bacilli in the Blood.—Schnitter (*Deut. med. Woch.*, 1909, xxxv, 1566) has examined the blood of tuberculous patients for tubercle bacilli. By means of animal inoculation it has been amply demonstrated that the blood and organs of patients dead of pulmonary tuberculosis or general miliary tuberculosis may, and usually do, contain tubercle bacilli. Schnitter has used the method proposed by Staubli, in which the blood is withdrawn from a vein (1 to 2 c.c.) and immediately placed into ten to fifteen volumes of 3 per cent. acetic acid. The whole is centrifugalized, and smears are made of the sediment and stained in the usual way. Of 34 cases of pulmonary tuberculosis examined in this way, 10 showed tubercle bacilli in the blood. Eight of the positive cases represented severe, advanced stages of the disease, while 2 positive results were obtained in patients in the second stage (according to Gerhardt-Turban). In one case of tuberculosis of the testicles and

bladder, tubercle bacilli were demonstrated in the blood; the lungs gave no evidence of an active process in this case. The diagnostic value of this procedure is largely the aid it offers in the differential diagnosis of general miliary tuberculosis, typhoid fever, and sepsis, as was well proved by one of the author's cases. (After the conclusion of his work the author found certain improvements in technique which appear not to have been thoroughly tested.)

Experimental Parotitis.—Herb (*Arch. Int. Med.*, 1909, iv, 201) isolated a diplococcus from the lung, testicle, cerebrospinal and pericardial fluids, bile, and parotid gland of a man who died having mumps. The organism is similar to that which has been previously isolated from the secretion of Steno's duct, and from the blood in cases of acute parotitis. The organism grows rather slowly on ordinary media, but more abundantly on a mixture of agar with saliva. A monkey inoculated through Steno's duct with a forty-eight-hour culture developed fever and an acute parotitis lasting six days. A similar reaction was observed in dogs. The pathological lesion was studied at various stages and consists in a non-suppurative parotitis, the infiltration being composed largely of mononuclear cells.

The Occurrence of Altmann's Granules in the White Blood Cells.—In hematology there is much confusion as the relationship of the large lymphocyte to the non-granular antecedents of the myelocytes (myeloblast). Schridde and Naegeli, in particular, have laid great stress on the presence of perinuclear, fuchsinophile granules in the lymphoblast and their absence in the myeloblast, and have sought to differentiate the two cells by the presence or absence of these granules, first demonstrated by Altmann. The staining technique is difficult and in most hands has given unsatisfactory results. Butterfield, Heineke, and Meyer (*Folia Hemitolog.*, 1909, viii, 325) have simplified the technique and have used it in the study of these cells, not only in the bone marrow, but also in the blood in health and disease. As a result of their work they find that Altmann's granules are present not only in the lymphoblast but also in the myeloblast, and they conclude that this method of differentiation is, therefore, valueless.

The Gaseous Exchange in the Lungs in Erythrocytosis.—Senator (*Zeit. f. klin. Med.*, 1909, lxvi, 349) has studied the further cases of erythrocytosis, bringing the total number of cases of this disease in which the gaseous exchange in the lungs has been determined up to 8. The reports in the literature show that definite abnormalities were noted in all. The volume of the respired air is increased quite constantly and with this increase there is an augmented output of carbon dioxide and a similar change in the consumption of oxygen. In a third case of erythrocytosis which recently came under Senator's observation the gaseous exchange which was high during the earlier examinations fell to normal in the course of a few weeks. Whether this was due to oxygen inhalations or not remains undetermined. Senator has examined other cases with polycythemia which lacked splenomegaly and were not characteristic of Vaquez's disease, and in none of these did the gaseous exchange of the lungs depart from the

normal. In the absence of polycythemia one finds an increased gaseous exchange during the digestion of a hearty meal, without a coincident increase in the volume of the respired air. Similarly, Senator finds that artificially induced hyperemia of the lungs (by the Saugmaske) leads to an augmentation of the carbon dioxide and oxygen. In one of the latter cases the results were quite unexpected, since the patient suffered from chlorosis (the hemoglobin being 27 per cent.). No increase in the volume of the respired air occurred. Senator believes that the increase of the gaseous exchange in Vaquez's disease cannot be attributed to any defect in the hemoglobin, since in many cases this has been found to be normal, and also because a decrease of the oxygen capacity of the blood could not explain the increased gaseous exchange, although it might account for the polycythemia and the activity of the bone marrow. Senator finds a striking similarity in the gaseous exchange of healthy individuals during digestion and in patients with erythrocytosis examined while fasting.

SURGERY.

UNDER THE CHARGE OF

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Rectal Anesthesia.—Legueu and Verliac (*Archiv. gén. de chir.*, 1909, lv, 551 to 661) describe a method of administering ether per rectum, and say that the essential points to be observed in order to avoid accidents are: (1) During the administration the ether should be kept below the boiling point by immersing the flask containing it in a wet bath at 33°. (2) The anesthetic vapor should be introduced slowly, in order that the normal intestinal gas should be admixed with a progressively increasing quantity of the ether, and that the anesthetic should not be substituted for the intestinal gas. All the ether introduced should be charged with air. When the operation is completed, the rectal tube should be disconnected and as much as possible of the ether expelled with the aid of abdominal massage. The technique of rectal anesthesia is far from being perfect at the present time. The employment of bromide of ethyl as a preliminary inhalation, of ether not heated, and of oxygen as a vehicle, has permitted the avoidance of the greater number of the accidents imputed to the older technique. Prudently conducted, rectal narcosis is not more dangerous than narcosis by inhalation. It has some contraindications (bad or doubtful conditions of the intestine, acute or chronic affections of the abdomen, operations upon the perineum or genital

organs). It does not offer any advantages over the other methods of anesthesia in operations on the extremities, but it does offer some indisputable advantages in operations upon the face, the buccal cavity, the neck (the field of operation being left free to the operator who is not disturbed or contaminated by the anesthetic), and upon the thorax (condensed ether vapors are injurious to the lungs). It is a method to be used only exceptionally.

Gunshot Wounds of the Kidneys.—CLEMENT (*Ann. d mal. d. org. gén.-urin.*, 1909, ii, 1281) says that the tampon should be employed in those cases in which there are no immediate signs of injury of the kidney or in which these signs come on slowly. In these cases we may consider that the lesions are benign and the hemostasis due to spontaneous clotting. Bleeding may, however, occur later in these cases, from the withdrawal of the tampon or the separation of a slough. Of 6 cases of nephrectomy for gun-shot wounds of the kidney, 3 recovered and 3 died. Anuria is due in the greater number of cases to a reflex inhibition, produced by the trauma of the injured kidney upon its fellow, and signifies only an important lesion of the renal parenchyma. The co-existence of a wound of entrance in the lumbar region is an indication for an exploratory incision. In two of the fatal nephrectomies, one case was complicated by grave stomach lesions, the other by similar lesions and by a wound of the pancreas, all complications which could have produced a fatal termination. In the 3 cases which recovered, the signs were distinct enough to permit early diagnosis and operation, before the hemorrhage had become serious or infection had developed. Early operation is very important. There is less danger from too early operation than from not operating early enough.

Cure of Prolapse of the Rectum Obtained by Tampon.—SICK (*Zentralbl. f. Chir.*, 1909, xxxvi, 1225) says that Ekehorn has described a simple but rational operative method of treatment for prolapse of the rectum in children. This consists in passing a needle carrying a silk suture alongside the sacrum into the lumen of the rectum and under the guidance of the index finger of the other hand, bringing it out of the anus. It is then passed backward from the inside of the rectum outward alongside the sacrum. By this suture the rectum and the included connective tissue in the sacrococcygeal concavity are securely fixed against the bone. The suture is removed in about two weeks, the resulting cicatricial tissue making the fixation permanent, Ekehorn secured good healing in 4 cases in children. Previous to the publication of Ekehorn's work, Sick had tried, with good success, to obtain the same result by simply tamponing the retrorectal space. A longitudinal incision is made in the raphé between the end of the coccyx and the circular fibers of the sphincter ani muscle, where there are no vessels, nerves, or muscular fibers to be injured. The superficial fascia and the deep pelvic fascia are divided, and the loose connective tissue behind the rectum exposed. The rectum is then freed on its posterior wall by a suitable instrument, as high as the promontory of the sacrum. In the cavity thus made, a strip of iodoform gauze of four to six thicknesses is laid, and the small external wound protected from the anus

by an adhesive plaster or collodion dressing. This method is applicable to those cases which recur after the usual treatment by recumbency in bed and keeping the buttocks together by adhesive plaster. It is simple and less dangerous than Ekehorn's suspension method, and gives more promise of a permanent cure, because of the greater cicatricial adhesion developed.

The Operative Reduction of Old Dislocations of the Elbow.—Dollinger (*Deut. Zeit. f. Chir.*, 1909, c, 38) says that he has treated thirty-four old dislocations of the elbow. In the first cases he attempted reduction without operation. In no case did he succeed when the dislocation was more than three weeks old. The operations which followed showed such anatomical changes as to render reduction impossible without exposure of the body ends of the joint. He now operates on any case that is more than three weeks old. In the beginning he tried to preserve the lateral ligaments intact. The joint was exposed externally, the trochlea and capitellum were freed from the surrounding tissues, and the reduction attempted by hyperextension, pulling, and flexion. Of thirteen cases in which this method was tried, only three were successful. In the remaining cases it was necessary to detach the lateral ligament from the epicondyle or to detach the epicondyle with the ligament. In all cases the ligament had become shortened. The force necessary to reduce the dislocation, without division of the external ligament, caused injury to the cartilaginous ends, and this influenced the function later. These considerations led to the adoption of the following method: The joint is exposed by an incision, 12 to 15 cm. long, extending on the outer side of the arm along the lateral intermuscular septum to the epicondyle, which is detached with the lateral ligament by a chisel. The ends of the bones are freed from the surrounding tissues only so far as is necessary to permit the bending inward of the forearm until it lies alongside the arm, when the dislocated joint ends can be pushed together out of the wound and freely inspected. If it develops that the bone ends have undergone such changes that physiological function is impossible after reduction, a resection is done. Of the 34 cases, in 14 resection was considered necessary and 20 were considered suitable for reduction. Even in those cases which were reduced, the articular fossa of the olecranon was filled with fat, remains of capsule, and detached fragments of bone, all of which had become bound together by cicatricial tissue. All these tissues were separated and removed, the joint cartilage being preserved. When the bones are thus prepared the reduction is accomplished not by a pull, but after stretching the internal ligament distalward until the inner articulating surface of the olecranon is brought against the inner articulating surface of the trochlea. The forearm is then bent outward from its position alongside the arm until the joint surfaces are in their normal relations. The elbow is then flexed to a right angle and the detached external lateral ligament re-attached by suture in its normal place, or, if necessary from shortening of the ligament, somewhat lower. The wound is then closed. Of the 20 cases in which reduction was accomplished, the end results are known in 12. In 2 the joint is completely ankylosed, and in 1 there is slight

motion. These 3 cases were complicated dislocations, and were more suitable for resections than reductions. The remaining cases have movable joints. 2 of them have complete flexion, and extension to 135 degrees. In the 14 cases in which resection was done, only the trochlea and capitellum were removed. The epicondyles were preserved and the lateral ligaments re-attached by suture to their normal points of insertion. Dollinger has reported concerning the later function in 11 cases. In 6 the elbows are stiff, in 5 movable. One patient has complete movement and two have motion of about 85 degrees, and two of from 40 to 50 degrees. These data show the functional end results to be better after reduction than after resection, and they will be better in the future from the employment of the method described here. It follows, therefore, that resection is indicated only when the joint surfaces have undergone such changes that normal function after reduction is impossible.

Treatment of Congenital Dislocations of the Hip.—Küster (*Deut. Zeit. f. Chir.*, 1909. c, 52) reports 10 cases of congenital dislocation of the hip in which reduction was made, one by operation according to Hoffa's method, and 9 by the non-operative method. The steps of the non-operative method as carried out by Küster are as follows: Extension is applied to the limb for a week to stretch the tissues and to bring the femoral head nearer to the acetabulum. Under complete anesthesia the pelvis is held firmly by the hands of an assistant against a firm support underneath. Then by pulling upon the thigh and pressure upon the trochanter the head is brought downward, and if this fails the thigh is brought slowly into extreme abduction. At the same time with the closed fist an assistant presses strongly upon the neck and head of the femur from above. All this is done gradually and without undue roughness. Finally, internal rotation of the femur is made slowly and carefully, at times also external rotation. This causes the head to be reduced with a snapping sensation, or it stands so firmly in place that it does not go back into the dislocated position. If a double dislocation exists, the other side is treated in the same way. The thigh is then fixed in the abducted position by a plaster bandage dressing, the legs below the knee not being included in the plaster. Should the *x*-rays show that the head has again become dislocated, the efforts at reduction should be repeated in from ten to fourteen days. The earlier the age at which the dislocation is reduced the more nearly normal are the joint surfaces. The early use of the *x*-rays is very valuable in making the diagnosis and in disclosing the nature of the joint conditions present. One should not become discouraged if a re-dislocation takes place after the efforts at reduction have been made. After repeated attempts a final success may be expected.

Myotomy and Myorrhaphy.—Bardenheuer (*Deut. Zeit. f. Chir.*, 1909, c, 63) says that lengthening of the muscles which have undergone fibrous degeneration (flexors), combined with shortening of the extensors, gives a better result than the Mikulicz resection of the forearm bones, or the plastic lengthening of the flexors as done by Schramm. In cases of infantile, spastic hemiplegia, the division of the markedly spastically contracted muscles (supplied by the median nerve chiefly),

together with the shortening of the tendons of the simultaneously spastically contracted but weaker muscles (supplied by the musculocutaneous nerve), is more effective than the plastic operation of Hoffa and probably more effective than the Spitz grafting of a portion of the median nerve into the musculocutaneous. In very extensive resections of joints, in which, for instance, at the shoulder, almost half of the humerus has been sacrificed, the muscles surrounding the joint may be removed, so that the defect in the muscles will correspond to that in the bone. This operation would exclude the formation of an extensive wound cavity, would bring the resected surfaces in contact with each other, would prevent the development of a flail joint, secure a movable joint, and render many amputations unnecessary. For the present, Bardenheuer prefers the Lexer transplantation of joints, because of the excellent idea involved and the good results obtained in these operations. The muscle resections may be employed also with resections of the bone in continuity.

A Case of Habitual Dislocation of the Patella.—Bunts (*Surg., Gyn., and Obstet.*, 1909, lx, 117) reports a case of exaggerated double external dislocation of the patella of an exaggerated type, in which the following operation was done: A linear incision, six inches long, was made on the inner side of the knee, through the skin and subcutaneous tissue, down to the capsule of the joint. A curved incision was then made through the capsule. The cut edges of capsule were overlapped by pulling the inferior margin under the superior margin by mattress sutures, while the patella was shoved forcibly inward. The free edge was then sutured to the lower portion, and the wound closed without drainage. Sterile dressings and a plaster bandage were applied and allowed to remain on for four weeks. Healing by first intention occurred in both knees. The final results have been only partially successful. The patient is able to walk without crutches. The left knee has remained cured up to this time, but the patient has had several attacks of pain in the right knee. The patella is not thrown out of place, but the symptoms are those of a floating cartilage, a condition that is sometimes associated with repeated attacks of dislocation.

Concerning the Search for Perforated Gastric Ulcers.—Ewald (*Zentralbl. f. Chir.*, 1909, xxxvi, 1281) calls attention to the great difficulty sometimes experienced in locating the small openings made by a perforated ulcer of the stomach, when the abdominal walls are tense, the patient in shock, and the small space in which the perforation lies covered with turbid, thin stomach fluid. He says that the perforation occurs almost without exception about 1 to 2 cm. above the pylorus, or the same distance below, and that the opening is usually exposed as soon as the overlying liver is slightly elevated. The whole area in which it may be found is rarely larger than about 4 cm. Ewald has so found it in all cases operated on in the last year. Lieblein found in 223 perforations, only 5 in the middle of the stomach and 12 in the neighborhood of the cardia. The duodenal ulcers perforate much more frequently in the upper transverse portion of the duodenum, and usually close to the pyloric ring. In many men with a sharp pre-

cordial angle, a low lying umbilicus, and flat or sunken abdominal wall, the pylorus will be made visible with difficulty on account of the falciform ligament of the liver. In stout men with emphysematous chests, the pylorus is so far from the epigastric line that the poorly movable pylorus is brought out and the ulcer exposed with great difficulty. For these reasons Ewald now makes the abdominal incision not in the median line, but parallel to it and through the right rectus muscle. The falciform ligament of the liver is then pushed toward the median line. The abdomen is opened first in the lower angle of the incision, the finger is introduced and pushes the falciform ligament toward the median line, and the peritoneum is divided upward. The edge of the liver is lifted upward, when one usually comes directly upon the perforation. The exudate clinging to the pylorus and first portion of the duodenum is then cleared away, and the infiltrated fatty appendages are removed. If the perforation has not been exposed by this time, the posterior surface of the pylorus is made accessible. Only in rare instances will this be necessary.

Direct Blood Transfusion by Means of Paraffin-coated Glass Tubes.—Brewer and Leggett (*Surg., Gyn., and Obstet.*, 1909, lx, 293) record the results of some experiments undertaken to find some simple method of direct blood transfusion. End-to-end suture as practised by Carrel and union by the ingenious cannula of Crile have been the methods generally employed in the past. A set of glass tubes was made, some straight with uniform caliber, others bayonet shaped, and still others somewhat tapering in shape and caliber, to be used for transfusion from a large adult artery to the small vein of a child. Each end of the tube is notched, to allow a ligature being applied after the tube is introduced into the lumen of the vessel The tube is sterzilied in an autoclave or by boiling, dropped into boiling paraffin, and the excess of paraffin removed by shaking the tube in the air. The following method of transfusion, as described for No. 1, was performed in all the experiments: The external jugular was exposed in dog No. 1, and the common common carotid in dog No. 2. The proximal extremity of the external jugular of dog No. 1 was temporarily clamped and the vein divided, allowing copious hemorrhage from the distal extremity. When the animal began to show signs of weakness and falling pulse, the distal extremity of the vein was ligated. The two dogs were then placed side by side, the carotid artery of dog No. 2 being double clamped and divided between the two constricted areas. The lumen of the proximal portion of the artery was then expanded by three mosquito forceps, and one extremity of the glass tube introduced and held by a silk ligature placed around the vessel in the notch of the tube. The orifice of the proximal portion of the jugular vein was next expanded in the same manner by means of three mosquito forceps. Before introducing the distal end of the glass tube, the arterial clamp was temporarily released and a few jets of bright arterial blood allowed to pass from the tube. It was then quickly introduced into the lumen of the vein and secured by a second silk ligature. As soon as the temporary clamps were removed, blood could be seen to pass from the artery of the donor to the vein of the donee, causing distinct pulsations in the vein, corre-

sponding to the arterial pulse of the donor. Dog No. 1 was quickly revived by the transfusion, which lasted about six minutes. The vessels were then ligated and the wounds closed and dressed. In thirty-one experiments, only three animals died, each from a too rapid transfusion of the blood, causing overdistention of the right heart. On one occasion only did the blood apparently clot in the tube, and that was undoubtedly due to the very small caliber of the tube used. In one other experiment clotting occurred in the vein near the extremity of the tube, due to an accidental separation of the paraffin coating. The writers believe that this is the simplest method yet proposed, that it can be carried out by any surgeon of ordinary experience without the necessity of previous training. It has been employed in one patient suffering from anemia due to gastric hemorrhage. The blood flowed for nineteen minutes through a medium-sized tube without clotting. The patient rallied well. His hemoglobin rose from 30 to 70 per cent. during the progress of the operation, but the patient died the following day from a recurrence of the hematemesis.

THERAPEUTICS.

UNDER THE CHARGE OF

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The Treatment of Erysipelas by Means of Carbolic Acid and Alcohol.—Judd (*Medical Record*, 1909, vii, 268) reports 82 cases of erysipelas treated by this method during the past eight years. His statistics show 67 complete remissions of symptoms in from twelve hours to four days. There were ten delayed recoveries, and the treatment failed in five of the cases. The technique of the method consists of painting with a swab of cotton the entire surface of the involved area, and extending about half an inch into the surrounding and apparently healthy skin, with a 95 per cent. solution of carbolic acid. This is left until the purplish color of the inflamed area is replaced by a pretty complete whitening of the skin. When large areas are involved, it is advisable that only a portion be painted at a time. The second step consists in going over the whitened areas very thoroughly with a second swab saturated with pure alcohol. The swabbing with the alcohol must be kept up until the whitened area becomes pink. Half an inch of the surrounding sound skin must be included, as the bacteria of erysipelas are found beyond the apparently involved area. This method includes the painting of the hairy scalp, the eyelids, the mucous membrane of the alæ of the nose, and the nipple of the breast, if necessary. Judd has failed to note any bad results from this treatment. He says that there has been no marked toxic action of the carbolic in any case so far observed, although the urine is sometimes darkened and of a characteristic odor. Almost the first result noticed by the patient is a complete cessation of the unendurable itching, burn-

ing, and throbbing. Usually within a few hours nausea, if present, subsides, and within twenty-four hours the temperature sinks to normal. The pulse falls very rapidly from 120 or more to nearly normal, the appetite returns, and, except for the swelling, which remains for one or two days longer, the patient is relieved from his distressing symptoms. Judd says that the most of his failures were in young children, and these cases also showed the greatest number of symptoms of absorption of carbolic acid. He says, furthermore, that this treatment has no permanent injurious action upon delicate skins.

The Action of Potassium Bitartrate.—Burwinkel (*Med. Klin.*, 1909, xvii, 627) confirms the statement of Eichhorst regarding the diuretic action of potassium bitartrate. He advises it especially in cases of ascites as a result of cirrhosis of the liver. He gives 4 to 5 grams three times a day. In anasarca from failure of compensation in valvular disease, he advises a combination of powdered digitalis and potassium bitartrate.

Chloroform—The Ideal Hemostatic in Pulmonary Hemorrhage.—Fish (*Jour. Amer. Med. Assoc.*, 1909, lii, 1918) reports excellent results from the use of chloroform in 19 cases of pulmonary hemorrhage. Fish states that the routine treatment of hemoptysis has always been uncertain and empirical because of the inaccessibility of the bleeding point. He explains the action of chloroform by a lessening of the heart action, a reduction of the blood pressure, and a diminution of the respiratory movement. Since chloroform produces coagulation of the blood in vitro, he says that it is possible that its hemostatic action may be aided by direct contact of the vapor with the bleeding point. He gives from 2 to 4 c.c. of chloroform by inhalation. Results should occur within five or ten minutes. Subsequently the inhalation of 15 to 20 drops every hour is continued for a few days. At the same time ammonium chloride and small doses of codeine are given. Fish also advises the administration of a teaspoonful of magnesium sulphate three times daily to remove excrementitious matter which, if retained in the blood, would stimulate the vasomotor centre and raise the blood pressure.

The Vaccine Therapy of Enteric Fever.—Semple (*Lancet*, 1909, ii, 1668) concludes from his results with the use of bacterial vaccines in enteric fever that the administration of such vaccine is a practical method of increasing the bacteriotrophic substances in the blood, as evidenced by an increase in the opsonic index of those treated by this method. When appropriate doses are given the method is devoid of all risk, is easily carried out, produces no apparent disturbance in the patient's condition, and does not interfere with any other treatment the physician may deem necessary. It would not be possible to generalize from the few cases which he cites as to whether vaccine treatment has a marked effect in cutting short the fever period. In 6 of the 9 cases treated, well-marked improvement set in after the vaccinations. This improvement was especially marked when autogenous vaccines were administered. In the remaining 3 cases there was an amelioration of the clinical symptoms.

The Favorable Effect of Extract of Cannabis Indica Butyricus in Exophthalmic Goitre.—Crämer (*Klin.-ther. Woch.*, 1909, xxiv, 590) calls attention to the recommendation of Sée for the use of the fatty extracts of cannabis indica in the treatment of functional disturbances of the digestive tract and heart. Sée considers that this drug is a very valuable sedative for the gastro-intestinal tract, with the advantage over morphine of not causing nausea or vomiting. Sée also found that it was useful in palpitation of the heart and other heart symptoms due to digestive disturbances. Acting upon Sée's suggestion, Crämer has tried this remedy in a number of patients having gastric symptoms. He found that the best effects were obtained in functional disorders. The symptom of pain was especially benefited. With these results in mind, Crämer was led to try its effects in the treatment of exophthalmic goitre. He relates the histories of several cases so treated, and advocates its use in this condition. Palpitation and tachycardia were improved, the patients began to eat, and they increased in weight and strength. The thyroid diminished in size, together with a diminution of the exophthalmos. Crämer believes that the operative treatment of exophthalmic goitre should only be advocated when all of the various medical procedures fail.

Vasodilators in High Blood Pressure.—Matthew (*Quart. Jour. Med.*, 1909, ii, 261) states that it has to be recognized that high blood pressure is not necessarily harmful to the individual. In some cases it is to be considered as purely compensatory; therefore, not every case of high blood pressure should be treated with vasodilators. Undoubtedly there is a tendency for the blood pressure to increase progressively, and our aim should be to prevent this increase from being too sudden and exaggerated. Matthew has studied the action of the vasodilators in patients with hypertension by means of the sphygmomanometer. Nitroglycerin, potassium and sodium nitrites, erythrol tetranitrate, and mannitol hexanitrate all showed a powerful vasodilator action, while cobalto-nitrate of potassium proved inert in this regard. Not all cases of hypertension however, responded with a fall in blood pressure. The nitrites produced little or no vasodilator action in certain cases. Moreover, a rise in blood pressure occurred in some cases of advanced Bright's disease. In heart and kidney disease when there is marked œdema nitrites do not act well. After the œdema has disappeared the usual vasodilator action may reappear. He made observations in many cases of hypertension when symptoms had developed, such as pain, headache, giddiness, epistaxis, etc. He invariably found that such symptoms were alleviated or disappeared entirely with a reduction of pressure amounting to about 30 mm. Hg. Furthermore, he observed that, if this fall could be maintained the symptoms did not reappear, and the patient's general condition improved. Matthew then determined the dose of various vasodilators that would produce and maintain such a reduction. Liquor trinitrine (nitroglycerin), in the dose of 2 minims, lowered the blood pressure 20 to 30 mm. Hg. very temporarily. Following this transient lowering there was an almost immediate gradual rise of the pressure, and in all cases the effect of the drug passed off in thirty minutes. Sodium or potassium

nitrite in doses of 2 grains produced a reduction of just over 30 mm. Hg. This action will last two hours and only after this is it necessary to repeat the dose. No benefit is obtained by increasing the dose, and a less dose will not give the desired result. Erythrol nitrate in the dosage of 0.5 to 1 grain will produce the beneficial reduction, and the effect will last about six hours. Matthew found that erythrol nitrate sometimes caused unpleasant symptoms, and recommends the smaller initial dose as safer. Mannitol nitrate acts similarly to erythrol nitrate in doses of 1 grain. Its maximum effect is attained more slowly than erythrol nitrate, and therefore it is probably safer. Matthew concludes by saying that the useful and suitable dose of a nitrite for each individual can readily be ascertained by noting the effect of the nitrite as to the amount of fall produced and the time the action lasts.

The Absence of Mucus from the Stomach.—Schalij (*Arch. d. mal. d. l'appar. digest.*, 1909, iii, 79) says that the absence of mucus in the gastric secretion aggravates the prognosis of hyperchlorhydria and of ulcer of the stomach. He believes that the mucus has a protective action against the corrosive action of the gastric juice. Schalij, consequently, advocates the use of various bland protective substances to replace the mucus. Among these he mentions olive oil, almond oil, and gelatin. He gives the olive oil or almond oil before meals, starting with 1 c.c. and increasing gradually to 15 c.c. He also advises the ingestion of fatty foods before meals. This method, Schalij says, has the further advantage of decreasing the hyperacidity.

The Treatment of Anemia in Infancy with Citrate of Iron Administered Subcutaneously.—Morse (*Jour. Amer. Med. Assoc.*, 1909, liii, 107) says that the percentage of hemoglobin in infancy gradually rises from 55 per cent. to 60 per cent. at the end of the first month to 70 per cent. at six months, where it remains during the rest of infancy. The number of red blood corpuscles in infancy varies between 5,500,000 and 6,000,000. Morse believes that this comparative deficiency in hemoglobin is due to the fact that the infant normally receives an insufficient supply of iron in its food and that the reserve of iron present in the liver at birth is not large enough to keep the percentage of hemoglobin at the adult standard. He is of the opinion that the anemias of infancy are usually of a chlorotic type, although probably not a true chlorosis. Therefore, Morse thinks that iron is especially indicated in the anemias of infancy. Because of the difficulty of giving iron by mouth to infants, and since iron is very apt to disturb the digestion, he advocates the subcutaneous administration of iron. For this purpose he has found an aqueous solution of the citrate of iron very serviceable. This can be put up in perles and sterilized, each perle containing a single dose. This solution is absolutely non-irritating, and the injections are never followed by induration or abscess. The injection, however, is sometimes followed by pain lasting from a few minutes to an hour. Morse gives the injection by means of a glass syringe with asbestos packing, fitted with a platinum needle. An ordinary steel needle is corroded by the solution. He gives an average dose of $\frac{3}{4}$ grain every other day. He says that he has used the citrate of iron in

this way in a number of cases—some of the chlorotic type, some mild cases not of this type, and some cases of severe anemia with splenic tumor—with quite satisfactory results, even in the severe cases. In most of the cases the rise in the percentage of hemoglobin has been more rapid than that in the number of red blood corpuscles. Morse gives the detailed histories of five of his cases, and concludes by saying that he believes that the subcutaneous use of iron is often most advantageous. The results are more marked and are obtained more rapidly than when the iron is given by the mouth, and there is much less danger of upsetting the digestion.

Intravenous Suprarenal Preparations in Collapse.—Sohn (*Münch. med. Woch.*, 1909, xxiv, 1221) recommends highly the use of suprarenal preparations in cases of acute cardiovascular collapse. Their action is prompt, and their use may often tide the patient past the danger point. He reports seven cases in which he has had good results from their use. In one of the cases he failed to follow up the remarkable benefit from a single injection, and ascribes the fatal outcome to his failure to continue the injections.

Pneumococcic Vaccine in Pneumonia.—Harris (*Brit. Med. Jour.*, 1909, ii, 1530), with regard to the treatment of acute lobar pneumonia by inoculation, concludes that: (1) Successful inoculation for pneumonia is possible; (2) the inoculation does no harm; (3) a vaccine from one or a number of virulent strains should be used; (4) it should be introduced as early as possible; (5) the estimation of the opsonic index is not necessary; (6) the estimation of the observation of the temperature and physical signs is in pneumonia a sufficient guide in gauging the repetition of the dose. Harris believes that infections of the lungs by the pneumococcus which fail to resolve after an acute pneumonia, as well as pneumococcic infections of other areas, ought certainly to be treated with a pneumococcic vaccine. He adds that these cases appear to afford a reasonable prospect of success.

The Treatment of Epilepsy with Calcium Hypophosphite.—Cicarelli (*Il. Polyclin.*, 1909, xvi, 1) records excellent results from the use of calcium hypophosphite in the treatment of a series of 25 cases of epilepsy. He was led to try this method because of the favorable results reported by other Italian physicians in the treatment of epilepsy with lime salts. Cicarelli believes that the phosphorus contained in the calcium hypophosphite has an additional stimulant action upon the nervous tissue of the epileptics. He gives the calcium hypophosphite in doses of from 10 to 15 grains three times a day. Cicarelli advocates this method of treatment either alone or in combination with the bromide method. When given in combination with bromides, the bromides should be given for one week alternating with calcium hypophosphite over a period of two weeks. He considers that the calcium salts are preferable to the alkaline bromides, as bromidism is avoided. In his series of cases patients on calcium hypophosphites were less depressed and their general conditions was better than that of the patients on bromides.

PEDIATRICS.

UNDER THE CHARGE OF
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Spina Bifida Occulta.—Spina bifida occulta is a congenital deformity of the spine characterized by a more or less evident vertebral cleft extending over one or more vertebræ, and without the existence of a tumor. In the dorsal and lumbar regions, where the condition is more common, there is often a profuse overgrowth of hair over the defective arches. The latter is a pathognomonic sign, but may not develop until after puberty. Hip dislocation, clubfoot, partial paralysis of the lower extremities, changes in tactile and thermal sensation, anesthesia, hyperesthesia, and trophic ulcers may be the result of this condition. The deformity is always congenital and usually due to traction of amniotic bands preventing union of the borders of the medullary groove; various theories exist to account for these bands. Symptoms and complications may not arise until the child becomes older. No treatment is indicated if no symptoms or complications exist; if symptoms exist and bands are suspected, they must be searched for carefully and divided under strict aseptic precautions. J. W. SEVER (*Boston Med. and Surg. Jour.*, 1909, clxi, 388) reports 11 such cases, 2 boys and 9 girls; 4 cervical, 1 dorsolumbar, and 6 lumbar cases. Paralyses existed in 4, dislocation of hip in 2, and scoliosis in 2. A careful study reveals them to be the usual varieties of spina bifida occulta. Hypertrichosis was not constant. Not a great deal can be done for the paralytic cases, except to attempt the correction of the deformities.

Fatty Infiltration of the Liver in an Infant Aged Three Months.—C. A. PRATT (*Boston Med. and Surg. Jour.*, 1909, clxi, 395) reports the case of a child, perfectly healthy at birth and remaining so until its eleventh week, when, without any other apparent cause, except the heat, the infant lost its appetite and vomited after almost every feeding. The child was breast-fed until its seventeenth day, the illness of the mother forcing modified milk feeding thereafter. In the course of a journey, non-sterilized milk had to be given, and vomiting became more frequent, the baby becoming more and more ill. Physical examination revealed nothing but a liver markedly increased in size; it extended from the anterior superior spine of the right ilium to the umbilicus. From then on until her death, no matter what form of milk was given, whether human, cow's, modified, or even condensed milk, symptoms of toxemia appeared. At the autopsy the liver was seen to be greatly enlarged, pale yellow, and firm. The microscope showed "no normal liver tissue," fat replacing the protoplasm of the cells. The other organs were reported normal. Pratt believes the excessive amount of fat in the mother's milk (5 per cent.) to have been the cause of the fatty infiltration.

Idioglossia.—Idioglossia is an inability to pronounce certain consonants and substituting other consonants or vowel sounds for them. Hadden was the first to describe this condition in the *Jornal of Medical Science*, 1891. L. G. Parsons (*Birmingham Medical Review*, 1909, xiv, 9)describes two such cases in brothers, respectively ten and fifteen years of age. Both are bright and intelligent, but rather excitable and nervous. They cannot keep still for any length of time, and concentration of attention is impossible. There is no evidence of mental deficiency in either, and the elder brother writes and copies well; neither stutters. Three other children in the family pronounce words properly. The only consonants the elder boy pronounces correctly are b, d, l, n, t, y, and the younger, l, n, p, t, v, x, y, and even they are not employed correctly in combinations forming words. Concerning the causes, Parsons discards the following theories: (1) That idioglossia is baby language; (2) that it represents atavism or "sport" in language; (3) that it indicates mental deficiency; (4) the theory that it is a congenital deficiency of audition, not amounting to deafness, and an analogous condition to color blindness, is an explanation for some cases, but not for the majority.

The theory explaining all cases is, that it is due to some fault in Broca's area. Of the exact nature of the irregularity we are still in ignorance. Intelligence is not at fault, as in babies where both Broca's area and the frontal lobes are undeveloped. Spontaneous improvement after eight to ten years of age and absence of the defect in adults points to this theory as the correct one. The ultimate prognosis is good and the treatment consists in oral training, best performed with the child in an institution, as the parents, being accustomed to the child's imperfection, cannot do the teaching well.

Influenzal Meningitis.—David Davis (*Archiv. Int. Med.*, 1909, iv, 323) reports the cases of twin brothers who died of influenza. They became ill on the fifth day after birth. The cases ran an identical clinical course and terminated fatally on the ninth and eleventh days respectively. There was little or no distinct evidence, clinically, of meningeal involvement. The autopsy on the first child revealed, as prominent lesions, acute purulent leptomeningitis and acute enteritis. From the meningeal exudate and from the peritoneal fluid pure cultures of the bacillus of influenza were obtained. The usual atria of infection—nasal and tympanic cavities, lungs, bronchi, and throat—were normal. Omphalitis was not present. The children were breast-fed and were healthy until the fifth day, when the bowel movements became green and contained mucus and curds. Both children then became drowsy and refused the breast, and later developed attacks in which they became cold and cyanotic. There was no rigidity, and no symptoms of meningeal irritation developed until the ninth day, when there were general twitchings, but no actual convulsions. A comatose condition and marked cyanosis preceded death, which occurred in one child on the fourth day of the disease, and in the other on the seventh day. The temperature during the disease ranged from 101° F. to 104.5° F. Neither the mother nor any of the family were afflicted with colds at this time. The autopsy confirmed the intestinal involvement and showed a mild peritonitis. The intestinal tract was the

suspected portal of entry, and the facts suggest a similar infection of common origin in the two cases, a common symptom in reported cases being diarrhœa. The meningeal exudate was greenish yellow, highly purulent, and friable, and was more copious and widespread than is usual in the other forms of meningitis. This type also contains a very large number of bacilli in the exudate, both inside and outside the polymorphonuclear cells. Adams found 21 cases in which the bacillus of influenza was isolated from the meningeal exudate in pure culture. Besides this, a number of cases are reported in which there occurred a mixed infection of the bacillus of influenza with a gram-positive diplococcus. The typical bacillus of influenza grows only on hemoglobin media, and profusely so on pigeon's-blood agar.

Acute Hydrops of the Gall-bladder in Scarlet Fever.—Montenbruck (*Deut. med. Woch.*, 1909, No. 24, 1065) reports the case of a boy, aged five years, who was taken suddenly ill with the characteristic high temperature and eruption of scarlet fever. Two days after the onset he developed violent vomiting and severe pains in the upper part of the abdomen which lasted, off and on, for about eight days. The vomiting then ceased, but the abdomen remained very tender to pressure. During this time there was a constant fever, ranging between 102° F. and 104° F. On the eleventh day the child was admitted to the hospital with the diagnosis of abscess of the liver. There was distention and rigidity of the abdomen and extreme tenderness over the lower border of the liver and the gall-bladder region. As the child screamed with pain, and the abdominal condition seemed to demand it, an exploratory incision was made over the gall-bladder region, under chloroform anesthesia.

An immensely distended gall-bladder, the size of three fists, presented. It was freely movable and on tapping it was found to have held 250 c.c. of greenish, sterile bile. No stone was found. The gall-bladder was drained for a short time through one end of the incision, the sinus then closed, and the child made an uninterrupted recovery. During the operation a fine desquamation was noticed over the body. The urine remained normal throughout.

Chronic Rheumatoid Polyarthritis.—O. J. Kauffmann (*Brit. Jour. Children's Dis.*, May, 1909) reports the case of a girl, aged eight years, who showed enlargement of the nearly all the joints in the body. The joints were fusiform in shape, there was no pain or tenderness on pressure, and no grating. All had a certain amount of passive motion except the right hip-joint, which was ankylosed from an osteophytic growth. The joints were elastic and soft, but there was great wasting of the muscles. The spleen was enlarged, as were the superficial lymph glands all over the body, but they were not tender.

Slight bronchopneumonic symptoms in the lungs soon disappeared. The Calmette ophthalmo-reaction was negative twice. The temperature ranged from 99° F. to 101° F. The child had had three attacks of pneumonia—at one, two, and three years of age; rötheln at four years, followed by pains in the legs but no joint swelling. In the sixth year the joints began to enlarge, with wasting of the muscles, and the

condition had progressed. Treatment was useless. Kauffmann suggests that rheumatoid arthritis in children may be due to a chronic pneumonic condition in the lungs, as shown by the repeated attacks of pneumonia in this case.

OBSTETRICS.

UNDER THE CHARGE OF
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Menstrual Bleeding during Pregnancy.—Vogt (*Zentralbl. f. Gyn.*, 1909, No. 36) has examined the literature of menstruation during pregnancy, finding a record, in 100 cases, of undoubted periodical bleeding in eight. He reports a case in which the patient was a twin child well developed in childhood, having its usual diseases. Menstruation began about the twelfth year and soon became regular, lasting from five to eight days without pain. There was no other discharge. Twenty-two years before coming under observation the patient had her first child normally. When the patient came under observation she was found to be undoubtedly pregnant, as the fetal heart sounds were strong and regular and could be plainly heard. She stated that up to the sixth month of her pregnancy she had menstruated regularly. The pelvis was normal and internal examination revealed nothing abnormal about the genital organs. Other sisters had exhibited during pregnancy the same persistence of menstruation.

Syphilis of the Kidney Complicating Pregnancy and the Puerperal Period.—Hirsch (*Zentralbl. f. Gyn.*, 1909, No. 35) reports the case of a patient aged thirty years, anemic and highly nervous, who had had three pregnancies, during two of which she suffered from œdema with swollen feet and symptoms of toxemia. In her fourth labor the amniotic liquid was unusually abundant; the patient had a chill during labor with a pulse of 120, and the child was dead-born. The fœtus had hydrocephalus, enlarged spleen with abundant deposit of fibrous tissue in the capsule, ascites, enlarged thymus, and enlarged liver. There were no signs of syphilis in the skeleton. The mother's puerperal period was free from fever, but the secretion of urine was scanty and contained albumin—4 per cent. The urinary bladder contained abundant round epithelium, recognized as coming from the kidney, in groups and in tubes. There were no casts and no leukocytes. Repeated examination found the same thing. On examining the mother, she was found to have grown steadily worse since her confinement. There was leukodema of the upper portion of the body, especially pronounced in the upper thoracic region, axilla, and lymphatic glands. There was pronounced caries of the teeth, and linear scars on the reddish mucous membrane of the lip; on the right tibia there was an exostosis; over both shins there was slight œdema. The history showed that the patient had never made a complete recovery since her third labor; although her appetite had

been good, she had never gained in weight, but had distinctly lost. Pain was often complained of in the region of the right kidney, and the feet often swelled, and all of these symptoms were exaggerated during the last pregnancy. The diagnosis was made, from the examination of the fœtus and the mother, that syphilis in the right kidney complicated pregnancy and the puerperal period. Iodide of potassium was given freely. The quantity of urine increased, albumin disappeared, kidney epithelium grew less, and the pain in the region of the kidney much diminished. The patient increased in weight, the urine becoming normal. Some time after the patient was first seen, she was again examined and symptoms of syphilitic involvement of the nervous system were present. The patient improved markedly under the treatment, although her general condition was not good.

Twin Pregnancy with Abnormal Attachment of the Ova.—Leo (*Zentrbl. f. Gyn.*, 1909, No. 36) reports the case of a woman in her second pregnancy, aged thirty-one years, who had considerable persistent abdominal pain during the pregnancy. The uterus was anteflexed, enlarged, and pregnant about the second month. There was considerable distention and tenesmus of the abdomen. Symptoms of abdominal irritation continued, and a diagnosis of ectopic gestation was made. On opening the abdomen, ruptured tubal gestation was found and intra-uterine pregnancy advanced about three months. The patient's recovery was complicated eight days after operation by slight hemorrhage from the uterus, which subsided with rest and very simple remedies.

Peritonitis Following the Escape of Amniotic Liquid into the Abdomen During Cesarean Section.—Veit (*Zntrlbl. f. Gyn.*, 1909, No. 32) reports a case of Cesarean section performed upon a patient, who had been for a long time in labor. An effort was made to clamp the peritoneum of the lower uterine segment to the abdominal wall by forceps. The child was removed by version, the clamp became loosened, and amniotic liquid, containing bacteria, gained access to the peritoneal cavity. Peritonitis developed, with the presence of a dark, tenacious exudate in the peritoneum. An effort was made to drain the abdomen by section and the use of a glass tube. The effort was unsuccessful, the patient dying. Autopsy showed degeneration of the heart muscle, with extensive poisoning of the peritoneum with Bacillus coli communis.

Two Cases of Cervical Cesarean Section.—Küstner (*Zntrlbl. f. Gyn.*, 1909, No. 36) reports two cases in which he performed cervical Cesarean section. The first patient had had three labors, the last ending in embryotomy because of a tumor in the pelvis. During the fourth labor she was repeatedly examined outside the hospital, and as it seemed impossible to deliver her, she was brought into the wards. On examination, a pelvic tumor was found, rendering delivery impossible. An effort to dislodge the tumor failed, and the abdomen was opened in the median line. The uterus was opened outside the peritoneal cavity, the child delivered by version, and the placenta removed. The peritoneal edges were sewn together, but the tissues did not unite well, as a fistula formed between the cervix and the abdominal wall. This

gradually closed, the patient making a good recovery. She was considered infected upon admission, as she had been repeatedly examined by those whose antiseptic precautions were not thorough. In operating upon this patient effort was made to close the peritoneum by the use of clamps, rendering the introduction of the uterine contents into the abdomen impossible. A second operation was made for contracted pelvis, and was an extraperitoneal cervical section. The bladder was moderately filled with 300 c.c. of water, and the uterus opened outside the peritoneal sac. The head of the child was delivered by the use of one blade of a pair of forceps. The child was large, asphyxiated, but readily revived. Incisions were then closed with catgut, and the abdomen closed. Both of these patients made good recoveries. In the discussion, Baumm stated his belief that it is a mistake to use the longitudinal incision if an effort is to be made to avoid opening the peritoneal cavity. In twenty-five such cases he was successful only twice in avoiding opening the peritoneum.

GYNECOLOGY.

UNDER THE CHARGE OF

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Malignant Disease of the Uterus; A Digest of 265 Cases Treated in the New Hospital for Women.—Anderson and Platt (*Jour. Obst. and Gyn. Brit. Emp.*, 1908, iv, 381) report the work done in the New Hospital for Women, in London, from 1895 to 1907. There were 217 cases in which the disease affected the cervix, and 48 in which it affected the fundus. Hysterectomy was done by the abdominal route 90 times, with a mortality of 6, and 38 by the vaginal route, with 3 deaths. The vaginal route was employed principally previous to the year 1901, and the abdominal subsequently. Of the 217 cervix cases, 93 were squamous-celled carcinoma; 24 adenocarcinoma; 93 were unspecified; 2 were sarcoma; and 4 were endothelioma. Of the 48 fundus cases, 37 were adenocarcinoma, 1 was chorionepithelioma, and 10 were sarcoma. Cancer of the cervix was present in 29 of the 38 cases of vaginal hysterectomy, and the disease recurred in 12 in the vaginal scar within a few weeks from the time of operation. In 58 cases of abdominal hysterectomy for the cervical form of malignancy local recurrence occurred in 4 or possibly 5. Anderson and Platt insist that recurrence after the extended abdominal hysterectomy is in the iliac glands, and their removal is, therefore, strongly recommended. Of the 29 cases of cancer of the cervix subjected to vaginal hysterectomy, one patient is known to be well now, seven years after operation. The after-results in 58 abdominal hysterectomies for the same conditions are: 26 women are known to be alive and well, between one and one-half and four years after the operation; 17 developed symptoms of recurrence, 11 in the first year and 6 within

three and one-half years; 15 cannot be traced. Of the 48 patients suffering from malignant disease of the fundus, 39 were subjected to hysterectomy, 30 of them by the abdominal route. Of these, 22 have remained in perfect health two to seven years, 7 developed symptoms of recurrence from one to five years later, 3 died, and 7 cannot be traced. Out of the 37 cases of adenocarcinoma of the fundus, 17 had fibroids as well; and of 10 of sarcoma, 4 had fibroids. The vast proportion of women with cancer of the cervix are over thirty years of age, the average being forty-four and one-half years, and the average number of children was five. Six were unmarried and childless. In the fundus cases the average age was fifty-eight years, and the average number of children was 1; 18 of the 38 were unmarried and childless.

A Study of 131 Consecutive Cases of Fibroid Tumors of the Uterus Demanding Operation.—A MacClaren (*Annals of Surgery*, 1909, l, 281) reports the last 100 hysterectomies for uterine fibroids he has done and the myomectomies he has done during the same period of time, 31 in number; 78 per cent. of the hysterectomy patients were forty-five years of age or older, 1 was twenty-five years, 1 was twenty-eight years, and 28 per cent. were between thirty and forty-five years; 87 per cent. of the myomectomy patients were not more than forty years of age. In 3 of 60 myomectomies MacClaren has done fibroids have developed, requiring a second operation, in two of them hysterectomy. In the 3, the second operation was required within two years following the myomectomies. Myomectomy during pregnancy was done 4 times, with one miscarriage. MacClaren does not advise removal, during pregnancy, of a symptomless fibroid, nor abstaining from such operation for proper indications during pregnancy, even though the child be not viable. Three deaths occurred in the hysterectomy series and 2 in the myomecties. A very interesting statement is made regarding his gall-bladder surgery. In 20 consecutive cases of pelvic operations in which the gall-bladder had been opened and drained there had been 3 deaths, while the general mortality of his gall-bladder work in other cases was but 2 per cent. MacClaren has consequently abandoned Kelly's plan of removal of gallstones incidental to pelvic operations. Twice after myomectomy MacClaren has been obliged to do vaginal section for hematomas, although in the habit of employing vaginal drainage after this operation. In 10 per cent. of the hysterectomies this procedure was employed. Seventeen of the 100 hysterectomy patients had never married; 22 of the married ones had either miscarried or never been pregnant; 17 other married ones had not had a child for twenty years; 25 had had the last child from ten to twenty years before operation; 4 had given birth to children during the last preceding three years, and the remaining 11 of the hysterectomy subjects had each had a child between five and ten years before operation.

Rare Metastases of Uterine Carcinoma.—Heinrich Offergeld (*Monatsschr. f. Geburtsh. u. Gynäk.*, 1909, xxix, 181) reviews the literature concerning rare metastatic growths from carcinoma of the uterus, and arrives at the following conclusions: In uterine carcinoma metastases occur very rarely in striated muscles; secondary growths occur with greater frequency in the myocardium. They occur in advanced cases

only, and arise hemogenetically without exception. The clinical symptoms of these metastases vary greatly, depending entirely on the function of the muscle affected (myocardium, eye muscles, muscles of the body; the latter not giving rise to any symptoms). On account of absence of all clinical symptoms the body musculature should be carefully examined at autopsy. The ureteral wall possesses a certain immunity against carcinoma; in two cases the carcinoma was inoperable, in the third case of this kind operation was performed successfully. Nothing definite is known of the mode of dissemination and of the clinical course. The apparently intact ureter should be examined microscopically at autopsy for possible microscopic carcinomatous infiltration of its wall. Carcinoma of the thoracic duct occurs more frequently in uterine cancer. The secondary carcinoma of the thoracic duct arises lymphogenetically through the medium of the inguinal, hypogastric, and lumbar plexuses, which empty into the thoracic duct. Carcinoma of the thoracic duct is of special importance, as it gives rise to further hematogenous metastases. Whether carcinoma of the thoracic duct in uterine cancer occurs in a large number of cases remains to be proved by future careful autopsies. Carcinoma of the supraclavicular and infraclavicular glands is found only in extensive glandular disease associated with uterine cancer. These metastases are of lymphogenous origin through the medium of the thoracic duct, with marked preference for the left side on account of the anatomical conditions. The mediastinal lymphatic glands are frequently found carcinomatous in uterine carcinoma, while secondary carcinoma of the mediastinum itself is very rare. The metastatic mediastinal tumors in uterine carcinoma arise lymphogenetically, either directly from the lymph glands, or by retrograde lymphogenous transportation.

The Treatment of Inoperable Cancer of the Uterus.—Gellhorn (*Amer. Jour. Obst.*, 1909, lix, 799) related to the Chicago Gynecological Society his technique of the application of acetone in the treatment of inoperable cancer of the uterus. While not claiming for this remedy curative properties, Gellhorn declares it the most efficient known in preventing hemorrhages and malodorous discharges. Two classes of cases are enumerated in which its application cannot satisfactorily be made. The first is composed of those *in extremis* when first seen. In these the body of the uterus seems to be merely a thin shell within which the ravages of the disease progress unhindered, even the most temporary amelioration being impossible, and the primary focus has lost all its significance. In the other class are placed cases in which the application of acetone would seem unpromising because of the attending unsurmountable technical difficulties. The principal difficulty is the unusual location of the cancer, as, for instance, the interior of the uterus, or the lower part of the vagina, or the vulva. This technical difficulty has brought about, as a substitute for liquid acetone, the white powder, acetone bisulphite, which can be used with a powder blower. It is less efficient than the liquid acetone.

DERMATOLOGY.

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A Case of Rodent Ulcer Cured by Radium.—Sequeira (*Brit. Jour. Derm.*, February, 1909) reports a case of rodent ulcer which has been cured, and has remained so, five years before. The disease had begun at an unusually early period, when the patient, a young woman, was between fifteen and sixteen years old, and was situated upon the lower lid. The diagnosis of rodent ulcer was established by microscopic examination. The ulcer had partially healed under *x*-ray treatment, but this had to be stopped on account of conjunctivitis, and a relapse followed. In February, 1904, six applications of 10 milligrams of radium in tubes, having an estimated radio-activity of 500,000, were made on six successive days; this produced a marked reaction, and on February 24 the ulcer had healed.

Lupus Erythematosus Apparently Cured by Zinc Ionization.—MacLeod (*Brit. Jour. Derm.*, March, 1909) reports a patient, a middle aged woman, having an erythematous lupus, of seven years' duration, in whom a cure had been obtained by the employment of zinc ionization. Two per cent. zinc sulphate was used, and a current of about 5 milliamperes, ten minutes at a time, to each patch. Nine sittings in all were given; and under this treatment the scaliness and redness had disappeared, leaving a pale, supple, slightly depressed scar.

Graham Little (*ibid.*) also reports a patient with erythematous lupus, a girl, aged fourteen years, in whom the same method of treatment had produced satisfactory results. He regards this treatment as the most rapid and efficient at our disposal in dealing with this obstinate affection.

The Treatment of Superficial Epithelioma by Curettage and Immediate Radiotherapy.—Lenglet and Sourdeau (*Bull. de la soc. Française de derm. et de syphil.*, 1909, No. 2) have found the following method most successful in the treatment of superficial epithelioma: Every epithelioma, whatever may be its seat, is submitted, if possible, before irradiation, to complete, methodical scraping. If this cannot be done on account of its situation, and extirpation is possible, it is removed surgically. The scraping should be preceded by complete anesthesia of the entire area to be curetted; and if local anesthesia is not possible, recourse must be had to general anesthesia. The curettement must be most thoroughly done. Bleeding is usually stopped by simple compression,

but exceptionally the parts must be lightly touched with the actual cautery. Immediately after the operation, before the wound is dressed, it is irradiated, six H. being given, rarely seven, the rays used being No. 6 or No. 7, the latter being preferred. The second séance is not given until the time of reaction is largely passed, usually about the twentieth to the twentieth-eighth day; and the séances are thus continued until cicatrization occurs. The importance of this preliminary scraping is shown by the fact that Lenglet and Sourdeau have obtained a cure in 12 cases out of 36, with one irradiation, and in 9 with two séances. They believe that this combination of curettement and radiotherapy gives results superior to any other method of treatment.

Impetigo Contagiosa.—Lewandowsky (*Archiv f. Derm. u. Syph.*, 1909, xciv, Heft 2 und 3) always found the streptococcus in 100 cases of impetigo examined bacteriologically, partly in pure culture, partly associated with the yellow staphylococcus, the former greatly exceeding the latter in numbers. In the great majority of cases of ecthyma the streptococcus was found in pure culture in the primary purulent lesion; in rare instances it was contaminated with isolated staphylococci. In one out of a small number of cases of pemphigus neonatorum and infantum examined, only staphylococci were found, while in the others streptococci in trifling numbers were also present. In impetiginous eczemas both streptococci and staphylococci were found, as well as in other serous lesions of various other dermatoses. Intra-epithelial inoculation with pure streptococcus cultures produced impetigo contagiosa; and epithelial cutaneous inoculations were followed by ecthyma. Inoculations with staphylococci taken from impetigo were usually without result, or produced an abortive pustule. Staphylococci obtained from pus, eczema, impetigo, and some other diseases were affected in the same manner by agglutinating sera.

Occupation Carcinoma.—Zweig (*Derm. Ztschr.*, February, 1909) under the designation occupation carcinoma, reports three cases of carcinoma of the skin occurring in workmen employed in making coal briquettes. These briquettes are made by mixing ground coal with pitch and tar, subjecting the mixture to a high heat in an oven and afterward making it into bricks by pressure. Zweig thinks the injurious effects of this occupation upon the skin are due either to the dust arising in the process of mixing or to certain volatile substances produced in the heating of the mixture. This form of carcinoma is identical with those previously described as chimney-sweeper's cancer, paraffin cancer, etc. Zweig thinks there can be no doubt of the immediate relationship between this form of carcinoma and the chemically acting agents used in these occupations.

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF
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The Bleaching of Flour.—Within a few years the bleaching of flour has become a process of extensive employment in this country and Great Britain, possibly in other parts of the world also. When flour stands for some six weeks to as many months, it gradually becomes whiter. This change is attributed to "aging." The seller takes his samples and on the appearance of these, which may be many months old, he contracts to deliver so many carloads of the same kind. Recently ground flour shipped to fill this order disappoints the buyer, because it is not so white as the sample upon which he made his purchase. This has led to artificial bleaching with peroxide of nitrogen. The machinery for this purpose is of varied construction, but the principle is the same in all. The oil of the flour is bleached, and nitrite is produced. The chemist distinguishes between bleached and unbleached flour by the detection and estimation of nitrite in the former. The question of the bleaching of flour has been for some months before the Secretary of Agriculture at Washington, and some scientific work, with the object of determining the effect of the bleaching process upon the health of the consumer, has been done. In Halliburton's work (*Journal of Hygiene*, 1909, ix, 170) attention is given to the influence of nitrite on the digestive enzymes. All experiments were made in vitro. Nitrite, when present in the very small proportion of 1 to 32,000, markedly retards the digestion of starch by saliva. The time necessary to reach the achromatic point when tested with iodine was taken as the standard of measurement. The time required to reach this point in the presence of nitrite (1 to 32,000) was thirty-three minutes, while digestion in the control tube was complete at eighteen minutes. In testing proteolytic ferments the protein was stained with carmine and the tint of the solution used as a comparative measure. In tubes containing potassium nitrite (1 to 8000) reckoned as nitrite, digestion was wholly prevented and when the nitrite was reduced to 1 to 32,000 the digestive activity was only one-seventh of that in the tube free from nitrite. The question arose as to whether previous treatment with nitrite, even when the nitrite was removed before subjecting the protein to the action of the ferment, would affect digestion. For the purpose of solving this question three sets of tubes were prepared as follows: (1) Those with nitrite, (2) those previously treated with nitrite, and (3) those to which no nitrite had been added. The results were: In (1), digestion was slight; in (2), slight but greater than in (1); in (3), complete. Halliburton states his conclusions as follows: (1) The presence of nitrous acid (even in the comparatively innocuous form of a salt) hinders enzyme action. (2) Previous treatment with nitrous acid alters a protein in such a way as to render it less readily susceptible to the solvent

action of digestive juices. He also found that the starch in bleached flour is less easily digested by the saliva than that in unbleached flour. A like result followed in his experiment on the peptic digestion of the glutin in the flours.

(Halliburton's finding that nitrite so markedly impedes the salivary digestion of starch is, to say the least, unexpected, since nitrite is a normal constituent of human saliva, or, at least, has been long regarded as such by physiological chemists.—V. C. V.)

Sulphite in Cider and Perry.—Durham (*Ibid.*, 17) thinks that the permissible limit of the addition of this preservative to these beverages should be expressed as "total sulphur dioxide" obtained by distillation with phosphoric acid, and that this should not exceed 100 mg. per liter. A trace should be defined as less than 10 (possibly 20) mg. per liter, and when this is exceeded, declaration of the presence of sulphite should be made on the label. All cider makers who desire to use or who do use preservatives should be registered and under official inspection.

Chronic Lead Poisoning.—Notwithstanding recent sanitary improvements in manufacturing establishments, chronic lead poisoning is by no means infrequent in large white lead factories. Animals about such factories vary greatly in susceptibility to the poison. Apparently it is without effect upon rats, which may infest the white lead factory in great numbers, but cats brought in to drive out these pests soon succumb to chronic lead poisoning. There has been more or less discussion as to whether or not poisoning may and does result from the inhalation of the dust that always permeates the atmosphere of white lead factories especially of those not adequately supplied with fan exhausts. Goadby (*Jour. of Hygiene*, 1909, ix, 122) has investigated this question experimentally, using cats, and has answered the questions in the affirmative. The material used consisted of: (1) Flue dust, containing from 50 to 60 per cent. of lead oxide. In the process of desilverizing, zinc is added to molten lead containing gold and silver; the zinc holds the precious metals, and is then separated from the lead by differences in melting point. The lead from which the zinc has extracted the gold and silver, known as "poor lead," is run into a pot and there treated with air and steam under pressure, for the purpose of ozidizing any zinc that it may contain, but some of the lead also is oxidized, and in the form of a free dust finds its way to the workmen. (2) Litharge is broken into large lumps by hand, and then these are ground in a mill. Some finely divided litharge reaches the men who feed the mill. (3) More or less white lead in the form of a fine dust escapes from the ducts leading from the machines used in packing dry white lead in barrels.

The first effect noticed in the cats caused to inhale these dusts was an alteration in the face due to the absorption of the orbital and buccinator fat, and giving the animal a pinched appearance much like that shown by men poisoned in lead factories. Colic was the next symptom. The cats were obstinately constipated, and showed much abdominal distress. Then the animals developed muscular weakness, shown

especially in the extensors of the limbs, giving a curious stiff gait. The poisoned cat walks on the tip of its toes, and in endeavoring to turn around is compelled to arch its back and draw its feet close together to prevent falling over. The back muscles are distinctly weak, so much so that when the animal is held up by placing the forefinger and thumb behind the ears it hangs down straight and can make no move to twist its body or claw the hand. When it jumps off a table it falls upon its belly, the extensor muscles being unequal to the strain. At first the loss of weight was progressive, but, after falling to a certain point, it remained constant, in some cases showing a tendency to rise later. The experiments were not pushed to a fatal termination, but the animals were killed. No blood examinations were made.

Transmission of Microörganisms through Berkefeld Filters.—Bullock and Craw (*Ibid.*, 35) conclude their study of this point with the following findings: (1) Of two filters with porcelain nipples, one gave a non-sterile filtrate with London tap water immediately, and the other after three hours and forty minutes. These filters had been boiled for one hour but not autoclaved. (2) Of three with metal nipples, two gave immediate contamination with tap water, and the third after two hours and forty minutes. These filters also were new and had only been boiled for one hour. (3) Two dried Berkefeld filters, one with metal and one with porcelain nipples, on immersion of a portion of their walls in a water culture of Bacillus prodigiosus, allowed this organism to pass into the anterior of the filters.

Antiplague Measures in San Francisco.—Blue (*Ibid.*, 1) sums up the measures that have apparently proved satisfactory in the eradication of the plague at San Francisco as follows: (1) Attack upon the habitation and food supply of the rat; (2) destruction of rat burrows and nests; (3) prevention of access of rats to food by concreting stables, warehouses, markets, etc.; (4) exclusion of rats from houses by the use of concrete or other impervious material; (5) filling of rat burrows with strong solution of chloride of lime; (6) disinfection of all places in which either human or rodent cases have been detected.

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