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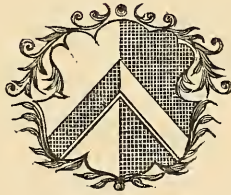
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JAMES ANDREW, M.D.

AND

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ON SOME OF THE SEQUELS OF TYPHOID FEVER.

BY

SIR JAMES PAGET, BART., F.R.S.

I am conscious of the danger which one who practises surgery usually incurs in writing on subjects that are naturally in the field of practical medicine. But among the diseases that are likely to follow typhoid fever there are some which occur in so advanced a period of the convalescence, and appear so local and so far dissociated from the fever, that they may be observed more often by surgeons than by physicians. I venture, therefore, to call attention to the frequent occurrence after typhoid fever of these following diseases, namely, phlebitis, periostitis with or without necrosis, especially periostitis of ribs, local paralysis of muscles.

Of the frequency of these diseases after typhoid fever I can have no doubt. I believe that I have seen, taking all together, more than seventy cases in the last ten years. I do not feel competent to deal with the question whether each fever has, as seems very probable, its own proper sequels, and is in this sense, though perhaps in less degree, as specific as in its fever-period; but, excepting the phlebitis, I have not yet seen any of the diseases I have enumerated after any other than typhoid fever, or a fever which I suppose to be closely related to it; and I have not seen after typhoid any corresponding number of cases of large lymph-glands, diseased joints, or other diseases of mere debility such as may follow any acute illness.

The fever which I have referred to as 'closely related' to typhoid, is that which in the last two or three years has been prevalent in Naples, Rome, and I believe other places in middle and southern Italy. Some call it typhoid; Neapolitans often call it Roman, Romans Neapolitan; in some cases I have ample reason to believe that it was genuine typhoid; and the likeness of its frequent sequels makes me ready to believe that it always was so.

It is common to all these sequels of typhoid fever that they appear when the patient is considered to be 'well of his fever,' when his temperature is normal, and I believe constant, when he is beginning to move about and becoming stouter and stronger. I do not remember to have seen or heard of a case in which any of them occurred during the continuity of the fever.

The *phlebitis* has in it no character widely different from those in which it is seen in other of its so-called adhesive forms. Its most usual seat, by very far, is in the femoral, especially the left femoral, vein. It is generally marked by aching and tenderness along the line of the inflamed part of the vein, with stiffness at the groin, and more or less of firm general swelling of the limb. There is no marked access of fever, no other disturbance of the general health than may be ascribed to the local distress. The duration of the painful symptoms is, I think, quite uncertain; but after they have ceased, some bigness and clumsiness of the limb usually remain for many months. In many cases the limb remains always larger than the other, but not weaker or less muscular; and in some, in which, I suppose, the femoral vein is in more than the usual length obstructed, the superficial veins become very large and tortuous.

I have not seen or heard of a case in which suppuration attended the phlebitis, or in which embolism occurred; and I do not know of any in which the affected veins were examined after death. I believe that the condition is one of inflammatory swelling and thickening of the walls of the vein, and consequent narrowing of its canal. Very probably, also, there is lymph-exudation or deposit in layers from the blood. I doubt whether thrombosis is ever a primary event, though it may occur as a consequence of the inflammatory changes in the lining of the vein.

The *periostitis* following typhoid fever has its most frequent seat on the tibiæ, but I have seen it on the femur, the ulna, the parietal bone. In one case it was symmetrical on the lower parts of the shafts of the tibiæ; but, with this exception, I have not seen it on more than one bone in the same person. It has always been circumscribed within a space of from one to three inches in extent, has always occurred in the convalescence, has never been attended with the delirium, fever, or other severe

symptoms attendant on acute necrosis. At first it is only marked by the limited, hot, and very tender swelling of the periosteum, and I have seen cases in which the periostitis has remained very long in this condition, and then slowly subsided without necrosis or any other abiding change of structure. But in two cases, at least, the patient has remained for months—in one case for three years—subject to repeated attacks of pain and swelling of the periosteum, yet without signs of suppuration or more change than a slight permanent thickening.

When the periostitis has been associated with necrosis, the extent of the dead bone has always been far less than that of the inflammation over it, and, with rare exceptions, only the hard compact structure of wall or outer table has perished. The separation of the dead bone has taken place in the usual way, and has been followed by healing, which, so far as I have seen, has been permanent.

The *periostitis of the ribs* is in so much unlike the kind just described, that I have never yet seen it associated with necrosis. It so nearly resembles the ordinary scrofulous periostitis of ribs, that I have sometimes thought it should be regarded too as only an evidence of scrofula educed by the feebleness of nutrition consequent on the fever. Yet I have never seen it associated with other similar signs of scrofula, and it has occurred, after typhoid, in some of so robust and apparently unblemished constitution, that it would seem absurd to impute scrofula to them.

In the usual course of this disease, beginning always, I believe, in the convalescence, a well-defined round or oval swelling is found on one or more ribs. Whether on bone or cartilage, the swelling is generally on the front of the chest. It is painful and tender, and suppuration slowly ensues in it. Then slowly it discharges itself through one or more channels leading obliquely through the overlying structures and through small openings in the skin. The flow of pus, thin and pale, continues usually for many months; I have known it continue for even two years; and not rarely fresh openings are formed for branches from the first channel of discharge. In the end complete healing takes place, with deep depressed scars and complete clearing-up of all the periosteal swelling.

I have never known necrosis of a rib in these cases, nor ever seen or heard of any advantage from laying wide open any of the sinuses. The general health never appears to be seriously affected by the abscesses, unless in persons of naturally feeble health. Commonly, indeed, the patients feel quite well, and can be active, suffering nothing more than slight local inconvenience.

All the cases of this sequel of typhoid fever that I have seen

have been singularly alike, varying only in the position of the abscess. In one, in which I believe that the disease began in the periosteum of the 11th or 12th rib, a great abscess slowly made its way between the abdominal muscles, and I had to open it at the groin. In that case alone have I seen life endangered.

Local paralysis of muscles in the convalescence of typhoid I have seen in only a few cases, and in these the muscles affected were those supplied by the peroneal or the anterior tibial nerve. Once, also, I have seen paraplegia; but I might have doubted whether the paralysis were in more than some accidental connection with the typhoid fever, if it were not that the frequency of nervous diseases in the convalescence of typhoid has been abundantly proved by others, especially by Dr. Nothnagel.* He has seen or found on record cases not only of local motor paralysis, but of hemiplegia, paraplegia, some muscular tremors and spasms, anæsthesia, hyperæsthesia, and neuralgia.

I may refer to his paper for its value in regard not only to these sequels of typhoid fever but to the general pathology of convalescence. I remember that the late Dr. Baly used to say that a book on the diseases of convalescence might be one of the most valuable that any physician could write. Perhaps I may hope that to such an one I have now contributed.

* Die nervösen Nachkrankheiten des Abdominaltyphus in the Deutsches Archiv für Klinische Medicin. Bd. ix. p. 480. 1872.

ON
PHRENITIS ÆSTIVA.

BY
SAMUEL GEE, M.D.

To the eighth volume of the Reports I contributed a paper upon meningitis, or rather upon that kind of meningitis which I believe to be most common, namely, that which is secondary to otitis interna. On the present occasion I will narrate some cases of a very similar form of disease, not secondary to otitis. But I cannot call these latter cases by the name of meningitis, for that is an anatomical term, and the anatomical proof is lacking here. Wherefore I choose the name Phrenitis, for that is a semeiotic term, suited to my present purpose.

I. *The definition.*—The meaning of the word phrenitis has been well set forth by that learned pathologist Robert Burton. He says:—‘Phrenitis, which the Greekes deriue from the word φρήν, is (1) a Disease of the Mind, with a continuall Madnesse or Dotage, which hath an acute feauer annexed, or else (2) an inflammation of the Braine, or the Membranes or Kells of it, with an acute feauer, which causes Madnesse or Dotage.’ I have called my cases Phrenitis Æstiva, simply because they all happened in hot summer weather, and because I believe that there is more than a mere coincidence herein. Indeed the term Heatstroke might perhaps have been used, but I prefer the name which does not involve any opinion.

II. *The conditions of the patients before the attack* were as follows: Their sex and age respectively—L. S., a girl, 1 year 10 months; R. M., a boy, 4 years; E. C., a girl, 6 years 5 months; W. P., a boy, between 6 and 7 years. Health, until attack of phrenitis, very good.

III. *The conditions of the attack* were these:—

1. *Concerning the time of the year.*—The dates of the attack

were respectively: L. S. on July 31, R. M. on June 24, E. C. on July 12, and W. P. on July 23.

2. *Concerning exposure to heat.*—L. S. was taken out in a perambulator by a servant on the day of the attack, a hot day; but it could not be learned whether the child had been unusually exposed to the sun's heat. R. M. sat for some hours on the day before his attack in a stand in Hyde Park, on the occasion of a review of the volunteers; the sun was at times very hot. E. C. was attacked in very hot weather; the night of the seizure was very hot and close; at noon of the day before the temperature in the shade was 91° F.; but she had not been out of doors excepting in the evening, her mother having taken especial care not to send her out in the hottest part of the day. W. P. was running about in the sun a great deal on the day before his attack, a very hot and close day; he ate a good meal at 7 P.M., and seemed in good spirits.

3. *Concerning food.*—The brother of L. S. vomited gooseberries and was purged without drugs at the same time as the girl herself was taken ill, but it could not be proved that she had eaten any fruit. Of R. M. I have no particulars under this head. E. C. brought up undigested raspberries at the beginning of her attack. W. P. had eaten ices, and perhaps other things of the same kind, on the day before his illness began.

4. *Concerning the time of the attack.*—L. S. was attacked in the evening of the day on which the exposure was supposed to have taken place. R. M. seemed well and slept well on the night after exposure; the next morning he was taken for a short walk, and his illness began. E. C. was attacked at two o'clock in the morning. W. P. was attacked the morning next after exposure.

IV. *The manner of the attack* was as follows:—

1. L. S. vomited in the evening; during the night she went off into a fit of convulsions, and was relaxed in her bowels. The vomiting was not repeated, but the convulsions and diarrhoea continued.

2. R. M., after a short walk in the morning, became very tired, complained of headache and pains in his feet; he vomited once. He became exceedingly cold, and in an hour or two feverish. The vomiting was repeated next day; bowels confined.

3. E. C. at 2 A.M. went to her nurse's bed, complaining of cold and shivering, at 2.15 A.M. vomited, soon afterwards became hot. The vomiting was repeated, and the bowels soon became relaxed.

4. W. P. in the morning complained of feeling cold; no vomiting; chilliness did not continue; marked lassitude, so that he

remained indoors all day. Second day, lassitude greater; much pain in left thigh; seemed hot. Third day, bowels freely opened by a pill his mother gave him; axillary temperature 104° in the morning. Fourth day, became comatose.

V. *The course of the disease* was as follows:—

Case of L. S.

On the 2d, 3d, and 4th days a little looseness of the bowels; no other symptom. On the evening of the 5th day the convulsions recurred, and lasted all night, on and off; bowels open several times; motions loose, bilious, mixed with much mucus and a little blood. On 6th day, at 8 A.M., saw her, with Mr. Cockell of Hackney; deep coma, incessant convulsions, temperature in axilla 108.5° , pulse imperceptible, breathing very laboured, skin purple and sweating, looked dying. Put into a bath of water at 58° , at once the convulsions ceased; she became more conscious, breathing easier. She was kept in the bath until the temperature in the rectum fell to 101° , was then wrapped in blankets, and beef-tea with brandy was given. 10 A.M., temperature in axilla 99.4° , marked tendency in feet to become cold. The convulsions did not recur; child remained much more conscious. On 17th day saw her again; looks pretty intelligent; takes notice when spoken to; sleeps quietly; no loss of flesh; swallows well; tendency to constipation; passes water freely; pulse regular; breathing natural; tache cérébrale; pupils large, rather sluggish, equal, no squint; constant carpopedal contraction, no rigidity elsewhere; no paralysis; can move limbs, but cannot stand; constantly rolling head about and fidgeting; does not shed tears, but cries much when examined. On 20th day, better, looks natural; more conscious, sleeps well; carpopedal contractions and rolling of head continue, but are less; she most likely can see. Six months after the attack, a state of incomplete general paralysis; weak in back; cannot stand; cannot talk; probably can both hear and see. Twelve months after the attack, well grown; looks well; laughs much, but does not look idiotic; cranium well shapen and sized; no doubt hears and sees well; does not speak at all; until a month ago could not even sit up, now can sit on floor, but cannot sit on a stool; if lying on floor, can raise herself and sit; cannot stand, but if held up by shoulders, bears much weight on legs; passes stools and urine into bed and clothes, if not carefully watched; urine clear, 1015, no sugar or albumen; no squint or other eye symptoms; in fact, no paralysis; in all respects of brain function, very much like a baby of twelve months old. Before her illness she could walk well and talk a little. No otitis at any time.

Case of R. M.

The second day, in the morning, seemed better, yet vomited once or twice; bowels confined; in the evening and during the night very restless and noisy, passionately screaming out at the top of his voice in fancied talk with persons not present, noise almost continual; no sleep. On 3d day much the same. On 4th day, in the evening became very low, thought to have slept a little; pulse, which for first day or two was full and at times infrequent, now became weak and more frequent. On 5th day saw him with Dr. Cumberbatch; restless, at times cries out; what he says cannot be understood; cannot be made to show tongue; cheeks mottled scarlet; pulse very weak, not very frequent, irregular; breathing quiet and regular; belly natural; pupils smallish, unequal; internal squint of right eye; great heat of head; limbs quite cool; body also cool in parts exposed; no tache cérébrale. Ice was applied to his head; the exertion of being raised to have his hair cut off wearied him greatly, so that afterwards he lay in a coma vigil, only occasionally moving; often trying to pick or beat away things in the air; cheeks, chin, neck flushed scarlet; scalp tender; pulse throughout excessively variable, sometimes very weak and fluttering, and anon full, only now and then irregular; breathing always regular; pupils became larger and equal. On 6th day, morning, much the same; no sleep last night; had not passed water, but did so on being held upright; bowels open frequently without help; noticed for first time to hold head stiffly back: evening, great improvement, slept much all day; is asleep now, eyelids quite shut, pupils closely contracted; as he awakened, they enlarged; squint persists; head still held back; pulse 96, regular; breathing easy, regular; belly not retracted; has passed water; swallows well; head hot when ice is not applied. On 7th day, much as yesterday; much natural sleep; has spoken rationally several times; head still becomes hot sometimes, feet tend to become cold, body not so; head still held back; face still flushes at times; pulse 84, regular. On 8th day, perhaps rather lower than yesterday; much natural sleep; pulse regular, but variable in frequency, sometimes 100, sometimes 120; head only occasionally hot, feet tend to become too cool; pupils natural in all respects; squint persists; head held back rather less; no other rigidities. On 9th day, improvement continues; head not hotter than rest of body, which is of natural heat; feet much warmer than before; moves about of own accord; no rigidity excepting in the neck; no paralysis; more irritable, dislikes being moved or uncovered, cries out loudly, yet does not seem to know anybody; speaks a short sentence now and then. On 10th day,

much the same, asked for the chamber-pot, but does not seem to recognise anybody. On 11th day, better, neck less stiff. On 13th day, most marked improvement; sleeps much, but sometimes lies awake for hours at a time with eyes half open; moves about strongly in bed; speaks properly; squint remains; no retraction of head or stiffness of neck. On 18th day, continuous improvement; talks much and sensibly; lies on a sofa; when raised, there is still some stiffness about his neck; squint as great as ever. On 27th day, walks by being held up on each side; pulse infrequent and notably irregular. On 41st day, steady improvement; walks supporting himself by furniture; muscles feel very flabby; takes no notice of anything said to him, nor indeed of any sound however loud; talks much about his own matters, and what he says is sensible, but he makes no attempt to answer by word or sign; general condition very good. On 54th day, no doubt totally deaf. Three years afterwards, totally deaf; speech has become thick and unintelligible except to his nearest friends; can read; well grown; slight squint; mother thinks he has not quite the proper use of his legs; pulse 50, irregular; heart natural. Never had otitis at any time.

Case of E. C.

After her attack (2 A.M.) she continued to vomit at intervals, became hot. 6 A.M., solid natural stool. 8 A.M., another stool, watery; vomited at same time green water; took some bread and milk, brought it up again almost instantly curdled. 10 A.M., quite rational, listened to reading; castor oil given; bowels open soon after, dark brown, watery, not foetid. 11 A.M., fell asleep for half an hour; on awakening was mildly delirious, talked nonsense, picked at bed-clothes. Soon afterwards, severe convulsions, lasted four or five minutes, followed by coma so deep that she could not be roused. 1 P.M., pupils largely dilated, immovable. 4 P.M., saw her with Dr. Metcalfe; looks dying; comatose, but can be roused somewhat; drink put into her mouth remains there until she is made to sit up; lips livid, but rubbing them gently makes them florid; skin everywhere injected, mottled, livid; temperature in armpit 105° ; feet and hands cold; any part of body becomes cold on exposure; pulse 200, very weak; breathing deep, regular, air enters lungs well; no physical signs of disease in chest, no dyspnoea, no action of nares, no tache cérébrale; eyes half shut, pupils largish; not wholly insensible, equal; no strabismus; slight nystagmus; abdomen retracted; has just passed a scanty mucous yellowish motion; rigidity of limbs, and somewhat of back. Ice was applied to head, and warmth to rest of body. 9 P.M., died comatose. No otitis at any time.

Examination of body twenty-two hours after death.—Rigor mortis well marked. Blotchy purple discoloration of skin all over body, not removable by pressure. Eyeballs singularly sunken. Removing scalp, much blood oozes from sutures. Calvaria natural. Small black clots in sinuses of dura mater. Arachnoid sticky, no effusion. Vessels of pia mater decidedly full of blood, but no exudation whatever anywhere; no tubercles. Brain looks swollen; convolutions much flattened; ventricles remarkably empty; on careful examination it seemed otherwise natural, not soft. Heart, lungs, kidneys, liver, spleen, stomach, natural. Intestines natural; nothing in them but air, and not much of this.

Case of W. P.

On 4th day, seen with Mr. W. B. Thorne and Dr. Baldock. At 4.30 P.M., half comatose; will not answer questions; pupils dilated, act slowly; frequent jumpings of limbs and trunk, palpitations or half-convulsive movements, has not been thoroughly convulsed; temperature in rectum 107.4° ; pulse 180, full, regular; breathing 32, natural; very great throbbing of heart and carotid arteries; no eruption; no tache cérébrale; physical signs of heart and lungs natural; no pain in left thigh now, limb quite natural; swallows well; no vomiting or looseness; spleen impalpable; urine turbid with urates, no albumen. He was put into a bath at 75° , gradually lowered to 68° , and was kept in it about a quarter of an hour. Palpitations of heart and lungs ceased almost instantly. When taken out he seemed better; temperature in rectum 105.5° , pulse 130, but consciousness not improved. Ice applied in a bladder to his head; a soap-and-water enema brought away some solid fæces; took five grains of sulphate of quinine three times at intervals of three hours, but vomited the second and third doses. 11 P.M., temperature in rectum 107° , coma; put into cold bath for fifteen minutes; temperature reduced to 105° ; afterwards, skin seen to be covered by reddish spots. A restless night, slight delirium, but sleep at intervals. Fifth day, 7.30 A.M., quite conscious; temperature in rectum 105.4° ; pulse 140, regular; no physical signs of disease; pupils natural; a few small papulæ and blotches of no very characteristic appearance upon the skin; delirious all the day, talking deliriously, yet, when spoken to, answered well; vomited twice, stuff pumped up almost without effort, and immediately after cold beef-tea. 9 P.M., very excited; temperature in rectum 105.4° ; pulse 180, indistinct, beats run into each other; feet tend to become cold. He was freely sponged all over with ice-cold water; he liked it much, became quite quiet; ice constantly applied.

to head throughout illness. Bad night, no sleep; frequent attacks of delirium, checked three times temporarily by ice-cold sponging; no vomiting. Sixth day, 9 A.M., looks exhausted; pupils very dilated; temperature in rectum 105.2° ; pulse 144, beat much more distinct; bowels not open; eruption mentioned yesterday has become petechial all over the trunk, looks like flea-bites, no fresh spots; complains much of pain in left thigh, but absolutely no signs of periostitis, phlebitis, arthritis, or any other disease there; lungs and heart natural. Put into cold bath of 68° for about ten minutes; he was much quieted, but the rectum temperature fell to 104.8° only. He is and has been kept in a dark and quiet room. He is now fed with milk and beef essence every half hour unless asleep, it being necessary to give him small quantities at a time so as not to provoke vomiting; all food ice cold. Brandy has been given in small quantities, but it always increases the excitement; the quinine he cannot keep on his stomach; ordered potassii iodidi gr. iij, potassii bromidi gr. x, e syrupo aurantii, quartis horis. Delirium all day, restlessness, no sleep; sponged twice with icy water, and soothed thereby. 9 P.M., very weak and exhausted; marked holding back of the head and stiffness about the neck for the last twenty-four hours; temperature in rectum 106° and more; body feels very hot, but feet are cold; whole skin livid; white mark left by pressure of finger, is very slow in disappearing; a cold wet sheet wrapped round the trunk, the limbs being kept warm; ice to head continued; ten grains of chloral hydrate given; much quieter after this for three or four hours, but doubtful if any real sleep; then became very restless again, and chloral repeated with a good effect for two hours. Seventh day, 9 A.M., half comatose; pupils dilated; rectum temperature 106.2° ; pulse 160, very weak, regular; breathing regular; champagne and brandy freely given did not now excite; carbonate of ammonia and compound tincture of cinchona given every two hours. 9 P.M., still lower; deep coma, yet swallows pretty well; limbs warmer than they were; skin less livid; tache cérébrale for first time; no more eruption, that which came out on the fourth day is fading; eyes half open, right pupil much smaller than left; rectum temperature 106° ; pulse and breathing regular; chest carefully examined, absolutely no physical signs of disease; no opisthotonos. Eighth day, soon after midnight between seventh and eighth day, began to sink rapidly, in spite of taking food, stimulant, and medicine regularly; pulse became more rapid and weak, yet regular; breathing became shallower; limbs cold; skin dusky; pupils remained unequally dilated, the larger one insensible to light. Died quietly at 3 A.M. No otitis at any time.

Summary of Symptoms.

	Onset.	Rise.	Height.	Fall.
L.S.	Day 1. Vomiting and diarrhœa Convulsions	Days 2, 3, 4. Diarrhœa	Days 5 and 6. Ardent fever Convulsions Coma Bilious diarrhœa	Days 7, &c. Development greatly arrested, in respect of mind and voluntary motion
R.M.	Day 1. Vomiting Chilliness and fever Lassitude and headache		Days 2, 3, 4, 5, 6. Fever [pation Vomiting and consti- Delirium and croci- dismus Sleeplessness Coma vigil Irregular pulse Unequal pupils, squint Cervical opisthotonos	Days 7, &c. General paraly- sis, temporary Squint { per- Deafness { man- ent
E.C.	2 A.M. Vomiting Shivering and fever	2 A.M. to 11 A.M. Vomiting and diarrhœa	11 A.M. to 9 P.M. Fever Lipyria Delirium and croci- dismus Convulsions Coma	Death
W.P.	Day 1. Chilliness Lassitude	Days 2 and 3. Fever Lassitude	Days 4, 5, 6, 7, 8. Ardent fever Lipyria Vomiting and consti- Delirium [pation Sleeplessness Coma Palpitations of limbs Throbbing of heart and carotids	Death

VI. *Analysis of the cases.*

After writing out my notes, I turned to the 'Epidemics' of Hippocrates, Books I. and III. There I found cases resembling mine as closely as cases could. Causus and phrenitis are the names given to them, and the commentators have written much to explain the meaning of those terms. A better commentary than my notes of cases could not be written; the diseases of Thasos are illustrated by the diseases of London. To seek to make the facts of Hippocrates tally with the intellectual abstractions of our textbooks and systems of medicine shows ignorance of his method. His causus and phrenitis are merely the names of symptoms common to many diseases. For the convenience of the reader I will transcribe the part of his general description which most concerns us here:—'About the equinox, and until the season of the

Pleiades, and at the approach of winter, many causi set in ; but great numbers at that season were seized with phrenitis, and many died ; a few cases also occurred during the summer. Immediately upon their setting in there were acute fever, small rigors, sleeplessness, thirst, nausea, small sweats about the forehead and clavicles, but no general perspiration. They had much delirious talking, fears, despondency, great coldness of the extremities, in the feet, but more especially in the hands. The paroxysms were on the even days ; and, in most cases, on the fourth day the most violent pains set in, with sweats, mostly coldish ; and the extremities could not be warmed, but were livid and rather cold ; and they had then no thirst ; in them the urine was black (dark, high-coloured?), scanty, and thin ; and the bowels were constipated. There was a hæmorrhage from the nose in no case in which these symptoms occurred, but merely a trifling epistaxis. And none of them had a relapse, but they died on the sixth day, with sweats. In the phrenitic cases, all the symptoms which have been described did not occur, but in them the disease mostly came to a crisis on the eleventh day, and in some on the twentieth. In those cases in which the phrenitis did not begin immediately, but about the third or fourth day, the disease was moderate at the commencement, but assumed a violent character about the seventh day.¹ It is remarkable that nearly all the histories which Hippocrates narrates in the first and third books of 'Epidemics' are instances of phrenitis ; and this combination of symptoms (high fever and delirium) seems to be the chief bond of resemblance between his cases, which are in many other respects dissimilar enough, being examples of pestilential fevers, puerperal fevers, pneumonia, erysipelas, and so on. I will transcribe his shortest history, which is a fair representative of the rest:—'The phrenitic patient, having taken to bed on the first day, vomited largely of verdigris-green and thin stuff ; fever, accompanied with rigors ; copious and continued sweats all over ; heaviness of head and neck, with pain ; urine thin, substances floating in the urine small, scattered, not subsiding ; copious dejections from the bowels ; very delirious ; sleeplessness. On the second day, in the morning, comatose (*ἄφρωνος*) acute fever ; sweats ; fever did not leave him ; palpitations (*πάλμοι*) over the whole body ; at night convulsions. On the third all the symptoms exacerbated, and he died.'²

1. *The fever.* In L. S. the axillary temperature rose to 108·5°; in W. P. the bowel temperature reached 107·4°; in E. C. the

¹ Epidemics, I. constit. iii.

² Epidemics, III. constit. ii., case 4.

axillary temperature was 105° , when the surface was cold ; in R. M. the fever was high. The temperature of the rectum should be taken, whenever possible, on account of the great tendency to coldness of the limbs and parts exposed.

If we reserve the name of ardent fever for fevers in which the bowel temperature rises to 106° or more, the fever of phrenitis is certainly often, if not mostly, ardent in this sense. Hippocrates constantly speaks of ardent fever and phrenitis together. Some pathologists have wished to prove that the delirium is directly due to the high temperature, a doctrine in which there may be some truth, but by no means the whole. We cannot lay down the aphorism, Ardent fever causes delirium : or this, Delirium, in fever, is due to high pyrexia. Something more than the fever is needed to cause the delirium. A boy, eight years and nine months old, with a temperature of 107.6° on the seventh day of typhoid fever, was not delirious ; he lay calm, quite clear in his mind, saying he felt very hot ; not flushed, but pale ; skin moist.¹

The relative heat of the head should be noted in all cases. For 'where is the heat in the body, there is the disease.'² In R. M. there was great heat of head, whilst the limbs were quite cool, and the body also was cool in parts exposed. In W. P. the heat of the head was a very marked symptom ; water poured on his head ran off quite hot. 'Heat of head great, and greater than the rest of the body, is a very bad precursor of delirium, coma, convulsion.'³

2. *Delirium and phrenitis*.—Fever and delirium : this is phrenitis. In the further diagnosis we have to distinguish—(1) the accidental fevers, such as scarlet fever, typhoid fever, hydrophobia ; (2) the constitutional fevers, such as rheumatic fever, and the ephemera of children ; (3) fevers such as septhæmia, erysipelas, icterus gravis ; (4) local inflammations, especially meningitis, pneumonia, pericarditis. When all these diseases are eliminated, there will remain a residue of cases, well exemplified by three of those which I have narrated. In R. M. and W. P. the delirium was great ; in E. C. it was very brief, and soon followed by coma. L. S. was not delirious ; and so I have done wrong to classify her case under the head of phrenitis, strictly speaking. My reasons for doing so are these. It seems as if infants do not suffer from delirium. It seems as if delirium (in infants especially)

¹ The whole course of this boy's fever is given in the Hospital Reports, vol. x. p. 6, table II ; he was not delirious at any time.

² Hippoc. Aphor. iv. 39.

³ Stoll. Aphor. 690.

may be replaced by convulsions: for instance, in purulent meningitis the fever is attended sometimes by delirium, sometimes by convulsions, and both delirium and convulsions end in coma.¹

3. *Coma and lethargus*.—Fever and coma: this is lethargus. Phrenitis, which cannot be subdued, ends in lethargus (phrenitis comatosa). The lethargus cannot be deemed a mere exhaustion, consequent upon the excitement of the phrenitis. In E. C. the delirium was very slight and short, yet was followed by the deepest and fatal coma. W. P. became lethargic on the fourth day, without foregoing delirium or convulsions: return of consciousness, on the fifth day, was soon followed by delirium, to which ensued a second and fatal lethargy.

The lethargus of heat stroke is sometimes quite protopathic. I have elsewhere published the account of a young man, who was brought to the hospital on July 22, 1868. The weather was very hot; the thermometer marked 90° in the shade of the Royal Exchange. He was wheeling a barrow in the sun, when he staggered and fell. Brought instantly to the hospital in a cab; his state shortly after admission was this: comatose, livid, universal purple mottling of skin not unlike typhus rash, loud pulmonary systolic murmur, temperature in axilla 109·5°. He died within an hour from his attack. The rash disappeared after death.

In R. M. there was, for a time, coma vigil; the child lay with his eyes open as if in a meditative state, yet he was unconscious.

4. *Convulsions*.—In L. S. the vomiting which marked the onset of the disease was followed, in a few hours, by convulsions; they then ceased, to return on the fifth day. In E. C. the vomiting of the onset was followed, in ten hours, by convulsions. There were no convulsions in R. M. and W. P., although the latter boy had palpitations of the body, which seemed almost convulsive.

In both L. S. and E. C. the convulsions were attended by high fever; in the former child, these symptoms were subdued by the treatment; in the latter they rapidly passed into a mortal lethargy. It would be well if there were a single word to express this combination of fever and convulsions. In all cases of convulsions it is most necessary to study the body-heat. Febrile convulsions stand apart from the non-febrile, in respect both of prognosis and of treatment. Convulsions kill by apnoea or by ardent fever; we must be ready to meet either of these dangers. Hippocrates says that 'convulsion following fever is dangerous,

¹ St. Bartholomew's Hospital Reports, vol. viii. pp. 25 *et seqq.*

but least so to children;'¹ and again, 'it is better that fever should follow convulsion, than convulsion fever.'² In discussing this subject we must keep close to matter of fact; it is a question, not of aetiology, but of mere sequence in point of time; not whether fever causes convulsion or convulsion fever, but whether one symptom distinctly precedes the other. Hippocrates speaks of two possible cases, and there is yet a third: i. the patient is in a febrile state, and convulsion supervenes; ii. the patient is in a convulsive state, and fever supervenes; iii. fever and convulsion exactly concur in their time of onset.

i. The febrile state precedes convulsion. The convulsions forbode evil. For instance, in the first three or four days of scarlet fever, convulsions and phrenitis, supervening upon the febrile state, not attending its onset; in confirmed typhoid fever; in meningitis, or other febrile brain disease. In whooping cough the case is not quite so simple; convulsions often supervene in the third or fourth week or later, and the child recovers. But whooping cough is not a febrile disease throughout; febrile whooping cough usually means whooping cough with severe pulmonary catarrh, and in that case, intercurrent convulsions are most dangerous.

ii. The convulsive state precedes fever. It is in these circumstances that the famous aphorism may be applied. 'Fever, supervening upon spasmodic diseases, ends them.'³ And the truth of this doctrine needs no support from me.

iii. The fever and convulsions concur in their time of onset. In this case the prognosis is favourable, whether the incipient fever be smallpox, measles, scarlet fever, chicken pox, mumps, catarrhal fever, pneumonia, or gastro-enteritis; the last being by far the most common accompaniment of the convulsions. The febrile disease may however be fatal, in itself; for instance, convulsions may attend the onset of tubercular meningitis.

In the course of a febrile disease (or rather, a sub-febrile disease, accompanied by slight fever only), the onset of an intercurrent fever may be attended by convulsions, and the prognosis still be favourable. For instance, in scarlatinal nephritis, a sharp attack of gastro-enteritis or pneumonia may be marked by convulsions and great exacerbation of fever, yet the child recovers. Indeed many of the convulsions occurring in scarlatinal nephritis are of this kind; they attend the onset of a local inflammation, especially pleurisy, and it is chiefly because this inflammatory disease is fatal, that the children die.

But even in this concurrent attack of fever and convulsions, happening to a patient who was not febrile before, there is always

¹ Coac. Prænot., 356 (Foes).

² Aphor. ii. 26.

³ Coac. Prænot., 354 (Foes).

the possible danger of ardent fever, which is mortal if not subdued.

This third kind of convulsion, that which attends the effervescence of fever, might be aptly called a convulsive rigor.

5. *Rigors*.—The rigors of phrenitis are, as Hippocrates says, small, and small in spite of the great rise of temperature. All the three children, who were old enough to complain, complained of chilliness.

6. *Coldness of extremities and lipyria*.—A marked symptom of the Hippocratic phrenitis is ‘coldness of the extremities, in the feet, but more especially in the hands;’ again, ‘extremities altogether cold, and could not be warmed, livid;’ again, ‘extremities could no longer be kept warm;’ and again, ‘extremities cold and somewhat livid.’ Coldness of the extremities with fever is lipyria. When the fever is high, lipyria is a most dangerous symptom. ‘In acute diseases, coldness of the extremities is bad.’¹

In R. M. there was ‘great heat of head, limbs quite cool, body also cool in parts exposed;’ and again, ‘head still becomes hot sometimes, but feet tend to become cold, body not so;’ yet this patient recovered. In E. C., temperature in armpit, 105°; feet and hands cold, any part of body becomes cold on exposure. In W. P., temperature in rectum, 106°; body feels very hot, but feet are cold.

7. *Lividity*.—Lividity accompanies the lipyria: as Hippocrates says, ‘the extremities were livid and rather cold.’ In L. S., ‘skin purple and sweating.’ In E. C., ‘skin everywhere injected, mottled, livid; lips livid, but rubbing them gently makes them florid.’ In W. P., ‘whole skin livid; white mark left by pressure of finger is very slow in disappearing.’

8. *Pain in the thigh*.—‘In fever an attack of pain in the thigh is bad.’² This pain in the thigh was an early and a marked symptom in the case of W. P. On the first day of his illness he complained of much pain in the left thigh; on the fourth day there was no pain; on the sixth day complained of much pain in left thigh, but positively no signs of periostitis, phlebitis, arthritis, embolus, or any other disease there.

I have noted the same symptom early in typhoid fever.

B., a boy, 15 years old, on the first day of his illness said he did not feel well; on the third day, headache; on the fourth day, headache and bilious vomiting; on the fifth day, delirium, and began to complain of left thigh; on the ninth day diarrhœa

¹ Hipp. Aphor., vii. 1.

² Coac. Prænot., 30.

began; no rigors at any time. On the tenth day I saw him with Dr. Smiles. Stupor; constant delirium; axillary temperature, 104° ; pulse 130, regular; meteorism; spleen impalpable; no spots; a little rale at top of right lung; complains greatly when left thigh is touched; left leg strongly everted; position recalls hip disease; no swelling; very careful movement does not cause pain. The question of acute periostitis, arthritis, embolus, or thrombosis, with secondary pyæmia, arose; but it seemed to be set aside by the consideration that, if the general symptoms were due to the leg, the signs of disease there would have been more marked: moreover, the disease did not begin with complaints concerning the leg. However, in order to make no mistake, we summoned Mr. Howard Marsh to our help. He gave a decisive opinion that there was no discoverable disease in the leg. A few hours later, the limb had lost the position described. A loose, light yellow stool was passed. There was no doubt that the disease was typhoid fever; and the marked meteorism and constant delirium so early as the tenth day made our prognosis unfavourable. On the twelfth day he passed into a lethargy and died.

This early pain in the thigh would seem to be a condition different from that which sometimes happens at the end of typhus, typhoid fever, and peripneumonia, and which Graves has illustrated well.¹ Whether the patients described by Hippocrates² and Van Swieten³ suffered from the same kind of disease I will not take upon myself to say.

9. *Jactation*.—In W. P., on the fourth day, frequent jumpings of the limbs and trunk, palpitations, or half-convulsive movements, were noted. Hippocrates, in his phrenitic patient, mentions ‘palmi over the whole body’ on the second day. Graves, narrating a case of typhus phrenitis, says ‘his body was agitated from head to foot by continual tremors.’⁴

10. *Throbbing of heart and arteries*.—Very strong throbbing of the heart and carotid arteries was noted in W. P. on the fourth day. One of the most marked effects of the cold bath was the removal of this symptom. It is sometimes very striking in meningitis. Hippocrates speaks of ‘constant palmus in the hypochondrium,’ and ‘continued palmus in the epigastrium throughout,’ in two of his cases.

11. *Vomiting*.—In three of my cases vomiting was one of the earliest symptoms on the first day. In the fourth case (of W. P.)

¹ Clinical Medicine, vol. i. lect. 19.

² Epidemics, book iii. case 5.

³ Comment. in Aphor., 772.

⁴ Clinical Medicine, vol. i. lect. 19.

it happened on the fifth day; the stuff pumped up from his mouth almost without an effort. In E. C. the vomiting was repeated at intervals, and soon became bilious. This symptom is noted by Hippocrates in the typical case which I have quoted. The remarks of Van Swieten¹ and Graves² upon the bilious vomiting of phrenitis are well worth study.

12. *Diarrhœa and Constipation*.—The bowels were spontaneously relaxed in L. S. and E. C. There was a tendency to constipation in R. M. and W. P. I have no instance of the copious bilious diarrhœa spoken of by Hippocrates and Graves.

13. *Crocidismus*.—‘When in acute fevers, peripneumonia, phrenitis, or cephalalgia, the hands are waved before the face, hunting through empty space, as if gathering bits of straw, picking the nap from the coverlet, or tearing chaff from the wall: all such symptoms are bad and deadly.’³ Yet in R. M. these symptoms were well marked on the fifth day, and he recovered. In E. C. they were bad and deadly.

14. *Sleeplessness*.—Sleeplessness was a marked symptom in many of Hippocrates’ patients; also in R. M., but especially in W. P. And the worst of it is that delirium takes the place of sleep. ‘When sleep puts an end to delirium it is good.’⁴ The first favourable symptom in R. M., who recovered, was that he slept much on the sixth day. ‘Deep untroubled sleep denotes a crisis upon which we may depend.’⁵ In E. C. sleep was followed by delirium, and she soon died.

15. *Rash*.—The body of W. P., on the night of the fourth day, after a second cold bath, was seen by Mr. Thorne to be covered with rose spots, much like those of typhoid fever. On the morning of the fifth day a few small papulæ and blotches of no very characteristic appearance were all that was left. On the sixth day the spots had become petechial, and looked like flea-bites. On the next day they began to fade; there was no fresh eruption.

16. Symptoms of an affection of the base of the brain and cervical cord were marked in R. M. on the fifth day—namely, pulse infrequent and irregular; unequal pupils; internal squint of right eye; deafness; cervical opisthotonos. All these symptoms

¹ Comment. in Aphor., 774.

² Hippocr. Prognostics, 4.

³ Coac. Prænot., 251 (Foes).

⁴ Clinical Medicine, vol. i. lecture 13.

⁵ Hippocr. Aphor., ii. 2.

were slow to disappear. The squint, infrequent and irregular pulse, and deafness persisted three years after the attack. In W. P. the only symptoms of this kind were stiffness of the neck on the sixth day, and unequal pupils on the seventh day, a few hours before death.

17. *Tache cérébrale*.—In L. S., on the seventeenth day, there was a brain streak. In R. M. there was none on the fifth day. In W. P. there was none until the evening of the seventh day, a few hours before death, when it was noted for the first time.

18. *The interval between the onset of the disease and severe cerebral symptoms*.—Hippocrates speaks of cases 'in which the phrenitis did not begin at once, but about the third or fourth day.' In L. S. the invasion symptoms of convulsions, vomiting, and diarrhœa were followed on the second, third, and fourth days by an interval marked by a little diarrhœa as the only symptom; on the fifth day convulsions returned with ardent fever. In R. M. there was a steady progress of symptoms. In E. C. there was no definite interval. W. P., for two days after the onset, seemed simply poorly, with feverishness and lassitude.

19. *Duration*.—The acute stage of the disease of L. S. came to an end on the sixth day; of R. M. also on the sixth day. E. C. died on the first day; W. P. on the eighth day. 'Phrenitis vera kills on the third, fourth, or seventh days, but seldom exceeds the last.'¹

20. *Sequelæ*.—The two children who escaped with life did not completely recover. L. S., one year after her illness, was in a state which may be best expressed by the word dementia. But I do not deem her dementia necessarily permanent; in fact, at the time of writing this paper she is improving. R. M. was left absolutely and permanently deaf; and three years after his illness there were other and slighter sequelæ, squint, an infrequent and irregular pulse, and some unsteadiness on the legs. I will illustrate this point by two more cases, although the details of them are not so full as could be wished.

H. R., a boy aged 3½ years, was in perfectly good health until, at the beginning of May, he vomited one night. First day, vomited, slept much. Second day, twitching of right side; he had become, in his mother's words, 'a perfect idiot.' Third day, delirious, convulsed for three or four hours. For the next fortnight he 'could not see, and took no notice.' Six weeks

¹ Boerhaave, Aphor., 774.

from the onset of his illness I saw him; has been able to play about for four weeks; has sense enough to be clean in his habits; well nourished; no otorrhœa, stiffness of neck, or strabismus; seems quite deaf; ophthalmoscopic examination impossible on account of the great restlessness. Nine weeks from beginning, very restless; does not look very idiotic; less clean than he was; probably deaf; speaks a great deal, but what he says cannot be understood. Tenth week, much the same.

L. O., a girl aged $2\frac{1}{4}$ years, was quite well until March 25, when she vomited. Second day, convulsions, which lasted on and off for a day or two. When convulsions ceased she was left unconscious; the unconsciousness lasted for six weeks, gradually becoming less. Fever was noted during the first day or two of her illness. Never had otitis. When she became conscious again, her speech was so bad that she could not be understood; she could speak well before. Weak in all limbs, compared by Mr. Herbert Taylor of Kennington to the paralytic state sometimes left by chorea; she lolled about on all sides when she was raised. Ten months after the onset of her illness, general paralysis; walks feebly, like a child just beginning to walk; arms clumsy and weak; no muscular atrophy; mind seems right, but she is less lively than before her illness; speaks distinctly; subject to nightmare; internal squint of right eye for three months past; clean in habits; no headache or vomiting; eyes natural to ophthalmoscope; heart natural. Six weeks later she was much the same.

The summary of these two cases is as follows: In H. R. a sudden attack of vomiting, followed first by delirium and convulsions, and then by coma; when coma disappeared, dementia and deafness left. In L. O. a sudden attack of vomiting and fever, followed first by convulsions, and then by coma; when coma disappeared, general paralysis left.

VII. Treatment.

i. *Cold*.—The ardent fever suggests the use of cold. But it seems as if ardent fever alone were an insufficient indication; we must distinguish our cases according to the chief accompanying nervous symptom. The ardent fever may be attended by—

i. *Convulsions*.—The case of L. S. will show that this combination of symptoms is a trustworthy indication for the cold bath. Her life was saved by it.

ii. *Delirium*.—W. P. was always relieved by the use of cold; in particular the violent palpitations of the heart, the palmar of the body, and the early coma seemed to be removed by the bath. Yet it was powerless to save his life. It is true that we did not

act up to Dr. Wilson Fox's rule of treatment, that 'a bath can hardly be considered to have produced its full effect unless, subsequently to the patient's removal from it, the temperature falls nearly to the normal range, and even below this;' ¹ but we did the most we could under the circumstances. And at least the temperature was kept down to a point by no means dangerous in itself.

iii. *Coma*.—The case of E. C. was not one of mere lethargus; there was yet another symptom to be considered with respect to the use of cold, and that was the distribution of the body heat in her. It would be hard to believe that lipyrria does not contraindicate the cold bath. According to the sentence of Currie, 'the cold affusion is not to be used when the extremities feel cold, whatever may be the heat of the central parts.' ² The utmost that can be attempted is to apply cold to the trunk, whilst the extremities are kept warm, which was done in the case of W. P. with as good a result as could be expected in a condition so desperate.

2. *Vital sedatives*.—Facts concerning other means of treatment I have none to give. I should be glad to know more concerning the merits of those sedatives to the vital constitution which were formerly so much in use, especially blood-letting and emetic tartar. I would refer the reader to the lectures given by Graves ³ on the employment of tartar emetic in the phrenitis of typhus.

¹ On the Treatment of Hyperpyrexia, as illustrated in Acute Articular Rheumatism, by means of the External Application of Cold, p. 43.

² Medical Reports on the Effects of Water, Cold and Warm, as a Remedy in Fever and Febrile Diseases, whether applied to the Surface of the Body or used Internally, vol. i. p. 14.

³ Clinical Medicine, vol. i. lectures 14, 15, and 16.

AN EXAMINATION
OF THE
OPINIONS HELD AS TO THE CAUSES
OF JAUNDICE.

BY
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Physicians have been long aware of the existence of two kinds of jaundice: one attended by marked disease of the liver and gall-ducts; the other with no lack of colour in the fæces and bile-passages, and no readily-discovered disease of the liver. The former, since it was set upon a sure experimental footing by Saunders¹ towards the end of the last century, has always been of easy explanation. The latter, however, has given rise to many theories, and to many violent modes of explaining facts hard to explain; and to this day a satisfactory solution of all the phænomena belonging to this kind of jaundice is still wanting. It is my intention in this paper to attempt to discuss the opinions held as to the various causes of jaundice in our own and earlier times, and to arrive at some opinion as to their trustworthiness in being applied in practice in the treatment of the disease.

Although the divers kinds of bile held so important a place in the medicine of Hippocrates, yet he does not seem to have conceived any relation between bile and the symptom of jaundice. Galen says, on the other hand, that the yellow bile, when it is carried all over the body, still keeping its own nature, brings forth jaundice.²

¹ Saunders, *A Treatise on the Structure, Economy, and Diseases of the Liver*. London. 1793.

² Galen, *De Med. Method. lib. i.* Kühn's ed., Lips., 1826, vol. xi. p. 74.

Aretæus says much the same thing, adding, however, that jaundice may arise from the stomach, spleen, kidneys, and colon, and is not solely formed from the liver, as some have supposed.¹ The jaundice caused by the liver is brought about in this fashion: if the liver continue to secrete bile, but the passages which convey the bile to the intestine be obstructed, the bile regurgitates; it therefore becomes mixed with the blood, and the blood passing over the whole system carries the bile into every part of the body, which acquires the appearance of bile. It would be hard to express in different language the doctrine of jaundice from absorption, now universally held. Much the same views are expressed by many other Greek, Latin, and Arabic authors,² some of whom, moreover, believe that a plugging of the cystic duct is enough to cause jaundice, in accordance with the physiology of the time.

It cannot be looked for that Paracelsus or Van Helmont should bring much that is valuable to the explanation of bilious diseases. Paracelsus looked upon the bile as a useless, unnecessary, and superfluous element in the animal economy. He could not conceive that an uncleanly flux of such base birth could bring disease, and he therefore passes by all bilious diseases in silence. Just the opposite view was taken by Van Helmont. He thought the bile an invaluable secretion, formed out of the best blood, the balsam of life. It was impossible, of course, that so noble a fluid should be the source of disease.³

Sylvius de le Boe denies that obstruction in the bile-ducts is an efficient cause of jaundice.⁴ He points out that in some cases of jaundice which he has dissected he had found no obstruction; and further, that in some cases the stools were coloured, or but little less so than usual. With this writer a most important age, not merely in the history of jaundice, but in that of medicine at large, is reached. It is the time of the discovery of the circulation by Harvey, of the lacteals by Aselli, and of the thoracic duct by Pecquet. With the changes in physiology, changes in pathology likewise appear. The history of jaundice must now be broken up into three heads: (i.) A theory which attributes the symptom of jaundice to changes in the blood and its colouring matter, thence called hæmatogenous jaundice. (ii.) A theory which makes the

¹ Aretæus, *On the Causes and Symptoms of Chronic Diseases*, book i. chap. xv., Adams' Translation, p. 324.

² For a general view of the opinions of these authors the reader may consult Adams' Translation of Paul of Ægina, vol. i. p. 582.

³ For this account of the views of Paracelsus and Van Helmont I am indebted to Eisenmann (*Die Krankheits-Familie Cholosis*, Erlangen, 1836, p. 13). I had not the patience to search in the writings of these authors for their views.

⁴ Sylvius de le Boe, *Opera Medica*, Amstelodami, 1679, p. 301.

bile to be merely separated by the liver from existing elements in the blood, not truly secreted by the liver. Jaundice may thus arise when the liver ceases to excrete the bile, the elements of which accumulate in the blood and cause the general yellow appearance. (iii.) A theory which attributes the phenomena of jaundice to the absorption into the mass of the blood of bile already formed. Under these three heads all prevailing doctrines of jaundice may be discussed.

I. The notion of a hæmatogenous jaundice is most certainly to be found in Aretæus. He speaks of divers causes of jaundice, not only from the liver, but from the stomach, the spleen, the kidneys, and the colon. But besides jaundice from the viscera, 'the general system is most powerful in producing icterus;' and 'if indigestion happen in the blood, the blood assumes the appearance of bile, but is distributed as nourishment to all parts, wherefore bile appears everywhere.'¹ These ideas do not seem to have taken much root among physicians, for there seems to be no mention² of the theory of hæmatogenous jaundice until Bianchi again distinctly enunciated it.³ This writer speaks of two kinds of jaundice: one caused by disease of the liver; the other by a solution of the blood, in which the motions do not become white, but are rather more deeply coloured. It is more quickly brought about than the other kind of jaundice, and is seen after the bites of vipers. This view is shared by certain obscure writers upon jaundice and bilious diseases.⁴ It was upheld for a time by Reil, who under the name of polycholia gave a long description of what is now called hæmatogenous jaundice, and rules for distinguishing it from true jaundice.⁵ The opinion was resisted by Cullen, who says that jaundice must be the result of bile, once

¹ Aretæus, *op. cit.*, p. 326. Leo likewise says that jaundice may arise from the conversion of blood into bile from heat of the system. (Quoted by Adams in his Translation of Paulus Æginetus, vol. i. p. 584.)

² I have been unable to find any trace of this doctrine in the writings of Sydenham. One passage (*Opera Omnia*, ed. Greenhill, p. 170) is thought by Eisenmann to have a faint likeness to this teaching. To me it seems merely the explanation of a kind of dysentery by invoking a disease of the blood, the hot and acrid humours of which are thrown upon the gut by the mesenteric arteries. Nor can I find anything in Baillou's chapter on the epidemics of 1575 which would remind the reader of the theory. (*Epidémies et Éphémérides*, Paris, 1858. Translated by Yvareu, p. 278.)

³ Bianchi, *Historia Hepatica*, Genevæ, 1725, 3tia ed. t. i. pars ii. cap. x. p. 185.

⁴ See Frerichs, *Klinik d. Leberkrankheiten*, Braunschweig, 1858, Bd. i. p. 82.

⁵ Reil, *Tractatus de Polycholia*, Halæ, 1782, p. 47. He withdrew the opinions expressed in this 'juvenile opus,' as he calls it, a few years later, and says that no bile, nor anything like bile, is made save by the help of the liver. (*Memorabil. Clinic.*, fasc. iv., Halæ, 1795, p. 48.)

secreted, being taken up into the blood-vessels,¹ by Donald Monro,² Eller,³ and Selle;⁴ they all plainly teach that the doctrine of jaundice from absorption is the only trustworthy theory.

No further important support is met with until the time of Saunders, who speaks of the experiments of Fourcroy and Vauquelin.⁵ These observers made a sort of artificial bile by heating together ox-blood and water. Saunders then adds, 'It would appear probable that, under certain morbid states of the body, the blood *may* acquire a bilious appearance independent of absorption or regurgitation from the liver.'⁶ It is worthy of note that the man who was the first to prove by experiment the existence of jaundice from absorption should likewise have given the weight of his authority to the doctrine of a hæmatogenous jaundice.

One of the next notices of a jaundice arising from changes in the blood, without any implication of the liver, may be found in the writings of Breschet. After pointing out the near relation which exists between melanoses and the colouring matter of the blood, he says that he presumes that jaundice is brought about much less by the bile than by the blood;⁷ and he attributes the jaundice so common in the new born to changes in the circulation rather than in the liver. Later on Virchow gave a great impulse to this notion. He made known the likeness⁸ between hæmatoidin and bile pigment both in crystallisation and chemical reaction.⁹ The identity of these two substances was thought to have been proved by Zenker and Funke,¹⁰ Valentiner,¹¹ and later still by Jaffé.¹² But Städeler¹³ and Holm¹⁴ deny

¹ Cullen, *First Lines of the Practice of Physic*, § 1817.

² Donald Monro, *An Account of the Diseases which were most frequent in the British Military Hospitals in Germany*; London, 1764, p. 206.

³ Eller, *Obs. de Cog. et Cur. Morb., Venetiis*, 1767, sect. ix. p. 177.

⁴ Selle, *De Curand. Hom. Morb., Berolini*, 1798, ed. Sprengel, p. 184.

⁵ Fourcroy and Vauquelin, *Annales de Chimie*, 1790, t. vi. p. 181.

⁶ Saunders, *A Treatise on the Structure, Economy, and Diseases of the Liver*, London, 1803, third edition, p. 105, note.

⁷ Breschet, *Considérations sur une Altération organique appelée Dégénérescence noire, etc.*, Paris, 1821, p. 21. Audral (*Clinique méd.*, Paris, 1839, t. ii. p. 287) would seem, with Breschet, to favour the notion of the colour in the newly born, or in yellow fever, to be due to changes in the circulation, or to a sort of general ecchymosis. The same thought is expressed (t. i. p. 601).

⁸ In the old Chinese system of physiology the reverse was believed; the liver was the source of all the pigments in the body.

⁹ Virchow, *Arch. f. path. Anat.*, Bd. i. 1847, p. 379. See also *Ges. Abh.*, Hamm, 1862, p. 849.

¹⁰ Funke, *Lehrb. d. Phys.*, Leipzig, 1860, Bd. i. p. 246.

¹¹ Valentiner, quoted by Steiner, *Arch. f. Anat. und Phys.*, 1873, p. 163.

¹² Jaffé, *Arch. f. path. Anat.*, Bd. xxiii. 1862, p. 192. He obtained bilirubin from an old hæmorrhage into the brain.

¹³ Städeler, *Annal. d. Chemie und Pharm.*, Bd. cxxxii. 1863, p. 323.

¹⁴ Holm, *Zeitschrift f. rat. Med.*, Bd. xxxii. Bericht f. 1867, p. 314.

this, and give rules by means of which hæmatoidin may be distinguished from bilirubin. Salkowski, on the other hand, says that a substance with many of the characters of hæmatoidin, which he found in the thyroid gland, and which must therefore have had its origin in the blood, was insoluble in æther and soluble in alkalies, which Holm and Städeler assert to be characteristic of bilirubin.¹ Thudichum, however, would affirm that the body which all these observers analysed was neither bilirubin nor hæmatoidin, but leucein,² the hæmolutein of Piccolo and Lieben.³ Thudichum further protests against the idea of the bile pigments being derived from hæmatoidin because they contain no iron.⁴ There is, however, a hæmatin free from iron, which is asserted to be polymerous with bilirubin; yet it seems strange that the hæmoglobin should so readily part with its iron. Dr. Young found, in some careful experiments under Gamgee's direction, that the ash of the bile was rich in iron,⁵ and this fact is also worthy of being kept in mind.

The great hindrance, to my mind, to receiving the belief that the bile pigments descend from hæmoglobin is the fact that, in animals with white blood, coloured bile is still found. Still it is impossible to deny that the two bodies are near akin; and the theory of the source of the bile pigments from hæmoglobin and of the colouring matters of the urine from the bile pigment is very plausible, and no one can wonder at the great support that it receives.

The notion of the identity of bilirubin and hæmatoidin being firmly set up in the minds of physiological chemists, it seemed no strange statement that the bile pigments should appear in the urine when any large number of the blood-corpuses were dissolved. This indeed is what Frerichs found after the injection of the bile acids into the blood, a fact explained by him in altogether another way.⁶ Kühne likewise upheld Frerichs' belief, and added a new fact, that the bile pigments appear in the urine after the injection of hæmoglobin into the circulation.⁷ M.

¹ Salkowski, *Med. Chem. Untersuch.*, 1868, Heft iii. p. 436, Herg. von Hoppe-Seyler. Hoppe-Seyler found bilirubin in a cyst of the breast (*Arch. f. path. Anat.*, 1862, Bd. xxiv. p. 10).

² Thudichum, *Proc. of the Royal Society of London*, 1869, vol. xvii. p. 253.

³ Piccolo and Lieben, *Giornale di Scienze naturali ed economiche*, Palermo, ii. 1866. Quoted in *Zeitschrift f. rat. Med. Bericht f.* 1868, p. 226.

⁴ Thudichum, *Zeitschrift f. rat. Med. Bericht f.* 1868, p. 223.

⁵ P. A. Young, *Journal of Anatomy and Physiology*, vol. v. 1871, p. 158. Robin (*Comptes rendus*, t. xli. 1855, p. 506) found iron in the ash of the red matter found in echinococci cysts in the liver; about '0002 per cent.

⁶ Frerichs, *Klinik d. Leberkrankheiten*, Braunschweig, 1858, Bd. i. p. 405. See also *Arch. f. Anat. und Phys.*, 1856, p. 59, note.

⁷ Kühne, *Arch. f. path. Anat.*, 1858, Bd. xiv. p. 337. See also *Lehrb. d. phys. Chemie*, Leipzig, 1866, p. 89.

Herrmann found them after the injection of large quantities of water into the veins;¹ Nothnagel, after the injection of æther and chloroform;² Munk and Leyden, after the injection of phosphoric acid:³ all these bodies having the power of dissolving the red blood-corpuscles and thus setting the hæmoglobin free in the blood-vessels.

The theory of hæmatogenous jaundice seemed thus to receive great support from experiment.

But it was assumed with too great haste that if the colouring matter of the bile were present in the urine that jaundice was thus of necessity at hand. It is indeed true that the urine is often the first to give notice of a coming attack of jaundice, even before the conjunctiva; but it is well to point out with Virchow⁴ that in none of these experiments has a real jaundice been brought forth. There has never been a yellow staining of the tissues, but only a reaction of bile pigment in the urine; and between these two states there is deep and wide division. No artificial jaundice has been as yet caused by injecting any quantity of bile into the blood,⁵ though it would seem possible that jaundice might follow if substances which destroy the blood-corpuscles were allowed slowly to act upon the economy.

The likelihood of this last observation of Virchow's to be true has of late years somewhat decreased. The foregoing experiments upon which so much stress has been laid have been repeated, and in general no bile pigment has been detected in the urine after the injection of substances which dissolve the blood-corpuscles. Naunyn was the first to repeat these observations,⁶ and he could not succeed in obtaining a reaction peculiar to the bile pigment after the injection of hæmoglobin into the circulation or under the skin. In both cases the urine might contain blood colouring matter, but no bile pigment. Steiner likewise repeated M. Herrmann's observations, and found that no bile pigment was present in the urine after the injection of large quantities of water.⁷ I myself have been unable to detect the presence of bile

¹ M. Herrmann, *De Effectu Sanguinis diluti in Secretionem Urinæ*, Diss. Inaug. Berolin. 1859.

² Nothnagel, *Berliner klin. Wochenschr.*, 1866, p. 31.

³ Munk and Leyden, *Die acute Phosphorvergiftung*, Berlin, 1865, p. 129.

⁴ Virchow, *Arch. f. path. Anat.*, Bd. xxxii. 1865, p. 120.

⁵ Dr. George Harley indeed states that he has succeeded in making a dog jaundiced by injecting the bile of other dogs under the skin. He failed, however, in two other experiments. (*Jaundice: its Pathology and Treatment*, London, 1863, p. 96.)

⁶ Naunyn, *Arch. f. Anat. und Phys.*, 1868, p. 410. Nasse (*Sitzungsberichte d. Marburg. Gesellschaft*, 1875, Nr. 2; quoted in Hayem's *Revue des Sciences méd.*, 1876, t. vii. p. 530) found that the urine gave no reaction with nitric acid after the injection of large quantities of blood into the stomach of dogs.

⁷ Steiner, *ibid.*, 1873, p. 160.

pigment in the urine after the injection into the circulation of the bile acids, notwithstanding the statements to the contrary of Frerichs and Kühne. Furthermore, Naunyn, after the injection of æther under the skin, found well-marked signs of the presence of hæmoglobin in the urine in all cases; but in only one out of four could a certain opinion be given as to the presence of bile pigment.¹

This great difference between observers so trustworthy may, I think, best be explained by the fact that Frerichs, Kühne, and Herrmann seemed to have used for their experiments dogs, in whose urine a substance which gives all the reactions of bile pigment is an almost constant constituent.² Naunyn, Steiner, and myself have used rabbits exclusively for experiment, and in the urine of these animals, unless fasting, bile pigment is not often seen. These later experiments would therefore seem more trustworthy than those of Kühne and Frerichs, and I do not think that their value has been destroyed by Prince Tarchanoff's recent observations; for although after the injection of hæmoglobin into the circulation bile pigment was found in the urine, yet the experiments themselves are not such as to command confidence. In the first place, dogs were used in some cases even without testing the urine beforehand for bile pigment; and further, it is not expressly stated that the animal was not fasting, and most of the experiments lasted over six hours, at the end of which the bile pigment was found.³ Even this observer found no bile pigment after a six hours' narcosis from chloroform.

Although Breschet was the first to found the doctrine of a hæmatogenous jaundice, supported by Andral and more lately by less known names, yet the theory seems to have made little progress in France. The history of hæmatogenous jaundice is almost entirely German. In England the doctrine has made but little way, the other view, that of jaundice from suppression, finding greater favour. It is chiefly invoked to explain the phenomena of icterus gravis. Under this head may be placed acute yellow atrophy of the liver, the jaundice seen in poisoning from phosphorus, in pyæmia and other acute diseases. In this way Leyden has laboured to explain the yellowness of the skin sometimes seen in chlorosis, pyæmia, heart disease, or after anæsthesia from chloroform. In these cases the yellowness of the

¹ Naunyn, *op. cit.*, p. 438.

² But this does not explain the matter throughout. Nothnagel used rabbits in all his experiments with æther and chloroform (*Berliner klin. Wochenschr.*, 1866, p. 31), and Kühne (*Lehrb. der phys. Chemie*, p. 89) expressly says that rabbits must be used in the hæmoglobin-injection experiment on account of the constant presence of the bile-pigment reaction in the urine of dogs.

³ Tarchanoff, *Arch. f. d. ges. Phys.*, 1874, Bd. ix. p. 53.

skin is out of all proportion greater than the bilious appearance of the urine. In jaundice from obstruction the reverse is seen. There is likewise no evidence of the presence of bile acids in the urine in these cases. Further, there is often proof in the cases of icterus gravis of the circulation of some poison in the blood able to destroy the red corpuscles, as in poisoning by phosphorus, yellow fever, and the like. The symptoms of icterus gravis resemble each other in having a tendency to stupor or delirium. In the fatal cases no impediment can be found after death to the flow of bile into the duodenum, the liver itself being free from a jaundiced tint and the fæces containing bile. A far-advanced fatty degeneration of all the glandular and muscular organs is likewise found.¹

I should long hesitate in forming any opinions from the statement that the urine is often less jaundiced than the skin, or that the liver after death shows no jaundiced appearance. Virchow, for instance, was formerly of opinion that a jaundice of the liver preceded a jaundice of the whole body;² but he has of late seen reasons to withdraw this opinion,³ and this admission is of the greater importance since Virchow was undoubtedly the founder in modern times of the doctrine of hæmatogenous jaundice. Nor can I attach much weight to the statement that in cases of slight jaundice the urine is much less jaundiced than the skin. I have often found in examining cases of slight jaundice after death that the serous effusions from the chest and belly gave an intense reaction with nitric acid, while the urine gave but slight indications of the presence of bile pigment with the same reagent. The serous effusions and the urine may, I should think, be looked upon as at least equal in importance for indicating the amount of bile pigment contained in the blood. The cases which I mention have been chiefly those of heart disease.

A point of which much importance has been made by Leyden is the absence of the bile acids in the urine in some cases of jaundice. Naunyn's observations have, however, destroyed all value which may be attached to this opinion, for he has shown that the bile acids may be found in every urine, even in that of perfect health.⁴ They appear to be absent in no cases of jaundice, except at the end of cases of long-continued obstruction to the bile-ducts, as Golowin has pointed out.⁵

It is, indeed, only from the fatal cases that any safe conclusions can be drawn. The fatal cases of which Leyden speaks all belong

¹ Leyden, Beiträge zur Pathologie des Ikterus, Berlin, 1866, p. 6.

² Virchow, Arch. f. path. Anat., 1847, Bd. i. p. 380.

³ Virchow, *ibid.*, 1865, Bd. xxxii. p. 121.

⁴ Naunyn, Arch. f. Anat. und Phys., 1868, p. 430.

⁵ Golowin, Arch. f. path. Anat., 1871, Bd. liii. p. 433.

to the class of icterus gravis. Poisoning by phosphorus would, I think, be accepted as a type of this class. The stools are sometimes coloured, sometimes free from colour. Yet Oskar Wyss¹ and Ebstein² found in the livers of dogs and men poisoned by phosphorus that the finer ducts within the liver were plugged with a colourless mucus which hindered the descent of the bile, a plain cause of jaundice. While this explanation of the jaundice remains available, it seems to me imprudent, to say the least, to search for other and less likely causes elsewhere.

The presence of deeply-coloured stools cannot always be received as proof of the freedom of the common duct. Indeed in jaundice the more deeply coloured they are, the greater suspicion should be aroused. It is well known that many substances given by the mouth make the stools dark, as charcoal, iron, or bismuth. What is more important in the present discussion is that blood passed into the alimentary canal causes the stools to have a very high colour. Now, in icterus gravis hæmorrhages are very common, and especially abundant in the stomach and intestines. It is therefore not surprising to find that in many cases the stools are reported to be dark. In a case of my own I found that the fæces in the large intestine were clay coloured; in the small, dark, almost black. I look upon the absence of colour in the stools as tolerable proof that the common bile-duct is obstructed; but I cannot accept the presence of colour in the stools as proof that the duct is free.

The theory of hæmatogenous jaundice must, to my mind, be rejected. It is necessary not merely to suspend judgment, but to declare that the statements upon which the theory is founded are so doubtful that no conclusion can be drawn from them. Yet the theory is one which should not be altogether forgotten. Impossible as it may now seem, the pendulum of medical opinion may yet swing round, when the physiologists have made a few more experiments, to the point when this theory may be looked upon as proved. The history of medical opinion in no way contradicts this belief.

II. The second kind of jaundice is that of jaundice by suppression. The great doctors of the early part of the eighteenth century taught that the secretions existed ready formed in the blood, and that the glands merely acted as filters to strain the secretions from the circulating mass. Glisson speaks thus of the bile.³ The doctrine of jaundice from suppression of the secre-

¹ Oskar Wyss, *Arch. d. Heilkunde*, 1867, p. 469.

² Ebstein, *ibid.*, p. 506; and 1869, p. 379. I have myself verified the statement that the smaller ducts may hold bile, the larger a colourless fluid. (*Trans. of the Path. Soc.*, 1874, vol. xxv. p. 161.)

³ Glisson, *Anat. Hepat.*, Amstelodami, 1659, cap. xxxviii. *et seqq.*

tion was a natural outcome of this physiology. It is therefore to be found in the writings of Morgagni, who distinctly attributes the jaundice in case of obstruction not to the absorption of bile already secreted, but because the bile-ducts being already filled by secretion, the bile cannot enter them, and thus accumulates in the blood.¹ Van Swieten likewise speaks of two kinds of jaundice: one from an impediment to the free exit of the bile from the bile-ducts; the other from a hindered secretion.² Towards the end of the eighteenth century this view of jaundice began to lose ground, even before the experiments of Saunders. It continued, however, to be taught and acted on by many noted physicians.

Andral,³ Sir Thomas Watson,⁴ Bamberger,⁵ Griesinger,⁶ and Trousseau,⁷ have all supported the doctrine of jaundice by suppression. Budd⁸ considers this kind of jaundice by far the most common. He believes, however, that the colouring matters only are made in the blood, while the bile acids are formed in the liver itself: a view which is supported by Skoda⁹ and Dr. George Harley.¹⁰ But about the date of the publication of the first edition of Budd's book the doctrine of the formation of the bile in the blood fell into discredit with physiologists, and in our own day, with the exception of one or two supporters, is universally disbelieved. With this, as a matter of course, the notion of a jaundice from suppression of secretion fell too.

The turn of the tide of medical opinion may be noted when Liebermeister, more than ten years ago, pointed out that he did not think the doctrine of the non-existence of the elements of the bile in the blood so surely proved as others then thought. One of his reasons is that jaundice sometimes breaks out after the liver-cells have completely disappeared.¹¹ There is no proof,

¹ Morgagni, *De Sedibus*, Ep. xxxvii. art. 9.

² Van Swieten, *Comment.*, § 950, *Ludg. Bat.*, 1785, t. iii. p. 127.

³ Andral, *Clinique méd.*, Paris, 1839, t. ii. p. 286.

⁴ Sir Thomas Watson, *Lectures on the Principles and Practice of Physic*, London, 1857, fourth ed. vol. ii. p. 602, lecture lxxxv. He says that Darwin was the first to introduce the doctrine into this country, since supported by Chevreul and Mayo. Erasmus Darwin (*Zoonomia*, Lond. 1801, vol. ii. p. 5) speaks of a paralysis or inability of the secretory vessels of the liver, but without bile in the stools or urine, and a skin like the colour of full-grown silkworms. It may well be doubted if he speak of a jaundice.

⁵ Bamberger, *Krankheiten des chylopoëtischen Systems*, Erlangen, 1857, p. 517.

⁶ Griesinger, *Infectionskrankheiten*, Erlangen, 1857, p. 78.

⁷ Trousseau, *Clinique méd. de l'Hôtel Dieu*, Paris, 1865, 2e éd. t. iii. p. 274. His opinions expressed elsewhere make it doubtful if he always held this belief.

⁸ Budd, *on Diseases of the Liver*, London, 1845, p. 373.

⁹ Skoda, *Deutsche Klinik*, 1859, p. 286.

¹⁰ George Harley, *Jaundice*, London, 1863, p. 20.

¹¹ Liebermeister, *Beiträge zur path. Anat. und Klinik d. Leberkrankheiten*, Tübingen, 1864, p. 241.

however, that this statement of Liebermeister's is correct. No doubt he has shown that in a large number of cases, hitherto unsuspected, the liver-cells are destroyed; in many cases of pyæmia, puerperal and typhoid fever; but I do not think he has proved that the jaundice has arisen after the complete destruction of the secreting tissue of the liver. Some writers have thought that acute yellow atrophy affords the best proof that the bile is made in the liver, because towards the end of the disease, as the liver-cells are destroyed, but little pigment is found in the urine, and no bile acids.¹

Dr. Moxon thinks that the colourless contents of the ducts in cases of long-continued obstruction prove that the bile is not formed in the liver, and that all cases of jaundice are caused by suppression of the secretion, not by reabsorption of secreted bile.² His views closely resemble those of Morgagni quoted above, but it cannot be thought that he has brought forward much evidence in favour of his belief.

The theory that the bile was not formed by the liver was thought to have been disproved by Johannes Müller,³ Kunde,⁴ and Moleschott.⁵ This last observer found that frogs, after their liver had been taken away, lived nearly three weeks; and yet no trace of bile could be found in the blood, muscles, gastric juice, lymph, or urine. The vital processes in these cold-blooded animals are, however, but slow. Leyden tied the common duct of frogs, and after eight to fourteen days found no trace of jaundice in the liver;⁶ so that if no jaundice be caused by ligature of the common duct, it would not be strange that none should arise after the taking out of the liver. If these experiments of Leyden's be trustworthy, a very important support to the view that the liver forms the bile is taken away.

Another fact thought to favour this last view is that the bile acids and bile pigments cannot be found in the blood, even in the blood of the portal vein. Of this last there is usually so little to be had that there is scarcely enough for a trustworthy analysis. But I believe that no observer in modern times has been able

¹ Hilton Fagge, *Guy's Hospital Reports*, 1875, p. 174; and Murchison, *op. cit.*, p. 229.

² Moxon, *Transactions of the Pathological Society of London*, 1873, vol. xxiv, p. 133.

³ Johannes Müller, quoted by Moleschott.

⁴ Kunde, *De Hepatitis Ranarum Extirpatione*, Diss. Inaug. Berol. 1850. Quoted by Moleschott.

⁵ Moleschott, *Arch. f. phys. Heilkunde*, 1852, p. 479.

⁶ Leyden, *Beiträge zur Path. des Ikterus*, Berlin, 1866, p. 19. He does not, however, expressly say that he examined the blood or urine; nor does he say that he fed the animals, a point of importance in judging of matters allied to the digestion and general powers of life.

in health, with certainty, to detect any of the elements of the bile in the blood. Naunyn, however, has found small but appreciable amounts of bile acids in the urine of healthy men :¹ and it is no uncommon thing to find that the urine of men and dogs show traces of the presence of bile pigment ; and this is especially the case after long fasting, when, that is to say, the liver has not for some time been stimulated to excrete much bile. It may further be alleged that if the elements of the bile be formed in the blood, it would be unlikely that they should be found in the serum. The liver seems, as shown by the experiments quoted just above, to have the power of rapidly excreting into the bile-ducts all the bile pigment and bile acids which may be brought into the blood. On the other hand, it may be pointed out that Schmulewitsch has succeeded in keeping up a continuous secretion of bile for two or more hours after death, by causing the same blood to pass and repass many times through the liver,² a phenomenon opposed to the notion that the bile is simply separated by the liver from the blood. The presence also of a small quantity of the bile pigments and bile acids in the urine may well be explained by supposing that they have been absorbed from the bile-passages after secretion. Such was the belief of Dr. Bence Jones,³ who says that the bile begins to pass out of the gall-bladder as soon as it is passed into it. Of this statement it must be admitted, however, that very little direct proof exists.

Some authors, especially Dr. Murchison,⁴ have looked upon the fact that in some cases of diseased livers without jaundice no bile can be found in the bile-ducts, as evidence in favour of the view that the bile is formed in the liver. Dr. Hilton Fagge, in a paper lately published,⁵ admits that this would be good evidence were the liver diseased in all the cases in which the bile-ducts

¹ Naunyn, Arch. f. Anat. u. Phys., 1868, p. 430.

² Schmulewitsch, Arb. a. d. phys. Anst. zu Leipzig, Jahrg. 1868, p. 113. Pflüger (Arch. f. d. ges. Phys., 1871, Bd. iv. p. 54) has, however, thrown doubt on the interpretation of this phenomenon. He thinks there is no real secretion of bile, but only a transudation of serum into the ducts, which pushes before it the bile already formed before death.

³ Bence Jones, St. George's Hospital Reports, 1866, vol. i. p. 190.

⁴ Murchison, Clinical Lectures on Diseases of the Liver, London, 1868, p. 305. I think these cases of colourless mucus in the bile-passages without jaundice are somewhat rare. Upon Haspel's cases (Maladies de l'Algerie, Paris, 1850, t. i. p. 262) I think much weight can scarcely be laid. He is speaking of abscess of the liver in which there was almost complete destruction of the organ. He adds that the bile was no longer secreted, and the bladder contained only a little white mucus. He does not speak of the state of the other bile-passages. See also cases reported by Andral (Clinique méd., Paris, 1839, t. ii. p. 275); Frerichs (*op. cit.*, Bd. i. pp. 86 and 322); Wertheimber (Fragmente zur Lehre vom Icterus, München, 1854, p. 3).

⁵ Hilton Fagge, Guy's Hospital Reports, 1875, p. 172.

were unstained. He then quotes Dr. Moxon's authority for saying that in only one out of four such cases observed by him was the liver diseased,¹ the other three being cases of pyæmia and pneumonia. The liver is so commonly diseased in these two morbid states, and as commonly overlooked, that it would be desirable to have further evidence as to the state of the liver before deciding that the liver is not always diseased when the ducts are free from colour.

When physiology gives forth so uncertain a sound upon a matter so weighty as the place wherein the bile is made, it may be forgiven to the physician if he suspend his judgment, and wait for new facts before he allow of the possibility of a jaundice from suppression. It is true that great names, both in England and in Germany, may be called in support of the theory. Nevertheless, in the present state of knowledge the pathological evidence in favour of the theory is so small, and the physiological phenomena upon which it is based of so uncertain an interpretation, that no one can look upon it as a probable opinion.

III. The third theory of jaundice is that which attributes this symptom to the absorption into the blood of bile already excreted by the liver. It is beyond all doubt the best founded of the theories of jaundice; and the one to which the morbid anatomist, as well as the experimental pathologist, will be the most inclined, as in the great majority of cases of jaundice which are examined after death some obstruction to the flow of bile into the duodenum is undoubtedly found.

It has been seen that, very early in the history of medicine, obstruction to the bile-ducts was looked upon as a cause of jaundice. No distinction, however, was made between the effects of an obstruction to the hepatic and the cystic duct; and Morgagni was the first to teach that the cystic duct could be obstructed without jaundice being caused. This writer did not, however, look upon the absorption of secreted bile as the cause of jaundice. He thought that the bile was unable to pass from the liver into the ducts, these being already overfilled, and that jaundice was caused by suppression of secretion. This doctrine, however, was overthrown at the end of the last century by the experiments of Saunders. He threw a ligature around the bile-duct of a dog; it was killed in two hours after. A bilious-coloured fluid was seen in the absorbents around the parts and near the thoracic duct. The serum of the blood taken from the jugular vein gave a yellow tinge to white paper dipped in it; much less, however, than the serum from the hepatic veins, the deeper colour of which was

¹ Moxon, *Trans. of the Path. Soc. of London*, 1869, vol. xx. p. 220.

well marked.¹ From these two experiments Saunders thought that it was proved that the absorbents as well as the hepatic veins are concerned in the bringing forth of jaundice.²

For nearly a hundred years this theory of jaundice from absorption has held its own, and been assented to by every physician and pathologist who has written on the subject. Among the theories of jaundice it is the only one grounded upon observation and experiment, and the only one to which universal assent may be demanded.

Three kinds of jaundice from absorption have been described :

- i. Jaundice from obstruction of the ducts.
- ii. Jaundice from absorption of the bile when the pressure in the blood-vessels of the liver is decreased.
- iii. Jaundice from incomplete destruction of the bile absorbed into the blood.

It is well known from the experiments of Heidenhain that the pressure under which bile is excreted is extremely small.³ In guinea-pigs the bile ceases to flow down the ducts when opposed by a column of water only 20 centimetres high, and prefers to pass into the circulation. It will be seen from this fact how little obstruction to the passage of the bile into the duodenum is needed to cause the bile to flow back into the blood. Nor does it seem needful that the obstruction should be complete, but merely that the bore of the duct should be narrowed enough to cause the bile to pass with some trouble into the bowel. It would almost seem that the bile passes as readily into the circulation as into the duodenum. Thus jaundice may easily be caused by a hyperæmic or catarrhal swelling of the part of the common duct which passes through the walls of the duodenum. Yet after death all trace of swelling will have disappeared and the duct will be fully patent. The only proof of the existence of an obstruction is the finding of

¹ Saunders, *A Treatise on the Structure, Economy, and Diseases of the Liver*, London, 1803, 3d. ed. p. 111.

² Lately repeating Saunders' experiment, I was unable to arrive at the same results as he did (*St. Bartholomew's Hospital Reports*, 1873, vol. ix. p. 176). Frerichs likewise (*op. cit.*, Bd. i. p. 99) says he has been unable to discover any bile pigments in the blood, serum, or lymphatics twenty-four hours after the ducts have been tied. Saunders seems to have tied the hepatic duct; in my own experiment the common duct was tied. I have no doubt of the general accuracy of Saunders' conclusions.

³ Friedländer and Barisch, *Arch. f. Anat. Phys., &c.*, 1860, p. 666. Kowaleswsky (*Pflüger's Arch. f. d. ges. Phys.*, 1874, Bd. viii. p. 597) found the pressure of the bile in curarised cats somewhat higher, from 12 to 20 millimetres of mercury; and varying, in direct ratio, with the arterial blood pressure. There is another very instructive experiment of Heidenhain (*Stud. des phys. Instituts zu Breslau*, 1868, Heft iv. p. 232). He allowed a solution of indigo carmine to flow under small pressure into the bile-ducts; the mucous membranes, especially the conjunctiva, of the animal soon became blue; the urine likewise.

the part of the duct below the obstruction uncoloured by bile. Even this evidence may be wanting if the examination have not been made with care enough; for the least pressure on the gall-bladder will send the bile down the common duct into the bowel, staining the duct of a bilious colour. More obvious causes, such as the presence of a calculus in the duct, or the pressure of a tumour from the outside, need not here be spoken of.

A spasmodic constriction of the common duct was thought by Cullen to be sometimes a cause of jaundice.¹ This method of explaining some cases of jaundice in which no obstruction could be found has of late years fallen out of favour, as the anatomists could find no muscular fibres in the gall-ducts. Quite recently Audigé has formed the opinion, based upon new anatomical and experimental researches, that the bile-ducts are able of themselves to contract. Notwithstanding, he rejects the doctrine of spasmodic jaundice.²

The second kind of jaundice from absorption is that which takes place when the pressure of the blood in the vessels of the liver is decreased. This is Frerichs' theory of jaundice. He says that of the two products of the liver-cells, bile and sugar, one passes into the hepatic vein, the other into the capillary bile-ducts. The flow of fluid towards the blood can only take place by diffusion; towards the bile-ducts, by filtration. How the two are separated is unknown. It must be assumed either that the rapidity of the diffusion into the blood of the elements of the bile is greater than that of sugar, or that the sugar has some attraction to some element of the blood which is wanting to the bile. This last hypothesis is improbable, while no constituent of the blood is known to possess an attraction for sugar. On the first hypothesis, the separation of the two substances would be incomplete; some of the bile passing into the blood, and some of the sugar into the bile, which indeed is really the case.³ If therefore the pressure of the blood upon the sides of the vessels in the liver be decreased, the bile will pass in that direction where there is least resistance, that is, into the circulation, and thus jaundice will arise.⁴

Heidenhain has given experimental evidence of the truth of this hypothesis. He found that on decreasing the pressure of the blood in the vessels of the liver, the bile already formed began to

¹ Cullen, *First Lines of the Practice of Physic*, § 1818.

² Audigé, *Recherches expérimentales sur le Spasme des Voies biliaires*, Paris, 1874, p. 46.

³ C. Ludwig, *Lehrb. d. Phys. des Menschen*, Leipzig and Heidelberg, 1856, Bd. ii. p. 232.

⁴ Frerichs, *op. cit.*, Bd. i. p. 89.

pass into the circulation.¹ Frerichs would in this way explain the jaundice seen in cases of plugging of the portal vein, in cases of pigment liver where a part of the capillaries is filled by masses of pigment, the jaundice seen in the new born, and after bleeding from the roots of the portal vein, as in yellow fever. This theory of jaundice must undoubtedly be allowed a place equal in probability to that of jaundice from obstruction. It may prove serviceable hereafter in explaining the jaundice which so suddenly arises after mental emotions, the bites of serpents, and after great general bleeding.²

The last theory of jaundice which remains to be discussed is that which attributes the symptom to incomplete destruction of the bile absorbed into the blood. In health the greater part of the bile pigment and bile acids poured into the duodenum is taken up again during the passage of the food through the small intestine. The bile therefore finds its way into the radicles of the portal vein, and undergoes a change of some kind, probably oxydation, after which it is thrown out of the system. When this metamorphosis of the bile in the blood is checked or brought to a standstill, jaundice comes on.

This theory may be found in the writings of Daniel Sennert, who speaks of an excess of bile secreted by the liver which cannot be excreted by the accustomed passages. In this disease, he says, not only is the urine coloured saffron, but the fæces are also highly coloured. Fever is present, and the hands and feet are hot.³ Distinct traces of the same doctrine may be found in the writings of Cullen, who regards it with disfavour,⁴ and in those of Portal⁵ and Gardien,⁶ who approve. Frerichs had no doubt some such theory in mind when he speaks of a jaundice, quite independent of the liver, caused by decreased decomposition and change of the bile in the blood. He rests, however, too much upon observations which are now known to be incorrect, those of the change of the bile acids into bile pigment.⁷ Dr. Murchison may be looked upon as one of the most prominent modern defenders of this theory, and the following account of it is taken from his work.⁸

¹ Heidenhain, Studien des phys. Instituts zu Breslau, Leipzig, 1868, Heft iv. p. 238.

² In this way I should be inclined to explain the jaundice seen in the two first cases recorded by Mr. William Smith (Brit. Med. Journal, 1869, vol. ii. p. 5). The jaundice appeared on the first and fifth days after great general bleeding. The stools were light coloured. In the two last cases I feel scarcely disposed to connect the bleeding with the jaundice.

³ Dan. Sennert, Epitome universam Doctrinam summa fide complectens, Edid. Bonetius, Col. Allob. 1655, p. 681.

⁴ Cullen, First Lines of the Practice of Physic, § 1817.

⁵ Portal, Samml. auser. Abh., Bd. viii. p. 8.

⁶ Gardien, Traité complet d'Accouchements, Paris, 1816, t. iv. p. 95.

⁷ Frerichs, *op. cit.*, Bd. i. p. 94.

⁸ Murchison, Clinical Lectures on Diseases of the Liver, Lond. 1868, p. 375.

The greater part of the bile after having been poured into the intestine is taken up by the radicles of the portal vein, and thus passes into the general mass of the blood. In health, the bile suffers a decomposition, probably an oxydation, and is cast out of the system by the lungs or kidneys. But in some diseased states this change does not take place, and the bile circulates with the blood, causing a jaundice. The diseased states which hinder these changes of the bile from taking place are (i.) the action of certain poisons on the economy. They are those poisons chiefly which cause an acute parenchymatous degeneration of the glands and muscles. (ii.) Nervous influences, as fright; (iii.) bad hygienic conditions; and (iv.) a very great increase in the secretion of bile, so that more is poured out into the gut than can be taken up and changed in the blood into colourless material. A jaundice therefore arises from the circulation of bile pigments in the blood.

This theory rests for support chiefly on the observations of Bidder and Schmidt, who found only slight traces of bile in the fæces of dogs.¹ But Hoppe-Seyler, ten years after, found abundance of cholalic acid in the fæces of dogs. He looks upon this substance as a product of a fermentation or sort of digestion of the bile acids, very like the splitting up of hippuric acid into glycin and benzoic acid in decomposing urine.² Further, Vanlair and Masius found abundance of stercobilin, a substance closely allied to the urobilin of Jaffé, in the fæces of men. They regard this as slightly-altered bile pigment, and believe that the greater part of the bile pigment is thus excreted with the fæces and not absorbed into the circulation.³ Resting on these observations, it would seem likely that both the bile acids and bile pigments are cast out of the body with the fæces. Still many physiologists believe that a good deal of the bile is again absorbed from the intestines. Even on this view, which has not many physiological facts for its support, it is quite unlikely that such absorption of bile should ever become the cause of jaundice; for all the bile absorbed by the vessels is at once carried by the portal system to the liver. The liver, it is known by the experiments of Schiff⁴ and Tarchanoff,⁵ has a marvellous power of

¹ Bidder and Schmidt, *Die Verdauungssäfte und die Stoffwechsel*, Mitau and Leipzig, 1852, p. 217.

² Hoppe, *Arch. f. path. Anat.*, 1862, Bd. xxv. p. 181. A year later, however, he expressed his belief that a great part of the bile acids pass into the blood, and are there oxydised (*ibid.*, 1863, Bd. xxvi. p. 535).

³ Vanlair and Masius, *Centralblatt f. d. med. Wiss.*, 1871, p. 369. Jaffé (*ibid.*, p. 465) denies the identity of stercobilin with his urobilin.

⁴ Schiff, *Arch. f. d. ges. Phys.*, 1870, Bd. iii. p. 598; and *Zeitschrift f. rat. Med. Bericht f.* 1868, Bd. xxxv. p. 216.

⁵ Tarchanoff, *ibid.*, 1874, Bd. ix. p. 332. See also Feltz and Ritter, *Journal*

drawing to itself the bile acids and bilirubin in the blood, and forthwith excreting them in the bile. It would therefore seem most likely that if any excess of bile acids and bile pigments were absorbed by the intestine, they would again appear in the bile without entering the general circulation; and without entering the general circulation no jaundice can be brought about.

The only instance in which this theory, to my mind, can be entertained is the case of a permanent obstruction of the circulation through the portal vein. Schiff has artificially obliterated the portal vein and found traces of the presence of the bile acids and bile pigment in the urine, which however soon disappeared.¹ It is known that in cases of thrombosis of the portal vein, jaundice is a common symptom; but it also appears to be a permanent, and not merely a passing, phenomenon.

de l'Anat. et de la Phys., 1870, p. 315. The experiments of Huppert (Arch. d. Heilkunde, 1864, p. 236) and of E. Bischoff (Zeitschrift f. rat. Med., 1864, p. 125) would seem in some degree to be against Schiff and Heidenhain. Huppert, after injecting bile acids into the blood, found only a fourth or a third excreted by the liver. Socoloff (Arch. f. d. ges. Phys., 1875, Bd. xi. p. 166) has lately denied that the liver has any special power of excreting bile acids injected into the blood.

¹ Schiff, *loc. cit.*

CASES IN SURGERY

BY

LUTHER HOLDEN.

1. *Compound Dislocation of the Shoulder—Reduction—Subsequent Necrosis of the Head and Neck of the Humerus—Recovery with a useful Limb.*

Thomas Turner, aged 19, an artisan, was brought to the Hospital on November 30, 1875, with a compound dislocation of the left humerus. The skin was torn across the axilla from one margin to the other. The muscular boundaries were also extensively lacerated. Through the wound the head of the bone could be felt and seen below the coracoid process. A small fragment of the head was chipped off. There was also a comminuted fracture of the left clavicle, and other flesh wounds below the jaw. The patient was extremely pale and suffering from shock.

The accident was occasioned by machinery. It appears that while the arm was extended over a rapidly-revolving bar of iron, his loose jacket became entangled so as to drag the head of the bone through the skin of the axilla. But for the timely stopping of the machine, the limb in a few seconds would have been torn from the trunk.

At a consultation with some surgical colleagues whom I had the good fortune to find at the Hospital, it was unanimously agreed that the dislocation should be reduced and an attempt made to save the arm. The grounds for this conclusion were the youth of the patient, and the apparent immunity from injury of the main artery and nerves, as evidenced by the pulse at the wrist and the sensation of the hand.

The reduction was easily effected under ether. No sutures were used. The wound was lightly dressed with oiled lint; and the arm, a little removed from the side, was laid upon a pillow. Slight venous hæmorrhage, which came on a few hours afterwards, was easily stopped by pressure.

2d day.—Patient has slept at intervals. Complains of thirst, and great pain in the shoulder. Has taken a pint of milk and half a pint of essence of beef. Pulse, 130; temperature, 103°; respirations, 30. Dressings removed and wound well washed with a solution of Condy's fluid. Oiled lint reapplied. Half a pint of brandy to be taken with his food during the twenty-four hours.

3d day.—Has passed a less painful night after a subcutaneous injection of morphia. Has taken something every half hour; either brandy, or stout, or essence of beef. The pulse (120) beats regularly at the left wrist. Temperature, 102.4°.

4th day.—Has had a fairly good night after the usual morphia injection; taking nourishment or stimulants at frequent intervals. Pulse, 108; temperature, 100.6°. Tongue dry and furred. Always complains of thirst, which is relieved by lemonade or milk. Has a water pillow under his back. Wound begins to discharge foetid pus.

6th day.—Shoulder and entire arm exceedingly painful and cedematous. Wound discharges pus and gas so offensive that it can be detected far away from the bed. The breath is almost as offensive as the wound. A solution of Condy to be injected three times daily through a soft catheter introduced into the joint so as to wash out the recesses. A solution of terebine to be used in the form of spray over the wound and the bedclothes.

7th day.—Bowels not having been relieved since the accident, to have an injection of olive oil.

9th day.—Bowels freely relieved many times since the injection. Has good nights. Wound looks more healthy, and pus is less offensive. A poultice to be applied covered with terebine spray.

12th day.—Early this morning he had a severe rigor followed by profuse sweating. His temperature rose suddenly to 105°; pulse, 128; respirations, 42. Soon afterwards he felt sick, and passed two copious semi-solid clay-coloured motions. His temperature fell in the course of an hour to 100.2°. Ordered 4 grains of quinine with dilute sulphuric acid every four hours.

13th day.—The sudden alarm of yesterday has subsided. Temperature, 101.6°; pulse, 99. Wound discharges freely less offensive pus.

15th day.—Progressing favourably. Pulse, 90; temperature, 98°.

For some weeks he continued much the same, making but little progress, and always complaining on the least movement of the shoulder. The large wound gradually healed, leaving two or three sinuses which discharged offensive pus, leading to the conclusion that there was dead bone at the bottom. At length he

began perceptibly to lose ground. The pain in the shoulder and arm, the constant discharge, a large slough over the bruised internal condyle, flagging spirits, disinclination for all food, and, worse than all, a troublesome cough which prevented sleep, made me very anxious as to the result. By way of a change, I moved him from the back ward into the front on the 62d day.

76th day.—The move from one ward into another, trifling as it may seem, was followed by a marked improvement in the cough, by better nights, better appetite and spirits.

104th day.—An abscess about the level of the surgical neck of the humerus was opened under chloroform. The opportunity was taken to explore the sinuses, and an extensive surface of dead bone was detected.

118th day.—Having reason to believe that the dead bone was loose, I made a free incision down the front of the shoulder and removed without much difficulty what turned out to be the head with the tuberosities and about one inch of the surgical neck of the humerus. There was free hæmorrhage from the vascular lining of the cavity which contained the dead bone.

The subsequent progress of the case was in all respects satisfactory. The wound filled up. He rapidly recovered strength, and on the 128th day was able to leave his bed and walk about with his arm in a sling. Soon afterwards he was sent to the Convalescent Hospital at Highgate.

I saw the patient in July, about seven months after the accident. The sinuses had healed. The arm was shortened to the extent of $1\frac{1}{2}$ inch. The muscles of the shoulder were atrophied. He was gradually regaining the use of the elbow and the hand.

It is due to my successive house surgeons, Messrs. Shuter and Edwards, to state that the well-doing of this case is mainly owing to their watchful care, especially during the early days after the accident.

This is the first compound dislocation of the shoulder, as far as I can ascertain, received within living memory into St. Bartholomew's Hospital. In surgical literature I find the following instances recorded.

Sir A. Cooper¹ gives one case (Dixon's). It happened to a man aged 55 while dead drunk. Reduction was easy. He had quite recovered fourteen months after the accident.

Erichsen² has seen two cases. Both were reduced and did well.

Hamilton³ mentions two cases. One (in a man of 30) was

¹ Treatise on Dislocations and Fractures, 6th ed., 1829, p. 403.

² Science and Art of Surgery, 6th ed., 1872, vol. i. p. 375.

³ Treatise on Fractures and Dislocations, 5th ed., 1875, p. 785.

reduced, and rapidly recovered with good use of the shoulder. The other was resected, and died.

Fergusson¹ has seen a case which was reduced, and recovered 'with tolerable use of the injured arm after some months.'

The 'Lancet'² records a case (Scott's) caused in a boy of 14 by a fall from his horse. Reduction was easy. As a measure of precaution the boy was bled to $\frac{3}{4}$ xvi. on each of the first three days after the accident. The case did exceedingly well.

The 'Nouveau Dictionnaire de Médecine et de Chirurgie'³ mentions three cases, of which two died before the arrival of the surgeon; the third was resected, and died on the sixth day.

Thus I have given the recorded results of eleven instances of compound dislocation of the shoulder, inclusive of the present. Of these, seven were reduced, and recovered with fair use of the arm; two were resected, and died; two died before the arrival of the surgeon.

2. Two Cases of Popliteal Aneurysm cured by Digital Pressure of the Femoral.

First Case.

John Smith, aged 34, was admitted into St. Bartholomew's Hospital on the 13th June 1876, with a small distinctly-pulsating aneurysm in the right popliteal space. He states that he is by occupation a builder; that he has generally had good health; that his attention was drawn to a stiffness in the knee about two months ago; that he first noticed a pulsating swelling in the ham about one month ago; that the swelling gives him no uneasiness, but is increasing in size. He attributes it to a sprain caused by a fall from a ladder four months ago.

June 15th.—After free evacuation of the bowels, compression of the femoral was attempted by an ordinary tourniquet. Patient being unable to bear it for more than an hour, digital pressure (assisted by a weight of 8 lbs. of shot in a bag) was commenced at 4 P.M. After continued pressure for six hours, all pulsation in the aneurysm ceased.

June 16th.—A very slight return of pulsation being detected this morning, digital pressure was kept up for nine hours. After this there was complete absence of pulsation.

June 17th.—Has passed a good night. No pain or return of pulsation in the aneurysm.

June 20th.—Feels well and strong.

¹ Practical Surgery, 5th ed., 1870, p. 226.

² 1836-37, vol. i. p. 812.

³ Tome 13ème, p. 477, art. 'Épaule.'

June 29th.—Discharged. Thus the aneurysm was cured by fifteen hours' pressure in two days.

The man came to report himself to me five weeks after his discharge. He was quite well. There was no trace of aneurysm. The tibial arteries pulsated nearly as strongly as in the other leg.

Second Case.

William Rowden, aged 28, was admitted into Pitcairn Ward, on the 24th January 1876, with a large popliteal aneurysm in the left leg. He states that six months ago he felt a sense of fulness in the back of the knee. He first noticed the pulsating swelling about six weeks ago, after doing very heavy work.

The aneurysm is about the size of a large hen's egg, and very painful, especially when he walks. The pain extends down to the ankle and prevents his sleeping. The affected knee measures in circumference at least 2 inches more than the other.

January 27th.—Digital pressure was applied for nine hours.

January 28th.—Pressure for fifteen hours.

January 29th.—Pressure commenced at 9 A.M. At 1 P.M. the aneurysm ceased to pulsate, and became suddenly exceedingly painful. The pain in the knee and leg was so severe that large doses of morphia were required before any relief was obtained. Pressure continued till 10 P.M.

January 30th.—Has passed a good night. Slight return of pulsation. Pressure applied to-day for thirteen hours.

January 31st.—Pulsation barely perceptible. No pressure to-day.

February 4th.—The aneurysm again pulsates distinctly. Pressure for twelve hours.

February 5th.—Pressure for fifteen hours. Pulsation scarcely perceptible.

February 6th.—Pressure for fifteen hours.

After this there was no return of pulsation. The aneurysm has become harder, and the knee has diminished about $\frac{3}{4}$ inch in size. Patient feels well and hungry.

The man remained about a fortnight longer in the Hospital; and left able to walk without pain, and in all respects progressing favourably.

In this instance the aneurysm was cured after ninety-two hours of pressure, extending over eleven days. The case indeed seemed cured on the fourth day, after fifty hours' pressure; but a return of pulsation on the eighth day required a resumption of the pressure for forty-two hours longer.

A CONTRIBUTION TO OUR KNOWLEDGE
OF THE
PHYSICS OF THE CEREBRAL CORTEX.

BY
W. AINSLIE HOLLIS, M.D.

The study of mental physics has, I believe, been much obscured by the prevalence of two antagonistic doctrines regarding the mechanism of the brain—the one which denies the possibility of any attempts at the localisation of special function in the cortex; the other which would map out accurately in the grey matter of the convolutions the material boundaries of each separate act of volition. The truth probably rests between these two hypotheses.

To speak in general terms, the active brain may be considered to eliminate two kinds of thought—the expressive, or that which terminates in some form of voluntary action; and the retentive, which depends on and reproduces more or less vividly the impressions of external nature conveyed to it by our organs of sense. In many of the higher intellectual operations both forms of thought exist with various modifications.

It will not repay us to consider here how such thoughts arise—whether by a direct stimulation of the cortical cells, as Meynert suggests, or by a reflex irritation conveyed thither from the great basal ganglia, according to the hypothesis of Luys. It is, however, probable that both forms of stimulation occasionally take place.

If we now turn to the anatomy of the brain, as described so ably by Meynert, we learn that the anterior or fronto-parietal portion of the cortex is more intimately connected with the corpora striata and crusta cerebri than either the temporal or occipital lobes, which, on the other hand, are closely connected with certain portions of the optic thalami and optic tract. In correspondence further to these diverse attachments of the fibrillar

elements in the two regions is a peculiarity in the shape of their cells, and in the arrangement of the layers of the grey substance—a peculiarity which exists in the brains of the lower animals as well as in man.¹ Such differences of structure in the two regions of the brain cortex might justify us in assuming an absence of exact functional correspondence in their elements, without such further proofs as I shall now adduce.

Immediately bounding the great transverse fissure of the brain, that of Rolando, are the two convolutions, named by Ecker the anterior and posterior central; by Turner, the ascending and descending parietal respectively. The anterior gyrus at its lower margin passes into and is more or less continuous with another plica of grey matter, called the third or inferior frontal convolution. The last gyrus bounds a portion of the fissure of Sylvius, and is intimately connected with the insula. It is to the whole of this portion of the brain that I wish at first to draw attention.

The experiments of Hitzig and his followers, confirmed by others both in America and this country, prove the existence of an intimate connection between the central gyri and the production of certain muscular movements in the limbs, neck, and back on the opposite side of the body. This association is absent in the very young, as Soltmann and others have noticed. It has, however, been urged that similar movements can be obtained by stimulating the surface of the striate bodies.

Pathologists generally admit that a lesion affecting the integrity of the grey matter in or about the third frontal convolution and the insula on the left side especially (I include circulatory affections), is followed by a diminution if not an entire loss of the power of applying words to things. Other parts of the grey substance of the convolutions do not appear to have any intimate connection with muscular movements.

Experience thus supports the hypothesis that certain portions of the fronto-parietal lobes are intimately associated with voluntary movements. The nature of this connection I shall next attempt to show. One peculiarity of our mental physics is the ready adaptation by our brains of any symbolic notation. Such an arrangement is convenient, as it epitomises, so to speak, our impressions of external nature. In articulate language we have the application of certain sound symbols, produced by muscular action, to certain objects, their attributes and relations. When a man loses the power of associating words and objects, of calling a pen a pen, although he be able to utter the word 'pen,' he has lost that regulative power over his co-ordinative ganglia by which he could voluntarily select the

¹ Jour. Ment. Sc., January 1876, p. 506. Meynert, *The Brain of Mammals*, Stricker's Handb. of Histol., N. S. S. ii. p. 390 *et seqq.*

proper muscular adjustments for the production of the required word. Now this regulative or selective function must be acquired by experience in each particular case. We may thus explain its absence in the young. The repetition of an action induces an acquired facility in its performance. The frequent exercise of any particular series of nerve cells and fibres appears so to increase their functional excitability that they will act without the stimulus of the higher or selective centres—they become ‘mechanically automatic.’ We may thus explain the purposive movements of a decapitated frog, and the results of stimulating the striate bodies in the higher animals. In the disease agraphia there is a similar loss of the regulative function of the cerebrum over the muscles of the hands, as there is in aphasia over the voice. It is probable that such complex symbolic actions as speaking and writing can never be entirely relegated to the lower ganglia for their performance. When, however, they are dissociated from any symbolic meaning, as in the mutterings of low delirium, or in simple verbal ejaculations, they may probably be ranked with the convulsive movements of the other voluntary muscles, and may originate after a while in the great basal ganglia of the brain. All movements of precision in the voluntary muscles require experience or education for their proper performance, and they probably exercise the regulative functions of the brain for their attainment. When once acquired, however, such movements, if frequently repeated, become automatic, and require little or no further expenditure of brain-energy for their continuation.

Besides this connection between the cells of the cortex and the production of muscular movements, there is, as its anatomical characters show, an intimate intercellular association in the grey matter of the brain. Although the remainder of the fronto-parietal lobes, other than the convolutions I have mentioned, yields only a negative result to physiological experiment, there is assuredly an indirect connection with such movements in accordance with its anatomical relations. If this hypothesis be correct, we should expect to find in the higher races of mankind a far greater development of the frontal lobes of the brain, corresponding to the greater variety of symbolic and other precise movements which they acquire, than is found in less civilised races, and *a fortiori* in the lower animals—and this is the case. The Negro Mongolian, and Carib (American Indian) are distinguished from the European by their laterally compressed and retreating foreheads; their facial angles are thereby rendered more acute. Professor Wallace in a recent address before the British Association, (1876) has called attention to the fact that light tints of skin are generally accompanied by some deficiency in the senses of smell,

hearing, or vision,¹ and that it is only by the greater development of his mental faculties over mere sense-acuteness that the xanthocroic race of mankind is able to compete with the darker races. The posterior part of a Carib's or Negro's skull does not differ materially in capacity from that of the white races; it is in the anterior region that the deficiency of skull-space is so clearly marked, and it is to this the mental inferiority above noticed must be due.

As we advance forwards from the central gyri to the posterior frontal gyrus, we find the regulative function passes from simple muscular actions to complex symbolic actions, such as writing or speaking. One of the highest mechanical operations of the human brain is that of calculation. We here use symbols to represent ideas of abstract numbers. The rudimentary methods of calculation in use amongst the lowest savage tribes (as mentioned by Tyler), when taken in conjunction with the small comparative size of their frontal lobes, justifies, I believe, the assumption that this faculty is seated in the forepart of the brain. That it is separable from, although closely connected with, the actual expression of mathematical results by muscular action, the following case will show. A woman in the Sussex County Hospital suffered from an inability to speak more than three or four words (Yes, Nay, T'be sure); there was no paralysis of the muscles of the mouth and throat. This patient, though ignorant of writing, was nevertheless able to calculate small sums. If, for instance, she was asked whether 2 and 2 made 5, she replied, 'Nay, nay; ' and on the other hand, 'Yes' immediately the right answer was given her. She showed in her actions generally a fair amount of intelligence. It will be for future investigators to confirm what I provisionally offer as a function of the frontal lobes—namely, the mechanical elaboration or symbolisation of ideas by words and numbers—talents cultivated by our authors and mathematicians respectively.

Besides the cerebral functions I have already considered, we possess, in common probably with the lower animals, the faculty of reminiscence. We observe an object with our sense-organs, and an impression of this object is left, which we can recall to our minds when the object itself is absent. Recollection does not entail the production of muscular movement for its completion. We can remember the features of an absent friend, or the sounds of a humming gnat or chirping bird, although we may be quite unable to express our ideas of them by muscular action. The production of such impressions has been variously accounted for, and in the educated brain it is, I believe, assisted, as Dr. Bastian and others have surmised, by the corrective actions of the various sense-organs and their muscles on each other's impressions. The

¹ See Dr. Ogle's Paper on the Sense of Smell, *Med.-Chir. Trans.*, 1870.

true concept of an object can only be obtained by repeated sense observations. The more frequently a particular impression of an object is renewed, the more firmly is it retained. Any striking peculiarity is readily fixed on our minds. As general attributes occur more frequently than special attributes, the former become more strongly retained in the mind than do the latter.¹ We thus gradually learn to think of the former as abstract ideas, occurring as they do in many objects. When, therefore, we think of vinegar, we at once attribute to it colour, sourness, fluidity—the results of previous experience. The autopsy of a person who has lost the power of appreciating such attributes in an object, may assist us in assigning a locality to such cerebral functions. In his essay on Aphasia Dr. Bateman gives the details of such a case.² He records it as one of amnesic aphasia, although he subsequently admits that such a title may be demurred to. I shall here only draw attention to a few symptoms in the case, as the details are fully published elsewhere. A gentleman suffering for some months from cerebral symptoms, became confused in his conversation, dropped objects from his right hand, and did awkward things at the table—on one occasion he poured vinegar on his repast for pepper. There was some loss of the memory of words, and things were called by their wrong names; for instance, being in a room where the fire was burning particularly brightly, he said, ‘How bright the poker looks!’ The person addressed said, ‘You mean the fire.’ ‘Yes,’ he said, ‘I mean the fire.’ At the autopsy, to pass over matters of minor importance in the present inquiry, ‘the frontal lobes were examined with great care, especially the third, and the substance between it and the corpus striatum, but these structures were found quite healthy. The disease was in fact limited to the posterior third of the left hemisphere,’ which was almost entirely destroyed. A characteristic symptom of the above case was a loss of the power of appreciating the qualities of objects—to mistake pepper for vinegar implies such a deficiency of brain function. The want of the faculty of applying correct names to things, which was also displayed by the patient, would be caused by this failure to appreciate the qualities of an object when it was perceived—its true concept was absent from the mind. Dr. Bateman, I believe, was entirely wrong, therefore, in supposing that ‘the idea was conceived, but the means of communication with the external world did not exist.’ Here is a case placed on record

¹ Following a somewhat similar line of reasoning, Dr. Osborne (Dubl. Jo. Med. Sc., 1833) explains the fact that in affections of the speech ‘nouns’ disappear before and return later than ‘verbs,’ &c.

² Jour. Ment. Sc., 1868, xiv. p. 352.

by Dr. Church.¹ A letter-sorter, who for two years had experienced a failure of memory, and an inability to pursue his vocation on that account, had two fits with convulsions of the right side within a few weeks of each other, and shortly before his death by coma. On examination *post mortem* a large tumour (2×3 inches) was found in the substance of the left temporal lobe, immediately posterior to the fissure of Sylvius. The right side of the brain was squeezed up by the increase in the size of the left. In this case, the first symptom, the loss of memory, probably corresponded with the gradual growth of the tumour in the temporal lobe. The occupation of letter-sorter requires the operator to have a clear mental picture of the position and relations of each opening in a large nest of pigeon-holes. It was this mental picture that first failed the man (as I ascertained from personal inquiry). He was unable to remember the situation of the various pigeon-holes, in order to associate with them a certain definite operation—the arrangement of the letters. In the two cases just detailed the mental impressions of certain objects were faulty, not from any failure of the organs of special sense, the conductors and modifiers of such impressions, but from a want of receptive power in the brain cortex itself. Pathology thus supports the hypothesis that the faculty of reminiscence, as far as it relates to objects, their attributes and relations, is situated in the posterior or occipito-temporal lobes, agreeably to what anatomy has led us to expect.

In the foregoing pages I have merely sketched out what I believe to be a correct system for the methodical study of the functions of the brain. I must warn students of this seductive branch of medical science not to attempt to localise in the cortex too closely the several faculties of the mind. It is preposterous to expect that similar cells are reserved for similar functions in all human brains, knowing what we do of the great diversity in man's mental nature, his various occupations, proclivities, and talents. Beyond the fact that there exists in our brains a posterior or retentive system, and an anterior or expressive system, our knowledge of this organ will not at present permit us to go. It will be for others to specialise more fully the peculiar functions of each division, and to show how they jointly elaborate new ideas.

¹ St. Bartholomew's Hospital Reports, 1869, vol. v. p. 207.

THE HISTOLOGY OF CERTAIN FORMS
OF
DEGENERATION OF THE TISSUES OF THE
NERVE-CENTRES.

BY
W. HENRY KESTEVEN.

This paper is intended as supplementary to some papers published by my father in the eighth volume of 'St. Bartholomew's Hospital Reports,' and in the 'British and Foreign Medico-Chirurgical Review,' July 1874, on the morbid histology of the spinal cord. The pathological changes, however, which are here described are not confined to the spinal cord, but also affect the brain and medulla oblongata. In the papers above alluded to the normal constituents of the spinal cord are stated to be—'1, blood-vessels; 2, nerve-fibres; 3, nerve-cells; 4, connective tissue and neuroglia.' These may also be considered as the constituents of the other nerve-centres.

The pathological conditions here called *degenerations* have more particular reference to the last three of these elements. The morbid conditions of the first element mentioned—namely, the 'blood-vessels'—do not come so immediately within the scope of this paper.

The connective tissue, or the neuroglia, forms the basis of the substance of the cord and brain, and in it are embedded the nervous elements and the connective corpuscles or nuclei. These nuclei are the *bioplasts* of the connective tissue of the nervous substance, as described in other structures by Dr. Lionel Beale. This view is borne out by the fact that these bodies take the staining of the carmine more deeply than the other structures, except

perhaps the axis cylinders of the nerve-fibres, which most probably consist of the same elementary components.

By the term *degeneration* is here meant a change in the normal structure which is often more qualitative than quantitative in character. It is the result of a morbid modification of the physiological process of repair, and consists in the substitution for the normal structure of an altered and more lowly-organised substance.

This degraded state is by far the most important pathological condition that can be met with in the nerve-centres, for the reason that it is by far the most common. In nearly every case in which disease of the nerve-centres exists may be found some one or more of its forms.

These lesions may be divided into—first, those that affect the nerve-fibres, connective tissue, and neuroglia; and secondly, those that affect the nerve-cells. Inflammation is the cause or the associate of many of these changes, but frequently there are no signs of inflammatory action to be met with. In a large proportion of cases the cause of the change is a vitiated condition of the blood supply, due to alteration in the blood-vessels.

Among the degenerations which affect the nerve-fibres, connective, and neuroglia, are the following: *sclerosis*, *colloid degeneration*, *amyloid degeneration*, *miliary degeneration*, and *granular degeneration*.

Sclerosis.—By the term sclerosis is here meant a condensation which is necessarily accompanied by a certain, though at times scarcely appreciable, amount of induration. Various forms of sclerosis have been described; in fact, the name seems to have been in some cases used to describe lesions which can hardly be called sclerotic. The true forms have been called '*sclerose en plaques*' by French authors; '*insular sclerosis*' by Wilks and Moxon; '*grey degeneration*' by Charcot, who met with it in the cord; and other names, dependent in some cases on the locality in which it was found, and in others on the appearances which it presented.¹

The following will, I believe, be found to be a fair description of the histological appearances presented by the various forms of true sclerosis.

If a thin section of spinal cord be placed under the microscope, simply mounted in glycerine, without staining, clearing, or any other than the necessary hardening process, the white matter of

¹ Miliary degeneration, when first described by Drs. Batty Tuke, Rutherford, and Kesteven, was called '*miliary sclerosis*;' but there is no good ground for describing that appearance as a sclerosis. This term has therefore been abandoned by the last-named observer. (See Brit. and For. Med.-Chi. Review, *loc. cit.*)

the external columns appears to be almost opaque, the grey matter slightly more translucent. This opacity is, as we know, due to the myeline of the nerve-fibres. If, however, the specimen examined is sclerosed, the portion thus affected presents a translucent appearance, and stands out in clear distinction from the normal structure. This is shown in the figure A, in which there is sclerosis strongly marked in the left lateral column, and slightly so on the right side. For the more satisfactory examination of this appearance, however, it is necessary to carry out the entire process of staining and clearing, as recommended by Dr. Lockhart Clarke. When this is done the sclerosed part of the cord is found to have taken the carmine dye more deeply than the normal structure. On a close examination the reason for this deeper coloration is found to be due to the closeness of the tissues, and to the increased number of the nuclei of the neuroglia. This increase in the number of these bodies is accounted for by the fact that sclerosis is the form of inflammatory degeneration in which the normal structure is replaced by a connective tissue and its ultimate modification, neuroglia. Under the microscope the sclerosed portion of the cord or brain is seen to consist of fine connective fibres, with an increased number of nuclei interspersed here and there. The divided nerve-tubes with their axis cylinders are relatively diminished in number. For the most part, however, these are almost entirely wanting, except in those places where the sclerotic process has not been carried to its furthest extent. This latter condition is seen on the right lateral and both the anterior columns of the section represented in figure A.

Drs. Wilks and Moxon consider that 'sclerosis and grey degeneration should be distinguished as different conditions—the former originating in the neuroglia, the latter in the proper nervous elements.' The term 'grey degeneration' used in this quotation accords with that used by Charcot.

There seems to be good reason for including this pathological condition among the *myelites*, as has been done by Charcot and others. It is without doubt the result of a subacute form of inflammation.

Colloid degeneration.—A good deal of confusion exists with regard to this form of degeneration, due mainly to the varying descriptions given of the appearances which it presents. Colloid degeneration has been often mistaken for amyloid degeneration, and *vice versa*. The frequency of this last form of change has, there is reason to believe, been overstated, in consequence of many observers having mistaken globules of spirit which have been introduced during the clearing process, for morbid products.

Under the microscope colloid degeneration consists of bodies

of variable size, mostly circular or oval in shape, with a smooth outline, perfectly homogeneous and nearly translucent. These bodies mostly lie in cavities slightly larger than themselves (see figure B); but sometimes they become detached, and then may settle in any fissure or outside the edge of the section. They do take the carmine dye, not, it is true, to such an extent as the true bioplasm, but still in an indubitable manner. It would seem then, from this fact, that these bodies consist of an albuminous substance; that, in fact, they are the nuclei of the neuroglia—according to Beale, the bioplasts—which have lost their power of forming the neuroglia, and have continued to grow simply as masses of albumen. The contiguous parts, in the meantime, not receiving their due nutrition from them, have wasted, and hence the cavities in which they are found and from which they are sometimes removed.

Amyloid degeneration.—This form of degeneration has been so fully described by various pathologists, more especially by Dr. Arlidge, that there is no need to enlarge upon it here. Suffice it to say that Dr. Arlidge considers that it consists in the deposition in the brain substance of amyloid bodies, which undergo a calcareous change and are the source of what he has termed ‘brain-sand.’

The appearance of amyloid bodies in the nervous tissue is very rare, and, as before stated, globules of spirit present with careful focussing a concentrically-ringed appearance which has often caused them to be mistaken for amyloid bodies, that being the appearance said to be peculiar to these bodies.

Miliary degeneration.—This form of lesion, as already observed,¹ was in the first instance designated miliary sclerosis. It consists of a morbid change in the neuroglia, presenting the appearance of millet-seed patches, and at first sight seeming to be simple holes in the tissue. These spots or patches may be either single or in clusters, or, as sometimes happens, two or more coalesce, thus forming patches more or less lobulated. In whatever shape or form it appears, however, there is always present, and taking the place of the normal tissues, a greyish-white granular and homogeneous substance. This substance is almost perfectly transparent, and is in no degree affected by the carmine dye. This form of degeneration would seem not to be inflammatory in its nature, but rather a simple process of retrograde metamorphosis. Commencing in the nuclei or bioplasm of the neuroglia, the nature and composition of which becomes essentially altered, it affects all the surrounding tissues, causing their destruction and the deposition in their place of the above-mentioned

¹ See footnote, p. 54.

granular homogeneous material. The only structures which do not seem to be so readily affected by this change are the blood-vessels, which may sometimes be seen crossing the patches. Some of these minute patches present, around the margins, appearances which lead one to the conclusion that the granular translucent masses contained in them have commenced to set up, by their presence, a certain amount of irritation. This is seen in an increase of the marginal neuroglia, with a slight degree of condensation of the contiguous structures.

The miliary form of degeneration is the form most commonly met with. It is to be found in specimens of nervous tissue taken from many and varying forms of disease. Dr. Kesteven in his description of the lesion mentions eighteen different diseases in which he has found it, and my own observations would add many more. As compared with colloid degeneration, miliary degeneration would seem to be due to a somewhat similar process, which, however, in its production assumes a much more active condition than in the production of colloid. It is also much more widely spread in the nervous tissues than is colloid. Figures C, D, show this form of degeneration when seen under high powers; figure E represents an entire section of the cord thus affected.

Granular degeneration.—This form of degeneration was fully described by Dr. Lockhart Clarke in Beale's 'Archives of Medicine.' It affects the central parts of the spinal cord and the grey matter of the medulla oblongata. It consists of large irregular cavities, which, when seen in a cord which has simply been divided, appear to contain a colourless jelly. In thin sections this is found to be translucent, and under high powers it is seen to be made up of a confused mass of granules, broken-down cells, fine fibres, and, according to Dr. Clarke, of corpora amylacea. Blood-vessels are sometimes seen crossing these masses. Where the vesicular part is affected by it the nerve-cells have entirely disappeared. This form of degeneration is found in many cases of paraplegia, particularly those accompanied by muscular wasting. Granular degeneration does not seem to be due to inflammatory action, but rather to impaired nutrition.

Insular atrophy.—This pathological condition cannot be designated a 'degeneration' in the sense in which that term has been used in the foregoing descriptions. It is more truly an atrophy. It does not consist in a substitution for the normal nervous constituents of any lowly-organised or other material. The appearances presented by it are as follows: In thin sections of nervous tissue, simply mounted in glycerine, and which contain this form of disease, small spots may be seen with the unaided

eye, which appear to be more translucent than the rest of the section. So far this lesion resembles sclerosis. When, however, the sections are stained and cleared, the resemblance between the two morbid conditions is found to cease; whereas in sclerosis the part affected is more deeply stained than the rest of the section, in insular atrophy exactly the reverse condition holds. The affected parts are here found, alone in the whole section, to have resisted the carmine dye. On examination of these patches with a high power ($\frac{1}{8}$ inch), they present the appearance of a skeleton leaf; the fine fibres of the neuroglia are seen with here and there a stained nucleus, but the contents of the nerve-tubes, the myeline and axis cylinders, are entirely wanting, large circular cavities being left in their place. The position of this lesion varies in each consecutive section, proving that the depth in the nervous tissue of each patch is not greater than its breadth, and that it is not persistently confined to any particular fasciculi of nerve-fibres.

The explanation of this pathological condition is not easy to find, as it is difficult to meet with satisfactory proof of any apparent solution of the difficulty, or to assign the reason why at isolated points in their course certain fasciculi of nerve-fibres should suddenly lose their myeline and axis cylinders, or why at these points the nuclei of the neuroglia should also to a great extent disappear. It is just within the limits of possibility that the explanation of this morbid condition may be, that the appearances which it presents are the results of a more acute and rapid form of decay than is carried on in the colloid and miliary degenerations, the process of decay being in this case of such rapidity and completeness as not to allow of the deposition of any retrograde product in the place of the structures which are removed.

The lesions above described, with the exception of granular degeneration—namely, sclerosis, colloid, miliary degeneration, and insular atrophy—may be coexistent in the same case. In the nerve-centres from a case of paralysis agitans, kindly sent to me by Dr. Dowse of the Highgate Infirmary for examination, these lesions were all found: not, however, uniformly throughout them; for the insular atrophy was confined to the cerebellum; the sclerosis to the posterior and lateral columns of the medulla in their cortical portions; the colloid to the cord and medulla; the miliary form alone pervading all these regions of the nervous centres.

Paralysis agitans would seem to be the disease of nervous degeneration *par excellence*; for in addition to the above mentioned, the nerve-cells in this case presented the various forms of degeneration to be hereinafter described.

Nerve-cells.—The degenerations of the nerve-cells have received names which depend upon the appearances presented by them. The characteristics usually described as belonging to the healthy nerve-cells being essentially such as are dependent on their translucent and homogeneous appearance, any variations in these characters are noticed, and it is for this reason that the names given to the degenerations of the nerve-cells are such as are descriptive of changes in these properties. They are called pigmentary and fuscous. There is, however, reason to believe that these different forms merely represent different stages in a general process of degeneration.

Fuscous degeneration.—This is the first step in the process, the end of which is seen in pigmentary degeneration of the nerve-cells. The most common character which this form of degeneration presents is shown in an inability of the cell, with the exception of the nucleus and sometimes of the parts immediately contiguous to it, to take the staining of the carmine. The parts not so stained have a yellowish-brown tint, due to the formation in them of similarly-coloured granules. The nuclei of these cells seem to be more brilliantly coloured by the carmine than is usual, but most probably this is due to the contrast between them and the rest of the cells. In the more advanced stages of this fuscous degeneration—when, in fact, it is merging into the black pigmentary form—the nuclei and nucleoli become much enlarged, and themselves become pigmented. The steps of this process may be traced in every case where this degeneration is found; they are—a gradual decrease of the staining area, a deposition in the unstained part of yellowish-brown pigment granules which gradually accumulate round the nucleus, the nucleus itself becomes enlarged and granulated, the granules become darker and darker, the processes disappear, finally there is nothing more left than a conglomeration of black granules.

Pigmentary degeneration.—When this form of degeneration is carried to its furthest extent, the nerve-cells thus affected appear to contain simply collections of granules of black or deep brown pigment. The granules thus collected seem to be so loosely attached to one another as to give one the idea that they would easily fall apart. There is no doubt that this condition represents a state of death, so far as the physiological functions of the nerve-cells are concerned.

The process of degeneration in the nerve-cells would seem to be in some degree parallel to the colloid form of degeneration of the neuroglia above described. The nuclei of the nerve-cells seem to have lost their power of elaborating and supplying to the rest of the cell the necessary nutriment, and instead, become themselves

enlarged. It is in consequence of this loss of power on the part of the nucleus that the cell undergoes this degeneration, the first step of which is the loss of that power peculiar to the healthy structure of taking the carmine dye. The process above described seems at times to vary somewhat. Cells are sometimes to be found in which the commencement of the degeneration, though still in the nucleus, is of a more acute and rapid kind. In these cases the nucleus of the cell is found to be the only part which does not take the carmine. It would seem that in this case the alteration in the composition of the bioplasm of the nucleus is more suddenly brought about, and the process of degeneration is brought to a climax at that point before the rest of the cell has time to be affected. This is shown in cells which have the appearance represented in figure G, in which the central parts of the cell, the nucleus included, are colourless and slightly granular, while the parts furthest away from the nucleus have retained their power of imbibing the carmine. The remainder of the process in this case is similar to that described in the first form, and the same conclusion is eventually reached.

Dr. Major has pointed out that changes similar to these are found in senile atrophy, and there can be no doubt that degenerations of the nerve-cells are due to impaired nutrition caused by a vitiated supply or condition of the blood.

In some parts of the brain—as, for example, the *locus niger* of the crus cerebri—collections of nerve-cells are to be found which are full of pigment. These cells, however, have not lost their processes, and are certainly not so manifestly morbid as are those described above. From the fact, however, that this pigmentation is not nearly so apparent in the brain of the seven months' fœtus, it may be doubted whether even this condition is of necessity normal.

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Fig. A

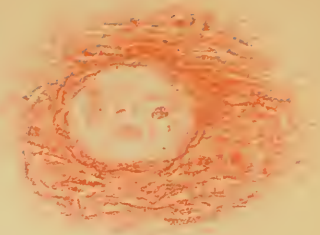
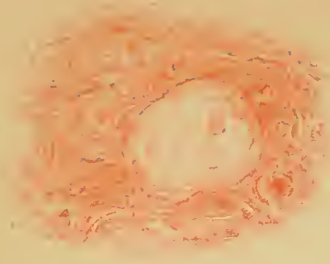


Fig. B

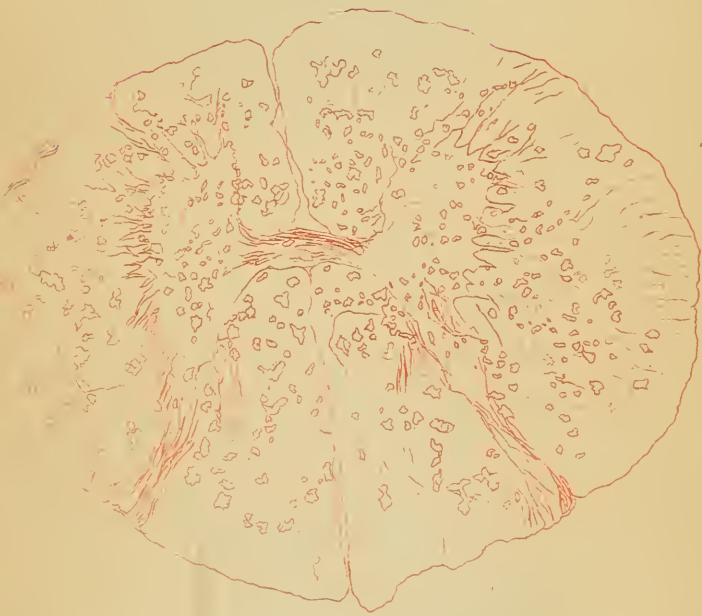


C

D



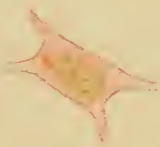
E



F



G



H



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EXPLANATION OF FIGURES

ILLUSTRATING PAPER ON THE DEGENERATIONS OF NERVOUS TISSUE.

- A. Section from lumbar region of a case of paraplegia, not stained or cleared, showing the contrast between the sclerosed and the healthy portions, $3\frac{1}{2}$ -inch glass \times 12 diameters.
- B. Nervous structure containing colloid bodies from medulla oblongata of a case of paralysis agitans, $\frac{1}{8}$ -inch glass \times 420 diameters.
- C, D. Miliary degeneration from cord of a case of tetanus, $\frac{1}{8}$ -inch glass \times 420 diameters.
- E. Section of cord from case of tetanus, showing the distribution of the miliary degeneration, $3\frac{1}{2}$ -inch glass \times 12 diameters.
- F, G, H. Nerve-cells, showing the various forms of degeneration, from a case of paralysis agitans, $\frac{1}{8}$ -inch \times 420 diameters.

ON THE
DIAGNOSIS AND TREATMENT OF
PLEURITIC EFFUSION.

BY

F. DE HAVILLAND HALL, M.D.

- I. The Physical Signs of Pleuritic Effusion.
- II. The Diagnosis of Pleuritic Effusion.
- III. The Treatment of Pleuritic Effusion.
- IV. Cases.

I. The Physical Signs of Pleuritic Effusion.

Since the days of Hippocrates pleurisy has always attracted the greatest attention from the medical profession, and in the diagnosis and treatment of no disease has greater progress been made than in this—this advance, however, chiefly dating from the time of Laennec, who by the introduction of the stethoscope threw light on a hitherto obscure affection, and enabled physicians to diagnosticate with almost absolute certainty the presence of fluid in the chest; whereas before auscultation was practised, pleurisy, pneumonia, and other causes of solidification of the lung, were very generally confounded with one another; and no wonder, when we consider how difficult it often is to distinguish between these diseases by the symptoms alone; and great praise must be awarded to the pre-auscultation physicians, who by inspection and by the most careful attention to the general condition of the patient so frequently arrived at a just conclusion. It will be to be regretted if we, living in these so-called enlightened days, by depending too much on such adventitious aids as the stethoscope, laryngoscope, ophthalmoscope, should neglect that earnest attention to the patient himself and his subjective symptoms which

so largely contributed to the success of our medical predecessors. I cannot but think that this point requires to be most strongly impressed on the rising generation of practitioners.

As an example of the means of diagnosis employed by one of the greatest physicians of the last century, I cannot do better than quote the words of Dr. Matthew Baillie:¹ 'Empyema may be distinguished with a good deal of certainty after inflammation of the pleura or of the lungs; by rigors having taken place; by a remission of the pain; by the cough and difficulty of breathing continuing; and by the person being able to lie more easily upon the diseased side than the other. There is sometimes a very evident enlargement of the side where the matter is accumulated, and always a want of that hollow sound on striking with the fingers the side where the empyema is, which takes place upon striking the chest when the lungs are sound.'

After these few introductory remarks, I will proceed with the proper subject of my paper—namely, the diagnosis and treatment of pleural effusion.

Before proceeding to discuss the various physical signs of pleural effusion, I would insist on the importance of having the trunk bare to the waist in the examination of any pulmonary affection. In the case of men and children there is not much likelihood of this being objected to, but some sensitive women may demur to the procedure; but for my own part I would refuse to give a positive opinion unless the opportunity of a thorough examination were conceded to me. While I am on this subject it may be advisable to quote a short paragraph from Dr. Sturges' able work on Clinical Medicine: 'A hasty or partial examination of the chest is worse than useless. It must be seen fully, and examined throughout by all the methods. It is not uncommon to see the operation performed in a manner which, however it may affect the patient, is ridiculously incomplete. A small part of one side of the chest is exposed by the patient pulling open some portion of his clothing. Into this aperture the stethoscope is thrust, and the practitioner assumes the attitude and aspect of listening as for some actual message which is to reach his ear from the assumed seat of the disease. But the stethoscope does not yield information in this sense; to such a listener, however he may conceal the fact, we may be sure that it says nothing. Unless the examination can be thorough, with the thing to be examined fairly in view, and each sign corroborated by the others, it is far better and honester to omit it altogether. Truer results are arrived at without it by trusting entirely to the more general symptoms.'

¹ Works of Matthew Baillie, M.D., vol. ii. p. 56.

Dr. Wardell of Tunbridge Wells, to whom I am indebted for much valuable advice in the treatment of pleural effusion, several years ago impressed upon me the necessity of a careful examination of the chest in cases of dyspnoea by narrating to me his experience of a patient who had been told by two hospital physicians that he had little the matter with him, whereas on examining the base of the left lung there were the evidences of a considerable pleural effusion, the mistaken diagnosis being due to the fact that the physicians in question had contented themselves with percussion and auscultation over the front and upper part of the chest only. This case is reported at full in the 'British Medical Journal.'

I myself very nearly fell into the same mistake recently, and as there is as much to be learned from the mistakes as from the successes in diagnosis, I will briefly give the notes of the case.

Thomas Roberts, aged 50, came as an out-patient to the Metropolitan Dispensary on October 2, 1875, complaining of dyspeptic symptoms; but as he made no remark about his breathing, I did not examine his chest. However, when he came the third time, on October 16th, as there appeared to be a little dyspnoea, I made a physical examination, and found that the left back was dull up to the angle of the scapula, and impaired up to the spine; no air entering at base; feeble breathing above; absence of vocal fremitus and resonance. Right side, good vesicular breathing. Has had a cough three weeks; breathing short since then; no pain; illness came on gradually.

Ordered—Potass. iod., gr. iij.; syr. ferri iod., ʒj.; aq. menth. pip. ad ʒj., ft. hst. ter die sumendus.

R. Tr. iodi; aquæ aa. ʒiiss, ft. lin. To be painted every night all over the affected side.

October 27th.—Improved resonance and a little air entering at base. Friction sound audible opposite angle of scapula.

April 29, 1876.—Resonance almost complete at base. Only very slight friction. Has dragging pain on taking a full inspiration.

I saw the patient frequently between October and April, and he made steady progress towards recovery. When he was discharged he appeared in very good health. For the last four months he took cod-liver oil in addition to the mixture, and he continued painting his side the whole time.

Now, if I had contented myself with a superficial examination of this man's chest, confining myself to the apices in front, I should have failed to have detected the pleural effusion, and I might have gone on treating his dyspeptic symptoms, which were evidently due to the pleural affection, as they disappeared as the fluid cleared up, till the man became tired of attending without

gaining benefit. So I say, strip your patient and examine the bases of the lungs carefully.

Dr. Aitken,¹ alluding to latent pleurisy, remarks, 'Whenever, therefore, the least suspicion exists of disease in the chest, especially in elderly persons or those liable to constitutional affections, percussion and auscultation must never be neglected,' to which I would add, that particular attention should be paid to the posterior part of the chest.

In the pursuit of any inquiry too much stress cannot be laid on proceeding in a methodical way, and in the investigation of disease this is especially important, as there is no such frequent cause of mistake in diagnosis as rashly jumping at a conclusion from one or two physical signs alone, and not going through a systematic examination. To obviate this it is advisable to have a definite plan on which to proceed; the most rational order to my mind is the following: 1. Inspection; 2. Palpation; 3. Mensuration; 4. Percussion; 5. Auscultation. I propose to take each of these methods separately, and to consider their value in the diagnosis of pleural effusion.

1. *Inspection*.—From the earliest ages sight was the sense which was first employed to elucidate disease, and so accordingly all the old writers mention the alteration in the shape of the chest produced by a large pleural effusion. If there be a considerable amount of effusion into the pleural cavity, the affected side will tend to become rounder, and the sternum is pushed forward, so that the outline of the sound side is also altered, though to a less degree; but these changes will be described more conveniently when we come to consider the method of mensuration. An encephaloid or other tumour may, however, also cause an enlargement of the chest, and Sir Thomas Watson² mentions an instance in which it was due to the extravasation of blood from one of the intercostal arteries and its coagulation in the pleural cavity. According to Dr. Fuller,³ the idea formerly held, that bulging only occurred in cases of empyema and not when the effusion is serous, is inconsistent with fact: 'It occurs more frequently under these circumstances than when the effusion consists of serum, for the simple reason that the costal pleura and subjacent structures are commonly involved in such cases, but there is no necessary connection between bulging and empyema.' Should the effusion be encapsulated, there may be local bulging and the conformation of the rest of the chest unaffected. The obliteration of the intercostal space is due to the cedema of the intercostal muscles

¹ Science and Practice of Medicine, 3d ed. vol. ii. p. 628.

² Lecture on the Principle and Practice of Physic, 5th ed. vol. ii. p. 123.

³ Diseases of the Lungs, p. 182.

and consequent loss of tone. Another significant sign is the loss of movement on the affected side, the chest remaining in the condition of deep inspiration. But the most important of all the signs derived from inspection is the dislocation of the apex of the heart, which I have seen beating in a case of left pleurisy close to the inner side of the right nipple. Displacement of viscera takes place to a much less extent in the pleurisy of children, owing to the yielding nature of their thoracic wall; but that it does take place is testified to by many authorities, and in my notes of the case of a child in the Westminster Hospital I find it recorded¹ that the heart was beating to the right of the sternum. Dr. Walsh² says in cases of left effusion the organ may pulsate outside the right nipple, and thirty-six hours will sometimes suffice to produce this amount of malposition. I have never seen so extreme a case as this, and some authorities even go so far as to doubt its possibility. This sign is especially valuable in cases in which the pleurisy is secondary to pneumonia, and is the one which would determine me to do thoracentesis if there were very great dyspnoea. In the following case it was only the absence of this displacement which prevented an erroneous diagnosis.

E. D., aged 20, admitted into the Westminster Hospital with the symptoms of an acute attack of lobar pneumonia. Physical examination, however, showed that in front, over the base of the right lung, there was a little crepitation, with distant and feeble bronchial breathing; posteriorly there was only very feeble breathing, vocal fremitus and resonance were diminished, the heart was not displaced, so that manifestly there could not be sufficient effusion to account for the comparative obliteration of the signs of air entering the lung. The post-mortem examination cleared up the difficulty, by showing that the bronchial tubes were occupied by a croupous exudation, which fully accounted for the anomalous physical signs.

2. *Palpation*, in my opinion, is one of the most important means of arriving at a correct diagnosis in pleural effusion, as in all such cases, except when there is only a very slight amount of fluid effused, there is obliteration of vocal fremitus, which serves to distinguish the dulness of pleurisy from that of pneumonia, as in the last-mentioned disease there is considerable increase of the tactile vibrations; but, as the case I have just related proves, this is liable to a fallacy, as any cause which prevents air entering a main bronchus will thereby abolish vocal fremitus over the affected part. In feeble persons, and in persons whose voices are hoarse or whispering, palpation will give us no information. It should

¹ See case No. 4, at end of paper.

² Diseases of Lungs, p. 262.

also be borne in mind that, as a rule, in health the vocal fremitus is more marked on the right side of the chest than the left. In the upper part of the chest, where the compressed lung is above the effusion, there is intensification of the vocal fremitus. By palpation, too, the displacement of the liver and spleen downwards may be recognised, and fluctuation may be detected in the distended intercostal spaces—a useful sign in right-sided pleurisies to distinguish where the dulness due to effusion ends and where the dulness due to liver begins, which is of course a matter of great importance when it becomes a question of tapping the chest. Dr. Ward¹ of New York mentions a modification of this, which consists in an assistant percussing in the second or third intercostal space, whilst one's finger is placed in the seventh intercostal space, when the feeling of fluctuation may distinctly be felt.

3. *Mensuration.*—Various appliances have been contrived for the purpose of obtaining an accurate measurement of the chest. Tapes give a very fallacious estimate, as the alteration caused in the thorax by the presence of fluid makes the affected side rounder, without of necessity increasing the circumference. M. Woillez by means of his cyrtometer enables us to obtain an outline of the chest, so that the healthy and affected side can easily be compared. Dr. Gee² suggests that 'a cheap and perfect cyrtometer may be made by two pieces of composition gas-pipe drawn out to a diameter of the eighth of an inch, and united by a piece of caoutchouc tubing.' All that is needed besides this is a pair of callipers to get the antero-posterior diameter accurately. Mr. Hawksley has made for me a modification of Dr. Gee's cyrtometer. Instead of having a pipe, Mr. Hawksley has employed a flat band of pewter united by a stout elastic webbing. The pewter is very pliable and possesses very little resiliency, and the webbing allows the cyrtometer to be accurately fitted to the spine.

In a case of pleural effusion which I reported in the 'Lancet' of February 1, 1873, the cyrtometrical tracings were very characteristic, and showed that the affected side was nearly the segment of a circle. The falling in of *both* sides after thoracentesis to the amount of 77 ounces was very apparent in the tracing made after the operation.

If mensuration is resorted to after thoracentesis has been practised, of course a considerable falling in on the affected side will be remarked, and in most cases this is so perceptible that the eye on glancing at the chest at once notices it; and it will also be generally found that the shoulder on the sound side is higher than the other. But if the operation has not been delayed till the lung, bound down by adhesions, is unable to expand, the patient

¹ Lancet, October 10, 1874.

² Auscultation and Percussion, p. 11.

may be comforted with the assurance that his chest will improve very much in shape, and may ultimately become almost normal. A child¹ who was under my care last year suffering from empyema, and who was considerably deformed after paracentesis was done, came under my observation a short time ago, and I was pleased to find that his spine was perfectly straight and the chest well formed. The shrinking of the affected side is often made more marked by the compensatory enlargement of the sound side.

4. On *percussion* an absolutely dull sound is returned whenever there is pleural effusion to any amount, and a practised observer can both feel and hear the difference between percussing over a lung solidified by pneumonia and over a chest full of fluid, as the latter gives a much flatter sound, and resembles the note that would be obtained by percussing over the thigh. In pleural effusion there is generally also a difference between the note obtained over the front of the chest from that heard behind. In front, the transition from the absolute dulness of the effusion to resonance is immediate, so that one can with confidence mark out the exact limits of the effusion; whereas behind, the resonance becomes gradually greater when the chest is percussed, beginning from below upwards. This appears due to the fact that the patient usually lies on his back, so that unless restrained by adhesions, the natural tendency of the fluid is to gravitate to the back, thus forming a thicker stratum at the lowest part of the chest, which gradually becomes thinner on ascending. In some cases there may be an alteration in the area of dulness by changing the position of the patient, but this, which is the rule in hydrothorax, only happens exceptionally in pleural effusion, as the fluid in this case is prevented by adhesions from obeying the law of gravity. If there be a large amount of fluid, and the lung be pressed up to the apex, a boxy or tympanitic note will be obtained over this part of the chest, called by some Skodaic resonance, after the celebrated physician who described it. The various theories as to the cause of this phenomenon are discussed by Dr. Walsh² at length in his work on Diseases of the Lungs. When the effusion is still larger, there may be absolute dulness all over the affected side; and in one instance of left pleurisy³ the dulness extended over the sternum to the right side. The heart in this case was necessarily much displaced, the apex beating half an inch below, and just to the inner side of the right nipple.

By percussion may be detected also the detrusion of the liver and spleen.

5. *Auscultation*.—The auscultatory phenomena of pleural effu-

¹ See case No. 5, at end of paper.

² Diseases of the Lungs, 3d ed. pp. 82, 83.

³ Case No. 4.

sion, as laid down in text-books, consist in an absence, more or less complete, of vesicular breathing, with frequently bronchial breathing along the spine; feebleness, or absence of vocal resonance, or the presence of ægophony, and puerile breathing on the sound side: but these signs vary so much in different cases that I think it advisable to consider them seriatim. The rule undoubtedly is, that as effusion takes place the breathing tends to assume a bronchial character, until so much fluid has been poured out that air is no longer able to find its way into the lung, thus causing an entire absence of breathing sounds, except along the spine; and it is seldom that the large bronchi in this region are so compressed as to prevent the entrance of air and consequent bronchial breathing. In children it is unusual to find an entire absence of breathing sounds, even when there is a large pleural effusion; and it is most important that this fact should receive the recognition it deserves, as doubtless many children have been allowed to die from exhaustion consequent on the presence of pus in the pleural cavity, owing to the fear of the medical man in attendance to perform thoracentesis, as there was evidence of air entering the lung. In one case which came under my observation two ounces of pus were drawn off by the aspirator, when the fluid ceased to flow, probably from a flake of lymph blocking up the tube. In spite of other manifest signs of effusion and the existence of hectic, no further operation was resorted to, as there was a certain amount of bronchial breathing over the affected side. The autopsy showed the presence of 27 ounces of pus. Comment is here unnecessary.

MM. Rilliet and Barthez¹ lay especial stress on the constant occurrence of bronchial breathing in the pleurisy of children.

In some cases of pleural effusion occurring in old or cachectic persons there may be a sudden and complete cessation of the respiratory murmur, but after the lapse of a few days breathing sounds may be heard, although the effusion is undiminished or even somewhat increased. Laennec² explains this by saying that the rapid effusion of fluid choked as it were the lung, so that it was unable to admit the air in respiration, but after a few days the lung becomes accustomed to the presence of the fluid and resumes its work.

Vocal resonance varies with the amount of fluid present. If it is large, there is an entire absence of voice resonance; but if it be small, there may be a bronchophonic character communicated to the voice sound, or in some instances the peculiar bleating noise called ægophony may be heard, though this is only said to occur when there is a thin layer of fluid with compressed lung behind,

¹ Mal. des Enfants, tom. i. p. 554.

² Ausc. Méd., tom. iii. p. 314.

and then it is audible at the upper limit of the effusion. But this is a sign on which very little stress can be laid, as it is generally absent; however, when present, I think that it may be looked upon as indicative of the presence of a small amount of effusion, as I have not heard it except under these circumstances.

Dr. Bacelli¹ has found that vibration of the voice is transmitted to a greater distance and more perfectly in the direct ratio to the thinness and homogeneousness of the fluid in the pleural cavity. Pus and serum are nearly of the same density, and yet sound is more readily transmitted by the latter than the former. Dr. Bacelli explains this difficulty by the theory that waves of sound passing through a heterogeneous medium like pus, which contains corpuscles, become lost by refraction, interference, and dispersion, in a similar way to rays of light under analogous circumstances. The variable transmission of respiratory and vocal vibration according to the nature of the effusion, form, according to Dr. Bacelli, 'the absolute criterion for differential diagnosis.'² A true empyema is the worst conductor of sound of all the effusions.

II. *The Diagnosis of Pleuritic Effusion.*

The affection which more commonly than any other is confounded with pleural effusion is pneumonia. As one point in the diagnosis, Laennec³ mentions the rapidity with which dulness may be met with in pleurisy and the extent of surface involved. In the case of pleurisy it frequently happens that in the course of a few hours from the attack the dull sound exists over the whole affected side, or at least over its lower half, a thing which is never, or almost never, observed in pneumonia. Except, however, in those rare cases in which pneumonia is complicated with a croupous exudation into the tubes, the diagnosis between it and pleurisy is comparatively easy, the absence of vocal fremitus, vocal resonance, and breathing sounds in pleural effusion contrasting markedly with the increased vocal fremitus, bronchophony, and tubular breathing met with in pneumonia. Then again, in pleurisy there are alterations in the shape of the chest, bulging of the interspaces, and displacement of the viscera, all which are absent in pneumonia. Moreover, in pneumonia the sputa are rusty, whereas if there be any expectoration in pleurisy, it consists of a frothy mucus. Lastly, in pneumonia there is an alteration in the pulse-respiration ratio, the proportion of 2 : 1 being not uncommonly met with. According to Dr. Gee,⁴ 'the two diseases

¹ Archivio di Medicina, Roma, 1875.

² London Medical Record, July 15, 1876.

³ Auscultation Médiante, tom. ii. p. 312.

⁴ Auscultation and Percussion, pp. 214, 215.

from which it is most difficult to distinguish pleural effusions are cancerous and hydatid tumours within the chest. Cancer of the lung causes no enlargement of the affected side, and the dulness of cancer does not usually follow the laws which have been explained with regard to effusions into the pleura. Hydatid tumours are uncommon, and hardly admit of diagnosis until the peculiar membrane is expectorated, or removed during the operation of paracentesis.'

Dr. Walsh¹ says that 'infiltrated cancer can only be confounded with diseases lessening the bulk of the lung;' but if the growth starts from the root of the lung pressing upon the main bronchi, the difficulties in the diagnosis are very great, as in this case equally with pleural effusion there would be absence of vocal fremitus. Dr. Clifford Allbutt² has the following remark on this subject: 'Not to speak of pneumonia consolidation, three times at least I have been distinctly wrong in diagnosing pleural effusion where intra-thoracic tumour was present, and many times my doubts have only been removed by the result of exploration.'

As regards the diagnosis of hydatid tumour, Dr. Murchison³ is of opinion that hydatid tumour is 'more likely to be regarded as an example of pleuritic effusion than pleuritic effusion mistaken for hydatid. The hydatid is mainly distinguished by its insidious growth and by the absence of constitutional symptoms. The possibility of a hydatid tumour co-existing with pleuritic effusion must not be lost sight of.'

Up to the present time I have not had a case of cancer or hydatid of the lung under my observation in which the fact has been verified post-mortem. In the beginning of the year I had under my care in the Westminster Hospital a man suffering, as I thought, from pleural effusion of a tubercular origin, but on making the autopsy, a mass of mediastinal lympho-sarcoma, such as Virchow⁴ describes, was discovered. In this case the pleural effusion obscured the signs due to the growth; but I have referred to it, as during the whole time the patient was under my care I was unable to satisfy my mind as to the cause of his cachectic appearance and loss of flesh, the pleural effusion, which was never very large in amount, not being sufficient, in my opinion, to account for these symptoms. A localised empyema situated on the left side of the chest may have a certain amount of impulse communicated to it; but, as Dr. Fuller⁵ says, 'the presence of well-marked symptoms of empyema, backed by the feebleness of the heart's

¹ *Op. cit.*, p. 539.

² *Medical Times and Gazette*, May 9, 1874.

³ *Lectures on Diseases of the Liver*, p. 60.

⁴ *Krankhafte Geschwulste*, p. 733.

⁵ *Op. cit.*, p. 184.

sounds over the seat of pulsation, by the absence of aneurismal thrill and murmur, by the equality in the radial pulse on the two sides, and by the absence of symptoms denoting pressure on the spine, the trachea, the œsophagus, the larger veins, and the recurrent nerve, ought at once to remove all doubt as to the real nature of the mischief.'

An enlarged liver may be mistaken for a pleural effusion. The points to be borne in mind in the diagnosis are these:—

1. As a rule, if the liver enlarges, the enlargement will be most noticeable in the downward direction, so that dulness over the right base of the chest is *a priori* more likely to be due to pleural effusion than to enlargement of the liver.

2. In pleural effusion the dulness extends higher behind than in front, and *vice versa* in the liver affection.

3. In pleurisy with effusion there is obliteration of the intercostal spaces; but when the liver is enlarged, though the whole side may be protuberant, the interspaces are normal.

4. There is a line of semi-resonance extending between the effusion and the liver, which of course is wanting when the dulness is due to the increased size of the liver. Lastly, the area of resonance is not increased by a deep inspiration in pleural effusion, but is increased when the dulness is due to liver enlargement.

On the left side an enlarged spleen may be mistaken for a pleural effusion, but the diagnosis rests on much the same ground as in cases of enlarged liver—that is to say, when the spleen is enlarged the dull area is decreased by taking a full inspiration; there is no bulging of the intercostal spaces; the posterior part of the chest is not much affected; and lastly, the heart is not displaced laterally but upwards.¹

A patient whom I exhibited before the Medical Society last winter was an instance of a possible source of fallacy. In this man there was apparently complete transposition of the viscera, so that had he been suffering with pulmonary symptoms, a careless observer might have diagnosticated effusion into the left pleural cavity, owing to the hepatic dulness in that region, the apex of the heart beating to the inner side of the right nipple.

There is one more disease which is liable to be mistaken for pleurisy with effusion, and this is phthisis. The points, however, to be borne in mind are that phthisis, as a rule, commences in the apex and advances from above downward; the dulness is never so absolute as in pleural effusion, and there is shrinking of the

¹ For the remarks on enlargements of the liver and spleen, and their diagnosis from pleural effusion, I am greatly indebted to Niemeyer's admirable Text-book on Practical Medicine.

chest wall; and lastly, as Dr. Walsh¹ says, '*tubercle*, in its ordinary seat at the upper regions, cannot be confounded with effusion, which accumulates below;' and '*tuberculous disease of an entire lung does not exist without implication of its fellow: any amount of effusion may exist in one pleura, the other remaining unaffected. In effusion, the respiration is null or weak, distant and diffused blowing; in phthisis, superficial, of various qualities, and attended with rhonchi.*' On the other hand, according to Trousseau,² in some cases of pleurisy all the stethoscopic signs of the third stage of tubercular phthisis may be met with: 'Nevertheless, the mode in which the disease commences and progresses, the dulness of the dependent parts, the displacement of neighbouring organs, the volume of the chest, the absence of lesions at the summit of the lungs—in a word, the general condition of the individual, usually enable us to form a diagnosis.'

The thermometer is of the greatest possible assistance in diagnosing the conversion of a serous into purulent effusion, as the evening rise is very marked in these cases.

M. Bouilly³ in '*Mouvement Médical*' has arrived at the following conclusions as regards the temperature in pleurisy:—

'1. There exists a pleuritic fever, the mean duration of which is from twenty-eight to thirty days, whether the effusion is abundant or moderate.

'2. Thoracentesis does not notably diminish the duration of the febrile condition shown by the thermometer, though it considerably diminishes its intensity.

'3. In serous pleurisy the thermometer does not afford the valuable results which it does in purulent pleurisy.'

The second axiom which M. Bouilly lays down I am not at all disposed to agree to as regards thoracentesis in empyema, however true it may be in cases of serous effusion; and an observation of Küssmaul's quoted by Wunderlich⁴ is quite in accordance with my experience: 'He noticed in cases of foetid and purulent febrile pleurisy (empyema) that after thoracentesis the temperature very quickly returned to normal. I have observed a similar case myself, whilst in a case of fibro-serous exudation (pleuritic effusion) the moderate fever which was present was not materially affected by the tapping.'

Dr. Bacelli maintains that the usual circumstances from which the diagnosis of empyema is inferred, viz., the duration of the effusion, the œdema of the side, the altered state of nutrition, and the presence of intermitting fever and of progressive anæmia, are insufficient for the purpose; and he asserts that, contrary to the

¹ *Op. cit.*, p. 280.

² Lectures on Clinical Medicine, vol. iii. p. 184.

³ See *Lancet*, May 3, 1873.

⁴ *Medical Thermometry*, p. 393.

opinion of Trousseau, the most violent and prolonged fever may accompany effusions which are not purulent.¹

III. *The Treatment of Pleuritic Effusion.*

As I do not intend to allude to the treatment of acute pleurisy in this paper, I will pass at once to the method I employ in pleuritic effusion.

In an ordinary case of pleurisy with effusion, when there exist none of the indications for thoracentesis to be hereafter mentioned, I always begin with the following treatment, if the symptoms be at all acute, and the temperature above 99° F. I order the patient to bed, and even in cases of latent pleurisy where there is little or no febrile reaction, quiet should be enjoined, as the process of absorption goes on so much more readily when the patient is at rest. After attending to the bowels, and securing a daily evacuation by means of a purgative if necessary—and if one be required, calomel or blue pill in combination with the extract of colocynth is the best—I then proceed to the more specific treatment, which consists in keeping the affected side thoroughly saturated with a weak iodine solution. I generally use one part of the tincture of iodine to three of water painted all over the side of the chest from the apex to the floating ribs, and I have a half jacket of flannel made and worn continuously, so that this becomes impregnated with the iodine, and helps to promote absorption. The internal remedies in which I put the greatest confidence are a combination of iodide of potassium and syrupus ferri iodidi, together with the use of cod-liver oil in debilitated subjects, and where it can be taken without upsetting the digestion; but in cases where the temperature is high, it cannot as a rule be borne. Blisters I do not advocate for universal application, but occasionally a blister will give a stimulus to absorption when that process is going on slowly, so that it is certainly worth while to try this form of counter-irritation in these cases. The blister should not be kept open, but allowed to heal as quickly as possible, as the good it effects takes place during the healing process. I have recently had a patient under my care in the Westminster Hospital in whom rest and the application of two blisters had the effect of removing a very large pleural effusion, the only medicine he had being a little liq. ammon. acetatis.

If the urine be small in quantity and high-coloured, a prescription containing tinct. digitalis, potass. iodidum, potass. acetas, and spiritus ætheris nitrosi has proved very useful in my hands.

Dr. Fuller² advises the use of the following solution externally :
℞. Hydr. perchlor., gr. iv.; tr. iodi, ℥vi.—℥j.; glycerini, ℥ij.;

¹ London Medical Record, July 15, 1876.

² Diseases of the Lungs, 2d ed. p. 210.

aquæ dest., ℥iv., M. ft. lotio. Or as an ointment: ℞. Hydr. perchlor., gr. iv.-v.; ungu. iodi, ℥iv.-vi.; adipis, ℥iv.-℥j. ft. ungu. And as a diuretic he gives a pill mass made up of digitalis, squills, and the pilula hydrargyri. This combination has met with the approval of many celebrated physicians. Dr. Matthew Baillie,¹ in speaking of pleural effusion, says: 'The medicine which I have found most beneficial has been mercury, combined with squills and digitalis. Five grains of the pilula hydrargyri, combined with one grain of the dried powder of squills and half a grain of the dried powder of digitalis, given twice or thrice a day, have in many cases under my care either very much mitigated or for a time removed the disease. There has been some advantage from the mercury affecting slightly the salivary glands. Squills and digitalis are by themselves much less efficacious than when combined with mercury.' Sir Thomas Watson² mentions this pill with approval, but advises less mercury—one to three grains of the blue pill, with the quantity of digitalis and squills stated above. But whatever treatment be adopted, the alliterative advice said to have been given by a celebrated living physician, of *beef and beer* being the best absorbents, requires to be followed; as, unless the patient's strength be maintained by good food and tonics, degenerative changes are apt to ensue, and a simple serous effusion become purulent.

This plan of treatment will be successful in a large proportion of the cases brought under one's notice; but in a certain number there comes a time when the question of thoracentesis is mooted. Dr. Fuller³ advises perseverance in the remedies to promote absorption, so long as the breathing is not seriously embarrassed and the general health does not decline; but for my own part, I am inclined to go farther, and say that if, after giving these remedies a fair trial—and I consider three weeks to be ample time—there were then no signs of absorption to any marked extent, I would advise the performance of thoracentesis; because, if done with the proper precautions, it is not a risky operation, and it is hardly fair to the patient to allow him to undergo a protracted illness, with the probability of imperfect recovery and a deformed chest, whereas, by accepting the responsibility of advising the operation, the physician would, in the vast majority of cases, have the satisfaction of seeing his patient rapidly gain ground.

Sir Thomas Watson⁴ is opposed to the operation in cases of serous effusion. He remarks: 'In simple pleurisy it ought never, in my judgment, to be performed, unless the life of the patient is, or seems to be, in jeopardy from the continual presence

¹ Lectures and Observations in Medicine, p. 180.

² *Op. cit.*, p. 138.

³ *Op. cit.*, p. 214.

⁴ *Op. cit.*, p. 142.

of the liquid within the thorax.' But in an addendum to his article on Pleurisy he says, 'The operation seems more extensively applicable than I had formerly supposed;' though his opinion as regards its use in simple pleurisy remains unchanged.

There are two sets of cases in which thoracentesis is required in pleurisy, when the effusion is believed to be serous:—

1. Those cases in which the effusion is so great as to threaten to be the immediate cause of death.

2. Cases in which the pleura seems unable to absorb the fluid, and the operation is undertaken to prevent the lung from being irretrievably bound down by adhesions.

As regards the first set of cases, the older physicians seemed to attach only a small degree of danger to an attack of pleurisy; and Dr. Louis went so far as to lay down a law that pleurisy is never an immediate cause of death. That this is far from being true, the instances narrated by Trousseau¹ are quite sufficient to demonstrate, as he says, that 'notwithstanding the famous law of Louis, it is possible to die, and to die suddenly, from acute pleuritic effusion.' The cause of the fatal termination has not yet been clearly made out; in some cases it is undoubtedly due to the dislocation of the heart so interfering with the proper performance of its functions as to cause sudden syncope. Dr. Evans,² in criticising the theory of MM. Blachez and Marrotte, that the obliteration of the pulmonary artery is the cause of sudden death in pleuritic effusion, suggests that the coagula thus formed in the pulmonary artery or its branches may be the cause of the imperfect recoveries so commonly met with in effusions of long standing.

Trousseau³ gives a most emphatic warning against trusting to the absence of oppression in breathing as an indication that there is no urgent necessity for operating; he says that 'oppression is one of the most deceitful of signs,' and goes on to say, 'It is from auscultation, and still more from percussion, that we must derive our most positive indications as to the opportune moment for performing paracentesis of the chest.' I have only notes of two cases in which paracentesis was done when the effusion was serous, and for the relief of dyspnoea. The first one has been reported by Dr. Wardell⁴ in his paper on Pleural Effusion; in this case, '63 ounces of clear greenish straw-coloured serum were drawn off to the patient's instant and great relief. That evening she could lie on the right side, which she had not been able to do for several weeks. For some days she appeared to pro-

¹ Clinical Medicine, vol. iii. p. 202.

² St. Thomas's Hosp. Reports, vol. ii.

³ *Op. cit.*, p. 266.

⁴ British Medical Journal.

gress favourably; she then, without apparent cause, became worse, and gradually sank on April 23d. Inspection was not allowed.' The other case I have already alluded to when discussing the subject of mensuration. This patient had left pleurisy, for which he was twice tapped; the first time 70 ounces of straw-coloured serum were evacuated, and nine days later 5 pints of clear straw-coloured fluid were let out. On each occasion the patient was suffering from great dyspnoea, and appeared in a very precarious condition; the relief he experienced was immediate, and the temperature fell. This is a typical instance of the good effect of paracentesis in serous effusion, as the patient eventually made a perfect recovery. One of the most usual of objections, and which would if true be a grave one, is that thoracentesis in a case of serous effusion, by admitting air, is likely to cause suppuration to take place in the pleural cavity; but the case I have just narrated shows that this is not necessarily so, and it would be easy enough to collect innumerable instances in which a chest full of serum has been tapped more than once without altering the nature of the secretion; in fact, I am prepared to go farther, and say that it is possible to convert purulent effusion into a serous one, and a case recorded in the 'Lancet' for December 6, 1873, will support this view. The notes are so interesting, and the treatment was so successful, that I venture to give an abstract.

Dieulafoy's aspirator was used in the first instance, and 106 ounces of thick inodorous pus were drawn off, and though there were no signs of the cavity becoming emptied, the operation was now stopped, as a violent fit of coughing came on. In a few days the fluid reaccumulated, and an incision was therefore made close to the former puncture, under a spray of carbolic acid. At least 10 pints of pus, of the same character as before, were evacuated. When the flow had diminished sufficiently, a broad piece of drainage tubing, about 4 inches, was inserted into the cavity, and a very large dressing of muslin and waterproofing applied over the opening, and the patient was loosely surrounded with cotton wadding prepared with carbolic acid, in order to prevent the putrefaction of the discharge. There was a very profuse discharge during the first forty-eight hours, but no decomposition occurred; the quantity rapidly decreased, its character changed, so that on the sixth morning it was entirely serous. On the eleventh day the discharge had ceased, and the opening into the pleural cavity closed up. About three weeks later the patient caught cold, and effusion into the pleural cavity again occurred, which proved to be of a serous nature, and was twice drawn off by Dieulafoy's aspirator. The patient eventually made a perfect recovery.

In the second set of cases in which thoracentesis is required,

the only *raison d'être* of the operation is that it should be done comparatively early, as 'every day that passes increases the liability of the lung to become bound down to the mediastinum by false membranes.'¹

I think that I may say, without fear of contradiction, that the modern school of medicine are unanimously agreed that thoracentesis should be performed immediately that the presence of pus in the pleural cavity is suspected, and, as I mentioned in last year's Reports, the suggestion made by Dr. Ringer, to use the ordinary morphia hypodermic syringe to clinch the diagnosis where the general symptoms and physical signs have been somewhat dubious, is most useful, and a great improvement on the grooved needle, as originally suggested by the late Dr. Thomas Davies.

Lichtheim² advises the operation of paracentesis as soon as the presence of pus in the pleural cavity is diagnosed, giving his reason in the following words: 'The fever is only the result of pus in a closed cavity. The best means of allaying it is to let out the pus.' Küssmaul and Lebert, however, delay operating until after the disappearance of the acute inflammatory symptoms. The course I think the best is as follows: Whatever treatment has previously been pursued in a case of pleural effusion, as soon as hectic or other symptoms of suppuration supervene, the aspirator should be used, and as much pus drawn off as possible without causing great pain to the patient, or until it has become of a sanguinolent colour. Dr. Bowditch's³ rule is to stop suction as soon as the patient begins to suffer from any uncomfortable symptoms, such as tightness across the chest, severe harassing cough. A mild cough is a favourable sign, as it indicates expansion of the lungs. When as much fluid as was deemed necessary had been removed, the orifice should be closed with a pledget of lint steeped in flexible collodion. If there were again a collection of pus to a small extent, and hectic, but only to a moderate amount, a second attempt might be made with the aspirator, and the opening again closed as before; but, as a rule, if the pus reaccumulate after aspiration, no treatment is of avail except making two openings and passing a drainage tube through, so as to evacuate the whole of the contents of the pleural cavity. If paracentesis is not done in empyema, the natural course of the disease is as follows: In a certain proportion of cases the patient dies from hectic and exhaustion; in others it points and bursts externally; in a few, Traube says about one in five, the pus will make its way into a bronchial tube, and complete recovery may take place, though death may result in these cases from suffocation or from exhaustion.

¹ Dr. Handfield Jones, British Medical Journal, May 11, 1871.

² See Practitioner, Dec. 1874. ³ London Medical Record, Aug. 15, 1876.

In one instance with which I am acquainted thoracentesis had been performed, but the fluid reaccumulated and discharged through a bronchial tube, and the patient made a perfect recovery. The only other natural mode of termination in cases of empyema is for the more liquid parts of the pus to be absorbed, and the remainder to become encapsulated and undergo cheesy or calcareous change; but even here a danger is to be apprehended, as in a post-mortem I made last year on a patient who died with acute tuberculosis, the origin of the infective material appeared to be an old pleural effusion in the right side, which had become caseous, as the miliary tubercles were much more abundant about the liver and the lower part of the right lung. This being the natural history of untreated empyema, there can be no hesitation to adopt the surgical treatment which has been so successful in the hands of all those who have employed it. To show how successful paracentesis thoracis has been in suitable cases, I need only quote Dr. Bowditch of Boston, who in the 'Cincinnati Lancet and Observer' for June last states that he has up to this time performed thoracentesis 328 times on 207 patients, and that none of his patients have died immediately or in consequence of the operation.

Supposing thoracentesis has been decided on, the following points have to be considered:—

1. The instrument to be used.
2. Whether it is advisable to employ an anæsthetic.
3. Place of puncture.
4. Whether the opening is to be closed or left open.
5. If left open, by what means should the opening be kept patent?
6. Are astringent or other injections indicated?

1. As regards the instrument to be used, the simpler the better to my mind. I have used several times lately a very convenient arrangement, introduced I believe by Messrs. Arnold & Son, by which a special receiver is dispensed with, the tube of the aspirator being set in a plug of vulcanised indiarubber, and this is then fitted into the neck of any ordinary bottle; I have generally used a transparent wine-bottle. In cases of serous effusion, Dieulafoy's aspirator, or some modification of it, has now almost entirely superseded the old trocar and canula, as it has the great advantage of preventing the ingress of air; but if the fluid be purulent, with flocculent lymph floating in it, the aspirator is apt to get blocked up. When the fluid has been evacuated by the exhausting apparatus, the lung in expanding may strike against the sharp and hard canula. To prevent this, M. Béhier of Pau uses a canula of soft metal introduced into the ordinary tube. When the pleura is emptied the soft canula bends down against

the parietes of the chest, and the lung does not suffer. One of the most energetic opponents of the aspirator in the treatment of pleural effusion was the late Dr. Fuller, so I have thought it well to quote some remarks of his *in extenso*, and afterwards criticise them. He says in regard to instruments designed to prevent the entrance of air during thoracentesis: 'My objections to their employment are both practical and theoretical. I object to their employment—1st, because they are unnecessary, and complicate a very simple and harmless operation; 2d, because the admission of air during the process of tapping causes no injury to the patient; 3d, because it is impossible by any contrivance to prevent the admission of a certain quantity of air during the withdrawal of the canula, and therefore, even on theoretical grounds, there can be no valid reason against the admission of a larger quantity; 4th, because they mostly occasion unnecessary pain, and when suction-syringes are employed a forcible strain is put on the parts, which is not only felt and complained of by the patient, but in some instances sets up fresh and serious local inflammation; 5th, because, although a certain quantity of fluid may be drawn off, it is physically impossible to *empty* the chest by their agency; and observation at the bedside has convinced me that recovery takes place less frequently when a small quantity only of the fluid is drawn off than when the chest is thoroughly emptied.'¹

These remarks of Dr. Fuller must be received with a considerable amount of qualification. The first and second division may be conveniently discussed together, as they are practically the same though expressed in different language. If Dr. Fuller is right in his statement, then all the precautions taken by the adherents to Lister's plan of treatment are uncalled for, and it is quite unnecessary to prevent the free access of air to any open wound, for how does the pleural cavity communicating with the external atmosphere differ from an open wound? In private practice in the country, where the patient is the sole occupant of a room, I grant that little damage may result from the admission of air into the pleural cavity; but even here I should be anxious to prevent it. When, however, the patient to be treated is in an hospital ward, however good the ventilation may be, I think the case assumes a very different aspect, and I believe it is one's bounden duty to take the greatest possible precaution to prevent air getting into the pleural cavity.

And now as to the third point. It seems to me quite feasible to prevent the ingress of air by having a pad of lint steeped in flexible collodion pressed upon the point of puncture as the canula is withdrawn.

¹ St. George's Hospital Reports, vol. v.

The fourth and fifth points I will take together. Of course if the chest is attempted to be entirely emptied without the admission of air simply by suction, the patient would doubtless suffer great discomfort and even pain; but this is a proceeding which I deprecate, as I believe it is sufficient to draw off a certain proportion of the fluid and leave the rest to be absorbed by the natural processes, because the absorbents, when once they have been relieved from the great pressure to which they have been subjected, will commence to work with great activity. As a parallel instance I may cite the rapid way in which some ascitic accumulations are cleared up after the free employment of hydragogue cathartics, which by unloading the portal capillaries allow the absorbents free play. Another advantage of preventing the ingress of air and using an aspirator, is that a certain amount of traction is exerted upon the collapsed lung, so that if the adhesions are only recent and slight, the lung will expand and occupy the space left by the withdrawal of the fluid; of course, however, I would not advise the employment of sufficient force to cause the patient pain, and the aspiration should be stopped whenever the fluid becomes tinged with blood. Moreover, the direct suction-action of the air-pump should not be used, as it is much safer to exhaust a receiver and connect this with the trocar.

2. *As to the employment of an anæsthetic.*—If the patient were an adult I should not think of using any means to produce unconsciousness, though if the patient dread the momentary pain of the puncture, ether spray might be used; but in children it is a very different matter, as it is often impossible to keep them quiet, and I am certain more risk would be run by subjecting them to the terror of the operation without an anæsthetic than if they were anæsthetised. I have never seen any ill effect from the employment of chloroform in these cases, and I intend to continue the practice I have hitherto followed of using it. When it comes to a matter of making a counter-opening, the pain experienced by the patient and the long time which this operation sometimes requires render chloroform or ether necessary even in adults.

I have recently had under my care as an in-patient in the Westminster Hospital a child with empyema on the right side, and signs of effusion, though to a less extent, on the left side. I determined to have a drainage-tube passed through the right side, and in spite of the dyspnoea the patient was suffering from, chloroform was administered and taken very well. The operation to some little time, as the counter-opening was difficult to make, so that the pain and alarm saved to the child by the use of the anæsthetic were considerable.

3. *Place of puncture.*

Mr. John Wood¹ gives the following directions for thoracentesis; 'Tap in fifth or sixth space under the arm just above the rib, in order to avoid any projection from the rib and the intercostal artery. If you go to the lowest limit of dulness, you may perforate liver or diaphragm. First make an incision, draw the skin down, then introduce your instrument.'

Trousseau² advises 'the sixth or seventh intercostal space, nearly 4 or 5 centimetres external to the outer edge of the pectoralis major.'

It is better, however, to be guided to a great extent in the choice of the site for the operation by the physical signs as revealed by auscultation and percussion, and I do not see the slightest occasion for over-anxiety to tap at the lowest level of the fluid. Dr. Handfield Jones³ says, 'I certainly prefer to find no breath-sounds in the spot where I am about to plunge my trocar;' but he goes on to say that weak and distant breathing need not deter the operator, as lung-sounds can penetrate through a notable thickness of fluid.

4. *Whether the opening should be closed or left open.*—The rules which I have laid down for myself as regards this question are as follow:—

(a.) Whenever the fluid is serous or sero-sanguineous, and so long as it continues such, I close up the opening, so as to prevent the entrance of air; and the best way to effect this is with carbolic plaster.

(b.) If laudable pus be evacuated, I close the opening on the first occasion, in the hope that the little left behind may become absorbed; and even a second time I would try the effect of sealing up the orifice, provided the pus continued laudable, and only a small quantity had reaccumulated since the preceding operation. Should these two tappings not be followed by a cure, then there remains nothing but keeping the openings patent, and this must be done in any case whenever the pus is fœtid.⁴

5. *If left open, by what means should the opening be kept patent?*

This is the question of all others in which I am most interested, as I feel certain that if the practice of making a counter-opening, passing a drainage-tube through, and tying the ends together externally, was more generally adopted, much greater success would attend the treatment of empyemata. 'Where only one opening is present, the pleural cavity may be likened to a barrel without a

¹ Lancet, May 9, 1874.

² *Op. cit.*, p. 271.

³ *Op. cit.*

⁴ The whole of this division of the subject appeared in my paper in last year's Reports; but I have ventured to introduce it again, to make this account of the treatment of pleural effusion as complete as I could.

counter-vent, and the escape of the fluid must be irregular, and only partial.'¹ The plan of 'drainage,' as introduced by Chassaig-nac for the healing of sinuses, consists in passing an india-rubber tube of the diameter of the sixth of an inch, perforated at intervals, through the cavity, so that the pus is able to escape as soon as formed through the perforations.

Mr. Campbell de Morgan, in an addendum to the article from which I have just quoted, gives a very clear description of the manner of introducing the drainage-tube. A firm, long iron probe, somewhat bent (for children I find a steel sound very useful), is passed through the first opening, and directed towards the back of the cavity at the most depending part. As soon as the point of the probe can be felt, an incision must be made down to the probe, which is then to be brought through the opening thus made. The drainage-tube is then attached to the eye of the probe, and drawn through the two openings, and the ends of the tube are to be tied together, which completes the operation. As regards the after-treatment, all that requires to be done is to envelop the affected side of the chest in picked oakum, which prevents any putrefactive change taking place in the pus after it has flowed from the cavity. Should the oakum irritate the orifices, a small piece of lint steeped in carbolic oil may be interposed at these places. It is astonishing how soon this simple mode of treatment suffices to effect a cure. The last patient I have had under my charge in whom I employed it, had absolutely no discharge on the second day after the operation, though upwards of a pint of pus had been evacuated at the thoracentesis; a few days later there was a little oozing, but this soon ceased, and the tube was withdrawn eighteen days after the operation. Dr. Peitavy² records two cases of empyema treated by resection of a portion of the rib, for the purpose of permanently widening the aperture, and so facilitating both the discharge of pus and the injection of fluids. I look upon this as an unnecessarily severe operation, and not required if the drainage-tube be resorted to.

In the able article by Dr. Goodfellow, from which I have already quoted, the history of a case of empyema of five years' standing is recorded. The discharge was profuse, and very foetid; a drainage-tube was introduced, and three months after the operation the discharge scarcely amounted to 2 or 3 drachms in the twenty-four hours; and the patient was able to walk in the garden, after being confined to bed for five years. In the other case described in this paper, the counter-opening was not made sufficiently low, so that the matter remained in the chest, became

¹ Dr. Goodfellow, *Med.-Chir. Trans.*, vol. xlii.

² *Berliner Klin. Woch.*, May 8, 1876.

decomposed, and extremely offensive. A second counter-opening was made as low down as the probe could be felt through the thoracic wall, and the pus soon lost its offensive odour, and rapidly diminished in quantity. This case affords a very good illustration of the importance of selecting the lowest point for the counter-opening.

6. *Are astringent or other injections indicated?*

In answer to this question, I may say that I have been unable to satisfy myself that I have seen much good result from this plan of treatment; and it is one which, if the advice about the drainage-tube be followed, is hardly necessary, as it is only in cases of fistulous empyema with a single opening, and that not at the most depending part of the chest, that there is likely to be any need for the employment of these injections.

If they are used, a dilute solution of liq. potass. permang. (ʒss. ad aquæ oj.) is about the mildest. Carbolic acid (glycerini acid. carbolici ʒv. ad aquæ oj.) is one of the most useful; and a weak solution of the tincture of iodine is said to be very useful in cases in which the pleurisy is of a tubercular or strumous origin.

Besides these three drugs, the whole armentarium of astringent remedies has been exhausted in trying to check the suppuration. The only use I find for injections is to remove fœtor in cases where it is present; but a free counter-opening soon supersedes the necessity for washing out the pleural cavity. Should the drainage-tube, however, be not sufficient to prevent the continual formation of pus, astringent injections ought certainly to be tried; and in the event of these failing, a fair trial might be given to Dr. Dubone's¹ plan of treatment by tannic acid.

His communication records the result of the treatment of eleven severe cases of fistulous empyema by tannic acid, given in a pill mass with the confection of roses, 10 to 25 grains per diem. In eight cases the treatment was most successful, in one there was partial success, and two died. One-half the pills to be given an hour before breakfast, and the other an hour before dinner.

Before finishing with thoracentesis I should like to mention some of the accidents likely to be met with during and after the performance of this operation. In the Hospital Reports² for last year I mentioned the precautions that should be taken to prevent a piece of tube being left behind on withdrawing it from the pleural cavity, and I then stated I had seen a patient in whose chest there were about 12 inches of drainage-tubing owing to the ends having been insecurely fastened together, but that no ill effect had resulted though the tube had not been recovered; and I

¹ Gazette Hebdomadaire de Médecine, le 27 Déc. 1872.

² P. 239.

have since seen two cases recorded—in one about 6 or 8 inches of drainage-tube slipped in, and in the other a gum elastic catheter, but in neither case were any signs of the presence of a foreign body in the thorax exhibited. A patient under the care of Dr. Wilks¹ had paracentesis performed, and the cavity was kept open by a plug of lint soaked in carbolated oil, and during a violent inspiratory effort the lint was drawn into the chest; but Mr. Durham succeeded in extracting it, using a pair of long slender forceps which he had designed for the removal of foreign bodies from the œsophagus. This case affords another illustration of the course of empyemata when treated by having a single opening, as in spite of the cavity being continually washed out, the discharge continued, and the patient died after about three years from amyloid disease of the viscera. As regards another accident of thoracentesis which has lately, and especially in France, received a good deal of attention—I mean the albuminous expectoration—I have not yet seen a case, nor so far as I know has one been met with in London; I am therefore unable to offer any remarks, as I am unwilling to theorise on a subject with which I have no practical acquaintance; those, however, who wish to read a full discussion on this interesting question cannot do better than study the remarks by MM. Terrillon Prodhomme, Béhier, and Féréol in the 'Union Médicale,' 1873. The phenomena attending this curious affection may be very interesting pathologically, but I trust that in my practice I may remain in my present blissful ignorance, as in some of the recorded cases death has taken place with great rapidity. Dr. Ernest Legendre² narrates an instance in which he did paracentesis with Dieulafoy's aspirator, having first told the friends of the patient that marked relief and certain cure would follow, as his previous twenty cases of paracentesis had been so uniformly successful and without the slightest accident. Unfortunately, however, there was in this instance a copious outpouring of secretion into the air-passages and cyanosis, death occurring from asphyxia within the space of five minutes. As Dr. Legendre says in alluding to this case, it will be always prudent to make reservations, and not to promise a certain cure.

IV. *Cases.*

In conclusion I will give some brief notes of cases I have had under my observation, and which bear on points I have discussed in this paper.

No. 1.

J. S., aged 21, labourer, admitted into John Ward, St. Bartho-

¹ *Lancet*, Nov. 22, 1873, p. 739.

² *Gazette des Hospitaux*, 1873.

lomey's Hospital, on February 5, 1873, and discharged unrelieved March 5th.

There was the history of an acute attack of pleurisy a year before admission, followed by effusion. For eleven months he has had a fistulous empyema on the right side, Nature having effected an opening between the fifth and sixth ribs; at first there was a very large quantity of discharge, but latterly it had diminished. He has kept his bed for nine months. While he was in the Hospital the discharge varied from a drachm or so to one or two ounces daily, and when he left the Hospital his condition was the same as on admission.

This patient is a typical instance of what Nature can effect by herself, and what she is unable to do, and therefore art must step in to complete the cure.

According to Niemeyer,¹ empyemata usually point in the neighbourhood of the fourth or fifth rib (in the case under consideration the fistulous opening was situated between the fifth and sixth ribs), and it is manifest from this that there is no chance of the pleural cavity ever being emptied of the pus unless some surgical assistance is given. Putting aside the empyema, the patient was a healthy man living in the country, with abundance of fresh air; and judging from the good condition in which he was, notwithstanding the drain of the discharge, he must have been well supplied with good food: but in spite of these advantages the cavity showed no signs of contracting, and from his own account he was in much the same state as a month after the chest-wall gave way. If an opening had been made in the most depending portion of the chest-wall, and a drainage-tube introduced through the original opening, and then out through the artificial one, and the two ends tied externally, I have no doubt, judging from the success which has attended this procedure in similar cases, but that a perfect cure might have been effected.

No. 2.

In my next case a partial cure was effected without recourse to the drainage-tube, but it took four months to accomplish.

G. W., aged 33, a carman, admitted into John Ward, August 8, 1872, discharged relieved October 4th.

Four months before admission he had an attack of pleurisy; two months later a swelling appeared about 1 inch below the left nipple and to its inner side, and in the course of a month it broke and began to discharge. Shortly afterwards two openings were made about 3 inches below the nipple, as there was evidence

¹ *Op. cit.*, p. 262.

of pointing in two places. Since then there has been a large amount of discharge, as much as a pint a day. After his admission the discharge gradually diminished, and by September 12th the openings were quite healed.

In this case the empyema pointed lower than it usually does, but even here I doubt whether the suppuration would have ceased had not surgical interference been resorted to. From the large quantity of purulent expectoration brought up by this patient, there was a suspicion that the pleural cavity was in part being emptied through the bronchi, and if that were the case, it would account for the removal of the fluid without an opening being made behind.

No. 3.

J. H., a boy, aged $4\frac{1}{2}$, had thoracentesis performed on May 2, 1873, and 20 ounces of very foetid pus were evacuated, and a drainage-tube was introduced through the single aperture. From this time till the boy left the Hospital on August 2d the disease pursued an up-and-down course. For a few days there would be little or no discharge, then it would be profuse, varying from 2 or 3 to 5 or 6 ounces per diem, sometimes foetid, sometimes sweet; but in spite of frequently washing out the pleural cavity with various astringent injections, there was still some discharge when the boy was taken away from the Hospital.

No. 4

affords a good instance of the benefit that would have resulted from making a free incision into the chest and passing a drainage-tube through.

Daniel M., aged 8, admitted into the Westminster Hospital on August 31, 1875.

My note on September 1st is as follows: Child lying on left side. Complexion pasty. Slight œdema of lower eyelids, none elsewhere. Body much emaciated. Pulse, 160. Apex beat of heart half an inch below and to inner side of right nipple. Respirations, 40. Has a troublesome cough. Right chest resonant back and front; abundant mucous râles with sibilus. Left chest dull all over, the dullness extending to right of sternum. No vocal fremitus or resonance. No air entering except along the spine. Urine, sp. gr., 1011; acid; a cloud of albumen.

September 2.—Thoracentesis performed with the aspirator, but only 2 ounces of pus were drawn off when the tube got blocked. Breathing has been easier since. General appearance improved. From this date he gradually got worse, he suffered from diarrhœa

and constant sickness, and died on September 11th. The temperature chart exhibited a very characteristic tracing, and showed a high evening rise, one day being over 4° F. higher than the morning.

The post-mortem revealed the following state of affairs: The left pleural cavity contained 27 ounces of laudable pus. Left lung compressed along the spine, readily inflates. Right lung, anterior margin collapsed, the rest of the lung healthy, except that it is engorged with blood. Deposit of thick rough lymph on the pleura covering pericardium. No part of the heart visible to the left of the sternum, the greater part to the right; pericardium adherent; adhesions readily giving way. The whole of both surfaces of pericardium covered with shaggy lymph, except posterior surface of left ventricle and corresponding part of parietal pericardium. A small quantity of turbid serum in pericardial cavity. No signs of endocarditis.

It is unfortunate that no second attempt at thoracentesis was made in this case, as the lung would in all probability have readily expanded, and good recovery might have been made. In my opinion it is almost as bad practice for a physician to allow a patient to die with his chest full of pus, as for an obstetrician to permit a woman to die undelivered.

No. 5.

On June 29, 1875, I was called in consultation to see Master C. M., aged 8. I found that the patient had been taken suddenly ill on May 3d with pain in the left chest and feverishness. On examination there were evidences of a large pleural effusion into the left pleural cavity, the heart being much pushed over to the right side. I accordingly determined to do paracentesis thoracis. The next day I drew off with the aspirator 12 ounces of laudable pus when the tube became blocked. For the first week after the operation he improved, but by July 7th hectic became marked again, and on the 10th the empyema was observed to be pointing. On the 11th I made an incision into the abscess (the patient being under the influence of chloroform), and about 2 pints of laudable pus escaped; then I passed a drainage-tube through and brought it out posteriorly, and tied the two ends together, and the chest was covered up with picked oakum.

July 12.—Very sick all night up till 4 A.M. Free discharge. Appetite bad. To have quinine and acid. sulph. dil.

July 16.—Appetite very good. Sleeps well. Very little discharge from the wounds, which are dressed with lint and carbolic oil, over which is oakum. To get up for two hours daily. Ordered ol. morrhuæ.

July 23.—Resonance all over left chest, and fair vesicular breathing. Side fallen in considerably. Not much discharge. Appetite good. Gaining flesh.

August 2.—Still some discharge. General condition admirable. Eating well, and running about.

August 31.—Tube kept in one opening only, the other allowed to close.

September 23.—Hardly any discharge. Tube removed entirely. The opening closed about a week later.

On May 4, 1876, I had an opportunity of examining this patient, and I found perfect resonance all over the chest, with good vesicular breathing. Hardly a trace of retraction. Has enjoyed excellent health since his late illness.

Nothing could have been more successful than the treatment in this case. When I was hastily summoned to do the second thoracentesis the child was almost moribund, and a medical man who had seen the child had taken a most gloomy view of his condition. Immediately, however, that the patient was relieved of the irritation of the pent-up pus he began to improve, and his progress towards recovery was uninterrupted. On examining his chest a year after the attack, I was unable to find any evidences of the grave malady with which he had been afflicted.

These are only a few out of the many cases which I might have brought forward to show the necessity and prove the success of the system of drainage in the treatment of empyemata, and it was for the purpose of calling the attention of the medical profession to this plan of treatment that I was first induced to begin this paper, which from various reasons has expanded to a much greater extent than I intended.

ON THE PATHOLOGY OF ONE FORM OF DENTIGEROUS CYST.

BY

ALFRED COLEMAN.

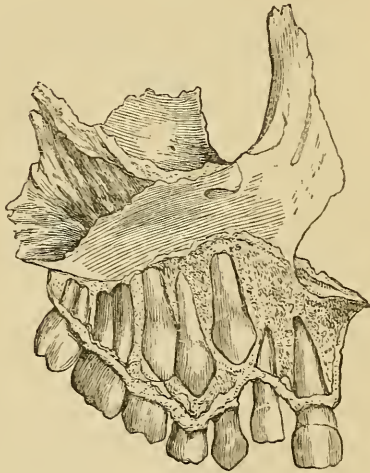
Under the head of dentigerous cyst has been frequently classed two or three quite distinct pathological phenomena. My present object is to offer an explanation for the origin of one only—viz., that consisting of a cyst filled with serous fluid, and containing within its cavity a tooth or a portion of a tooth which has not been normally erupted.

To fully comprehend the explanation I would offer, it is necessary first to consider the probable cause for the eruption of the teeth under ordinary conditions. To account for the eruption of the teeth two views have until recently been advanced—one in which the process is attributed to the growth of the fangs of the teeth causing their crowns to be raised out of their alveoli; and the other to a growth of bone at the lower portions of the alveoli, squeezing as it were the teeth out of their sockets. The first is still the view entertained by some. In the most recent work on dental surgery the following appears: 'The eruption of the teeth is a process of gradual elongation of the teeth on the one hand, and of simultaneous absorption of the superimposed tissues on the other. The absorption commences first in the overhanging margins and front walls of the alveoli, which gradually disappear until room is afforded for the free passage of the advancing tooth. The growth of the tooth keeps pace with this absorption, and the crown at length pressing against its membranous coverings, these undergo atrophy, and becoming by degrees thinner, and at last transparent, give way and disclose the advancing crown.'¹

In my first course of lectures at the Hospital in the year 1867, I pointed out and illustrated the assertion by preparations—viz.,

¹ The Student's Guide to Dental Anatomy and Surgery, by Henry Sewell, M.R.C.S., 1876, p. 27.

how inaccurate was the first-assigned cause; for there were the cases of teeth not erupted and with fangs fully developed, which would, had the individual survived, been erupted in due course, as well as others in which the additional length of the fang would not when completed have brought the crown to more than the level of the alveolar process. The woodcut, taken from a preparation in the Museum of the Odontological Society illustrating the normal conditions under which the permanent teeth are erupted, illustrates these points.



For the second and a later assigned cause there is actually no ground, as it cannot be shown that there is greater development of bone taking place during the eruption of the teeth at the apices of the alveoli than at other parts of that district of the maxillæ. The only probable explanation which it appeared to me could be offered was in the growth and advance of the whole bone towards the surface, carrying with itself the contained teeth. This assertion consequently implied a continual growth of bone from its nutrient centre towards its circumference, and that at the latter absorption must be frequently taking place, as we have the evidence it occasionally does during the process of the absorption of the temporary eruption of the permanent teeth, when portions of bone—platforms they might be called—supporting the former are often seen extending much above the general level, as seen in the woodcut, which become absorbed again directly the sustained teeth fall out. There is one condition inseparable from this explanation—

viz, that as the process of growth is continuous, it must not merely effect the eruption of the teeth, but eventually in time actually cause their exfoliation; and such indeed would be the case did they not meet with an opposing force such as is chiefly supplied in the contact of the teeth of the opposite jaw, and in a lesser degree by the resistance offered during the natural process of mastication. That such is the case is rendered evident by the fact that teeth which have lost their antagonists, and roots which have no antagonists, are eventually exfoliated, and also that animals suffer in like manner when for a naturally hard food is substituted a soft one, as seen in the case of indoor dogs thus treated. This latter condition, no doubt, to a great extent explains that which is becoming so common in the present day—viz., a premature loss of the teeth by exfoliation.

According, then, to the views I have advanced, a tooth developed in the maxilla is carried with the growth of that bone towards the surface. Arriving at the surface, its bony crypt becomes absorbed, and the more so as it advances until the crown projecting above the surface meets with some opposition to its farther advancement and becomes held back, when the bone immediately surrounding it—its alveolus—becomes more dense in character and less rapid in growth than the surrounding cancellous interalveolar portions. These harder portions of bone being more stationary, we have doubtless, so to speak, bone currents continually existing in the maxillæ, but more especially during the periods of the first and second dentitions, when those bones are in a condition of more than ordinary active development. In connection with this subject may be mentioned a curious phenomenon often witnessed at the period of the second dentition—viz., where a tooth erupted fails for a time to meet an opposing force, and consequently projects unduly beyond its fellows, but which, when that force is supplied, is made to recede to its normal limits. The same condition is more frequently met with and rendered more apparent in the treatment for irregularities in position of the permanent teeth, where a plate worn relieves a tooth from opposing force which protrudes considerably from its socket, but which soon is pressed back to its normal position when the plate is removed, and it becomes for a time subject to the whole force employed in mastication. On the other hand, teeth subjected to abnormal pressure due to the loss of their comrades are often seen unduly pressed into their alveoli.

Views nearly resembling those I have stated to account for the eruption of the teeth have been advanced and the subject exhaustively treated by Herr Robert Baume in the '*Vierteljahrsschrift*;'¹

¹ Translated in the *Monthly Review of Dental Surgery*, vol. i.

and to that writer I must fully accord priority of publication, if not of originality of idea; and they certainly to my mind offer a satisfactory explanation for a process which a most able writer in dental anatomy and physiology still regards as obscure and hitherto unexplained.

If we now direct our attention to the subject immediately before us—viz., the probable cause of that form of dentigerous cyst spoken of—we must, in the first place, remember that it invariably occurs in connection with teeth the eruption of which is retarded or prevented from their being developed in an abnormal position, whereby they become impacted between or about the still layers of dense bone which surround the fangs of the adjoining teeth and form their alveoli. We must also bear in mind that these tumours most commonly manifest themselves at a period succeeding that when the alveolar portions of the maxillæ have been in a state of active development, in which they can readily furnish such an amount of bone as will perfectly envelop them. By and by, as this state of activity lessens, such will not be the case, and there will be a tendency to the existence of an hiatus at that portion of the tooth most distant from the nutrient or medullary portion of the bone. The capsule of the tooth, the remains of the so-called enamel organ, has been shown by Tomes to be, after the calcification of the enamel, quite free and detached from that structure, and therefore, being attached only to its surroundings, will be carried away from the surface of the enamel with them; a space will thus be left, into which as a matter of course serous fluid must under atmospheric pressure be effused, and thus a cyst be formed, the walls of which will be the dental capsule, including the projecting crown of the tooth. The further progress of the disease will be the approach of the cyst towards the surface, where it will, if not interfered with, eventually evacuate itself. Before this takes place it not uncommonly happens that it has so exposed the fang portions that the tooth has lost its hold and falls into the cyst. In alluding to this form of dentigerous cyst, Mr. Tomes refers to the fact before mentioned—viz., that when the development of the enamel is completed, its outer surface becomes perfectly detached from the investing soft tissue, and that a small quantity of fluid occasionally collects in the interval so formed; but when from some cause the eruption of the tooth is prevented, it may increase in quantity, and gradually distend the surrounding tissues in the form of a cyst. Whilst this view has been generally accepted by pathologists, I trust that what I have offered may afford a fuller explanation for the cause of the dilation of the cyst, and its advance to the surface.

A CASE
IN WHICH
ABDOMINAL SECTION WAS PERFORMED
FOR INTUSSUSCEPTION.

BY
HOWARD MARSH.

On the 11th of last August I was asked by Dr. Galabin to see John M., 9 months old, brought an hour before to his out-patient room at the Children's Hospital with intussusception. For the history of the case and many of the following notes I am indebted to the house surgeon, Mr. Cant.

For a month the child had been subject to 'diarrhœa.' On August 5th, six days before admission, he passed two or three drops of blood from the bowel. On the 6th the intestine was found for the first time projecting from the anus. A surgeon returned it, but it soon came down again, and the protrusion gradually increased till the patient was brought to the Hospital. The bowels acted twice on the 6th, and once or twice a day subsequently, and flatus was often passed, so that nothing like obstruction had been present. Sickness had occurred only twice. Once the vomit was of curdled milk, once it was 'yellow and sour smelling,' probably consisting of bile-stained fluid from the duodenum. On admission the child was pale, wasted, and much exhausted, with sunken half-closed eyes, and a small and quick pulse. The volvulus projected between five and six inches beyond the anus, and the finger could be passed all round it high up in the rectum. The portion that was external was not dusky or livid, but of a dark florid tint, and felt soft and flaccid rather than tense, or firm and brawny. At its lower end could be seen the central canal of the intestine, and a little to the side was the slit-like opening of the ileo-cæcal valve. Through the abdominal wall the intussusception

was readily felt in the rectum and sigmoid flexure, but at about the level of the crest of the ilium it was gradually lost, and above this only some slightly increased resistance in the line of the colon could be traced. The abdomen was large but soft, and there appeared to be no tenderness on pressure.

It seemed doubtful whether this was a fit case for abdominal section, for the child after a month's illness was weak and feeble, and the intussusception was of a portentous length. However, it was determined to perform the operation on the following grounds: (1.) The patient's condition was hopeless if things were left as they were. (2.) There seemed no hope that an intussusception by which the cæcum had been protruded more than five inches from the anus could be reduced by any means short of operation. (3.) That the child had been sick only twice; that the bowels had acted daily, and that flatus was often passed by the anus; that only two or three drops of blood had been seen on a single occasion six days before; that there had been little discharge, either mucous or serous, from the intestine; that the volvulus was neither tense nor dusky,—all these points, when looked at by the light of former experience, seemed almost conclusive evidence that neither adhesion nor strangulation was present. The hope that the child might be saved by sloughing and discharge of the volvulus could not be entertained, first, because there was no prospect that sloughing would take place, for there was no strangulation to produce it; and secondly, because, even if it did, the loss of so much intestine would certainly be fatal.¹

Operation.—A catheter having been passed to empty the bladder, and the child being under chloroform, an incision was made in the middle line downwards from the umbilicus for about two inches, and the abdominal cavity opened. Two fingers were then passed down to the left iliac fossa, where the intussusception had been already felt; but it was found impossible, in tracing the swelling upward, to make out which was the upper end of the volvulus, or at what point traction should be made. The difficulty was caused by the loose and flaccid state of the volvulus and its sheath. I could feel no tense border to the sheath where it turned inwards upon itself, nor any entering portion of gut. It was therefore necessary to bring the parts into sight at the wound. In consequence, however, of the great length of intestine that was involved, the mesentery and mesocolon of which had gone with it into the sheath, the intussusception was held down at the back of the abdomen, and could not without some slight force be drawn forwards. But when it had been brought into the wound, and the

¹ To make this account distinct for use in other cases it should be stated that no attempt was made by insufflation or otherwise to effect reduction.

entering intestine was discovered, I found that by no amount or method of traction that seemed justifiable could any portion of the volvulus be drawn out. I therefore tried the expedient successfully used by Mr. Hutchinson in his second case, of acting on its lower end by pushing it backwards towards the mouth of the sheath. The part that had projected beyond the anus having been replaced (this had been done before any attempt at reduction was made), the sheath was taken between the finger and thumb immediately below the volvulus and gently squeezed; and this squeezing was constantly repeated, while the finger and thumb were gradually shifted upwards in the long axis of the gut. By this manœuvre the volvulus was with the greatest ease made to 'back out,' being lifted in a retrograde direction, and pushed higher and higher till the cæcum and its appendix were seen to emerge from the sheath. The intestines that were now lying on the surface of the abdomen were returned, though, as in previous cases, not without some difficulty and delay, and the wound was closed with hare-lip pins and superficial sutures.¹

When the operation was finished, the child was very pale and almost pulseless, but being placed in a nurse's arms near a fire, and cautiously fed with small quantities of brandy in milk-and-water, he gradually revived. The bowels acted four times in the course of the next nine hours, and flatus was freely passed. No sickness occurred. Towards evening the patient rather suddenly became restless and collapsed, and died at 9 P.M., ten hours after the operation.

A post-mortem examination was made next day. The abdomen was distended and tympanitic. On opening its cavity, about two teaspoonfuls of brown turbid serum were found in the bottom of the pelvis; but no recent lymph was to be seen. Both in the mesentery and mesocolon were several small extravasations of blood, and in two or three places small lacerations were observed. It has already been mentioned that some slight force was used in bringing the intussusception forward into the wound, and no doubt here were the evidences of mischief that was then done. The intestines were moderately distended. Throughout their entire length they were healthy in appearance—neither over-vascular nor thickened, nor presenting any sign of recent constriction—except in the last four inches of the ileum and first three of the colon. These portions were congested and œdematous, and when laid open, their mucous membrane was found to be reddened and turgid, and in the colon

¹ In these cases, as in large hernial protrusions, the intestine requires a particular method for its reduction. The operator must search for one end of the protruding loop, and commence reduction by acting on this part, returning first that portion which has last escaped, and proceeding on the same plan till all has been replaced.

was a piece about two inches long that was of a dark green tint from colour-changes in blood effused into the submucous tissue; but there was nothing in the least degree indicating gangrene, nor any ulceration of the mucous surface. This discoloured portion seemed to have formed the free end of the volvulus.

Thus the intussusception had left scarcely any trace behind it—so little that I think no one who was ignorant of the history of the case would have suspected, from the appearances found after death, that it had been present.

Remarks.—The case was a typical one of that chronic form of intussusception to which Mr. Hutchinson has called particular attention. The ileum had slipped into the colon as into a loose sheath, by which it was so slightly constricted that its circulation was but little interrupted, and its canal so little compressed that both flatus and fæces could still pass through it. Probably the absence of constriction was to some extent due to the weakly state of the child, the muscular coat of whose intestine was feeble and relaxed, and ready to yield to any distending force. And this circumstance may also explain the great extent to which the intussusception had advanced. The date at which invagination began cannot be fixed, but whenever it occurred, both its onset and subsequent progress were very insidious; for besides the presence of the tumour in the course of the colon, and its ultimate protrusion from the anus, not one of the standard symptoms of invagination was present in anything like a characteristic form. It need not, therefore, be a matter of surprise that the real nature of the illness had not been made out before the child was brought to Dr. Galabin. Yet the case may serve the useful purpose of recalling the fact that intussusception, even though it involves a considerable length of intestine, does not necessarily produce any symptom beyond those that commonly attend slight catarrhal enteritis. The development of symptoms depends upon the occurrence of constriction. In this respect an intussusception is like a hernia, which may be 'down' without being strangulated or even obstructed. And to say that vomiting, constipation, and the discharge of blood and mucus or serum from the bowel are the symptoms of invagination is inexact, in the same sense in which it would be to say that vomiting and constipation are symptoms of hernia. In both cases alike these various symptoms depend not on the mere displacement of the intestine, but on the constriction which the displacement has produced. In other words, just as a hernia, so long as it is unconstricted, will declare itself by no symptom beyond that of its mere bodily presence, in the shape of a swelling that may be felt, so it is with an intussusception. A number of cases might be collected in which the

affection was not so much as suspected till the volvulus protruded from the anus. Yet no doubt in these instances the swelling formed by the invagination might have been readily discovered. It is obviously necessary, therefore, to take the easy precaution of examining the abdomen, to ascertain whether any tumour can be felt, in all cases in which 'masked intussusception' may perhaps exist. These cases cannot, it must be owned, be very clearly defined, for (to speak of them only in children) they include all those in which bowel derangement is present. Nevertheless the trouble of making an examination is not beyond our duty. Is it not as bad to overlook an intussusception as a hernia or a stone? And it must be remembered that the success of our treatment depends very largely on an early diagnosis. Probably a majority of intussusceptions might be reduced, if they were recognised at their commencement.

One of the chief points of interest in the case relates to the manner in which the invagination was returned. This was the second time that an intussusception which could not be drawn out was reduced with the greatest ease, by pushing it back in the fashion I have described above. Are not these two cases sufficient to show that this is the true method of reduction, especially as there are other grounds on which it may claim the preference? Thus, in the instance just related, the upper end of the intussusception could not be at first made out; and it was found only by bringing the sausage-shaped swelling forward into the wound, and tracing its length from below upwards: and this was not done without some delay, and some free handling of the intestine; yet the lower end was perfectly obvious, and could be reached and manipulated without disturbing its connections. Secondly, it has been found hitherto that an intussusception cannot be drawn out while it lies *in situ* at the back of the abdomen. It must be brought forward, so that the sheath can be held with one hand, while the volvulus is drawn out with the other. And this is a proceeding that may be both difficult and dangerous. Although every care was used, I found afterwards that in bringing the tumour towards the wound, several lacerations of the mesentery had occurred. Though further experience is required to prove it, I think it most likely that the plan of pushing out the volvulus could generally be effected without bringing the tumour forward.

A last word may be said as to the necessity for a certain degree of judicious boldness in the management of these cases. For want of sufficient experience of its results when performed under favourable circumstances, the value of abdominal section as a means of treating intussusception cannot yet be determined. The operation

no doubt involves a considerable danger to life; but how great this may be is not known, even approximately. Whatever it may be, however, the hope is that it can be diminished; for although the general plan of operating is agreed upon, there are probably many important points still to be observed, in which one case may differ from another, many details of treatment yet to be worked out. I cannot too strongly express my opinion that these cases should be classed, in respect to the urgency for operative interference, with hernia. If the diagnosis is complete, if strangulation is recent—say of not more than eighteen hours' duration—or if, in chronic cases, there is room for good hope that inflammation is not present; if other means of reduction have failed, and if there is no other circumstance in the case that makes failure a foregone conclusion, or leaves little hope of success, the operation should be performed, after the common rule in hernia, at once. Till this becomes the accepted practice in these cases, it will never be known how many lives among them may be saved by abdominal section.

EXAMPLES
OF
MALFORMATION OF THE HEART.

BY
NORMAN MOORE, M.D.

Last year I described in the Hospital Reports two examples of malformation of the heart. This year I have had the opportunity of examining after death two more such cases. The first is remarkable on account of the age to which the patient lived. Robert Nicholls, aged 57 years, was admitted into Luke Ward on April 19, 1876. His face, neck, and ears were slightly deeper in colour than natural. An increase of cardiac dulness, a heaving and somewhat irregular cardiac impulse, and an indistinct systolic murmur, loudest at the base of the heart but very faint everywhere, and not louder to right than to left of the sternum, were the physical signs noticed on examination of his chest. On palpation no thrill was felt. His pulse was regular, and slightly sudden in its impulse. He complained of attacks of severe pain about the base of the heart. He had served his full time in the army. The suffused condition of his head and neck, the faint murmur and the pain in his chest led me to conjecture that he had aneurysm of the aorta. He died on May 22, 1876, and the post-mortem appearances were as follows: General dropsy; hardness of the liver, spleen, and kidneys; the heart greatly hypertrophied and dilated in all its chambers, but the valves normal; the aorta abruptly narrowed, so that it would only just admit the tip of the forefinger at the point of junction of the ductus arteriosus (which was closed). At this constriction there was a slight internal ridge, and beyond the ridge the aorta was of the normal width.

The narrowness was no doubt congenital, and was found at the usual point. It is remarkable that, though ultimately the cause

of the patient's death, the constriction should not have interfered with his duties as a soldier.

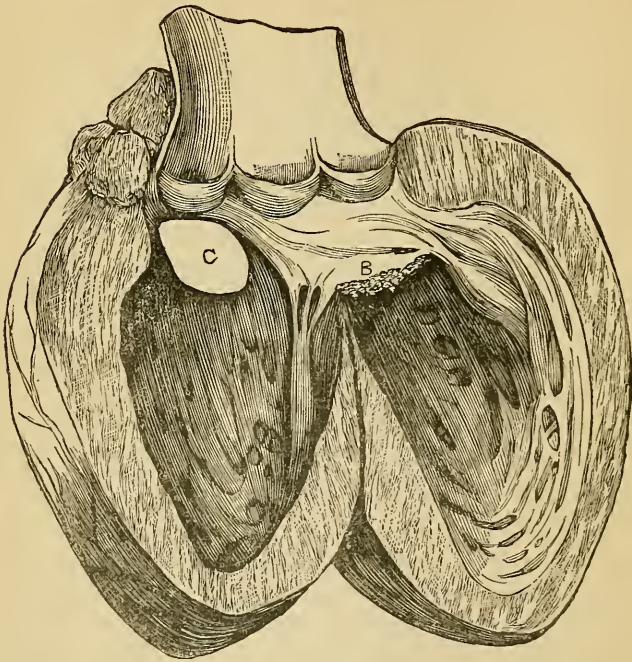


Fig. 1.—HEART OF EMILY REID.

B, Mitral valve with growths. C, Opening in septum of ventricles.

The second case was that of Emily Reid, an artificial-flower maker, who died, at the age of 20 years, in Elizabeth Ward on February 16, 1876. She had been under my observation for about four years. She was of short stature; her fingers, toes, and nose were clubbed. She had been short of breath from birth. Her complexion was purplish, but the colour was not deep, except after exertion or during one of the pulmonary catarrhs to which she was subject. The physical signs, which were constant, were a slightly increased impulse and a very loud systolic murmur, loudest at a point midway between the left base and the apex, and audible, but with diminished loudness, at the angle of the scapula. Besides these signs, on some occasions a præ-systolic murmur was to be heard at the apex, and a slight thrill to be felt over the cardiac area. Her pulse was always feeble and irregular. She had several fainting fits in June 1875, and was

admitted into Elizabeth Ward. After a month she left the Hospital and was able to work at her trade. In January 1876 a slight cold caused her to be unusually short of breath; she became dropsical, and died three weeks after admission to the Hospital. During her life it had often been discussed whether her symptoms and physical signs were due to early and severe disease of the mitral valve or to congenital malformation of the heart. The post-mortem examination answered both questions in the affirmative.

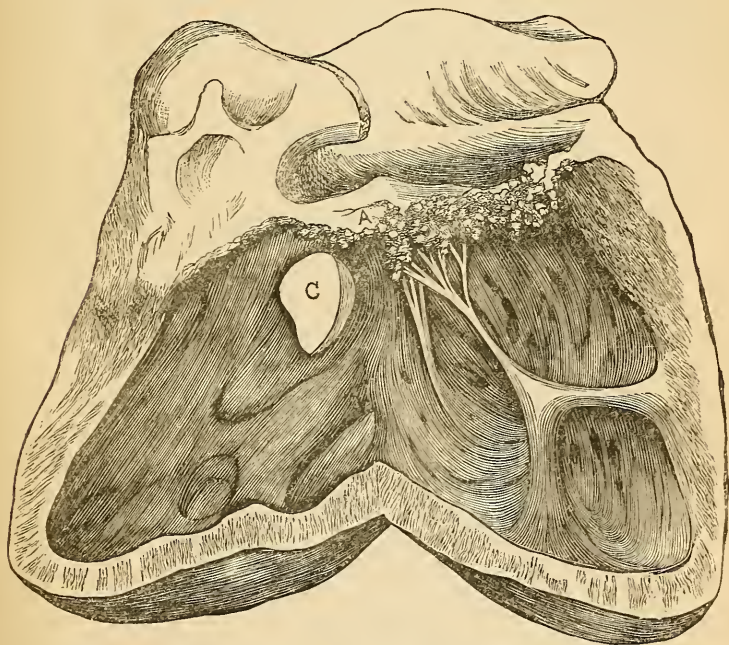


Fig. 2.—HEART OF EMILY REID.

A, Tricuspid valve with growths.

C, Opening in septum.

The heart was hypertrophied. The right ventricle was markedly dilated. The tricuspid valve (*a*, fig. 2) was fringed by closely-set growths. There was a similar series of growths on the mitral valve (*b*, fig. 1). The pulmonary orifice was contracted by partial fusion of its valves. The ductus arteriosus was closed. The aorta and its valves were normal. At the uppermost part of the ventricular septum there was an opening about equal to a sixpence in area (*c*, figs. 1 and 2). This opening had rounded muscular edges, and its upper edge was close to one of the aortic

valves. It of course admitted free communication between the ventricles.

A cardiac murmur is usually the index of some one form of valvular disease. This case shows how essential to accuracy it is to keep the observation and the inference distinct; to think of a murmur as of such a time and place, and not as of such a valve. This heart represents the case of the highest possible complexity, but one of the causes of a systolic murmur. The loud systolic murmur, with its occasional præ systolic commencement, was probably the physical sign of six lesions: tricuspid obstruction, tricuspid regurgitation, pulmonary obstruction, mitral obstruction, mitral regurgitation, imperfect septum of the ventricles.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

BY

NEVILLE HART, M.B.

The following cases of epidemic cerebro-spinal meningitis have occurred in the wards of the Queen's Hospital, Birmingham, during the past nine months. I am enabled to record them through the courtesy of the medical staff.

CASE I.

W. C., æt. 25, labourer, admitted Dec. 27th, 1875 (Dr. Sawyer).

Prominent symptoms.—Rigors. Stiffness and pains in head, back, and throat. Retraction of head. Insomnia. No tenderness of spine. Lies frequently on belly. Phosphatic urine. Temperature, 103° F. eighth day; 101° F. ninth day; remained so until twelfth day, when it became normal. Pulse—average rate, 90; highest rate, 104; lowest rate, 84.

Principal treatment.—Dec. 27—Hydrarg. chlor., gr. x., statim. Chloral hydrat., gr. x.; pot. bromid., gr. xx.; aqu. \bar{z} j., h. s. s. This was repeated in two hours' time, and gave a fair night's rest. Dec. 28—Pot. bromid., gr. xx.; pot. iodi, gr. v.; aqu. \bar{z} j., 4tis horis. Emplast lyttæ, 3 × 12. Dec. 30—Liq. hydrarg. perchlor., \bar{z} j.; aqu. \bar{z} j., 6tis horis. Gums sore on Jan. 6, when pot. iodi, gr. xx.; decoct. cinchon., \bar{z} j., t. d. s. Jan. 9—To have half-doses of last mixture. Milk diet. Brandy, \bar{z} ij. daily.

Duration of disease.—Twenty-one days.

Result.—Cure.

CASE II.

S. L., æt. 7, admitted Jan. 12th, 1876 (Dr. Johnston).

Prominent symptoms.—Constant pain in head. Slight pain

in neck. Herpes on lips. Sickness, constant at first, slight returns of it during whole illness. Great emaciation. Temperature—chart appended. Pulse—usual rate, 80; highest rate, 136; lowest rate, 42.

Principal treatment.—Pot. bromid., gr. vj.; chloral hyd., gr. ij.; syr. zingiberis, m. xx.; aqu. Ziv., 4tis horis for four days. Pulv. Ipecac. co., gr. ij.; hyd. e creta, gr. ij., om. nocte for five days. Blister to neck. Perchloride of iron and ol. morrhue during convalescence. Brandy, ʒj. daily.

Duration.—Seven weeks.

Result.—Cure.

CASE III.

R. W., æt. 35, groom, admitted Jan. 21st (Dr. Sawyer).

Prominent symptoms.—Rigors. Pains in head, back, calves of legs. Great retraction of head. Vomiting. Insomnia. Great emaciation. Deafness, which became absolute for some days preceding death.

Principal treatment.—Pot. bromid.; chloral hyd.; morphia hypodermically, which gave great temporary relief; pot. iodid.; cinchona. Milk diet. Brandy.

Duration.—Fourteen weeks.

Result.—Death.

Post-mortem appearances.—Thoracic and abdominal organs healthy. Skull—meninges of brain dry; convolutions flattened; veins distended with fluid blood; cerebellum and medulla oblongata attached by old adhesions to adjoining bones; large quantity of fluid in sub-arachnoid space; small quantity in ventricles; brain substance apparently healthy. Spinal cord—increased quantity of cerebro-spinal fluid; spinal pia mater congested and thickened; spinal medulla softened, especially at upper part.

CASE IV.

G. M., æt. 33, labourer, admitted Jan. 26th (Dr. Johnston).

Prominent symptoms.—Pains in head, back, and limbs. Marked retraction of head. Insomnia. Delirium, sometimes getting out of bed. Slight deafness. Herpetic spots on lips, especially left side, on left side of neck, and on left conjunctiva, causing great congestion of that tunic. Herpes during second week of illness. Masturbation. Great wasting. Subsultus tendinum. Low typhoid condition last few days of life. Temperature—chart appended. Pulse—average pulse-rate, 96; lowest rate, 84; highest rate, 136, during last stage of illness.

Duration.—Four weeks.

Result.—Death.

Post-mortem appearances.—Thoracic and abdominal organs healthy. Skull—slight roughening and purulent effusion on dura mater, and on upper surface of brain, near right sylvian fissure. At base of brain, in sub-arachnoid space, especially in posterior fossa, was distributed some semi-solid gelatinous matter; large quantity of fluid in ventricles; in posterior cornua of lateral ventricles were patches of lymph; vessels of brain injected; central portions of brain under fornix softened, and broken up into fringe-like processes. Spinal cord—dura mater healthy; increased quantity of cerebro-spinal fluids semi-purulent; nerves coming off healthy; thickened pia mater; cord congested; no lymph, adhesions, or softening.

CASE V.

C. M., æt. 3, admitted Feb. 9th (Dr. Johnston).

Prominent symptoms.—Vomiting. Irritability. Retraction of head. Dilated pupils. Insomnia. Delirium. Bronchitis. Emaciation. Incontinence of urine. Masturbation. Herpetic spots on conjunctiva. Bed-sores. Tardy convalescence, with occasional vomiting. In September 1876, child fat; no incontinence of urine; manner more irritable than before illness. Temperature—highest, 102° F.; lowest, 96° F.

Principal treatment.—Pot. bromid.; hyd. chloral; hyd. e creta; pulv. Ipecac. co.; vin ferri; ol. morrhua. Lin. belladonn. and lin. camph., equal parts, rubbed over spine. T. ferri perchlor. Milk. Brandy. Lime-water.

Duration.—Nine weeks.

Result.—Cure.

CASE VI.

W. M., æt. 5, admitted Feb. 9th (Dr. Johnston).

Prominent symptoms.—Patient a brother of Case V. They were both admitted with severe bronchitis, which gradually disappeared. Pains in back and limbs. Almost constant vomiting of thin greenish fluid, as in all the cases; at times milk returning curdled. Vermes lumbricoides in early part of illness. Retracted head. Dilated pupils; in latter stages sight affected. General rigidity of body, with flexed extremities, worse some days than others. Difficulty of swallowing. Fed through an elastic urethral catheter. Trismus coming on, catheter passed through nose. Paralysis of sphincters. Great emaciation. Slight bed-sores, considering that patient was almost a skeleton.

Subsultus tendinum. Temperature—chart shows very irregular curves; generally between 98° and 100° F.; twice it rose to 102.4°; eight times during illness it was 95° in the evening. Pulse—average rate, 106; lowest rate, 58; highest rate, 160.

Principal treatment.—Pot. iodid.; pot. bromid.; ferri iodid.; hyd. c. creta; pulv. Ipecac. co.; vin ferri; ol. morrhuae. Santonin. Nourishing diet. Brandy.

Duration.—Seven weeks.

Result.—Death.

Post-mortem appearances.—Great emaciation. Thoracic and abdominal viscera healthy. Skull—sub-arachnoid effusion; lateral and fourth ventricles much distended with clear fluid; substance of brain firmer than normal; foramen of Monro admits a finger; middle commissure of third ventricle easily breaking up on being touched; no lymph or granular matter at base of brain. The fluid found in ventricles was slightly acid; it contained abundant chlorides, a trace of albumen and phosphates; no trace of sugar. Spinal cord not opened.

CASE VII.

B. M., æt. 12, admitted April 16th (Dr. Mackey).

Prominent symptoms.—Screaming. Muscular spasms. Retraction of head. General tremors. Vomiting. Flexion of joints. Pustular eruption on body and legs.

Principal treatment.—Pot. bromid.; ammon. carb.; pulv. jalap. co.; hydrarg. chlor.; pot. bicarb.; T. acunit.; ol. morrhuae.

Duration.—Nine weeks.

Result.—Cure.

CASE VIII.

T. G., æt. 22, blacksmith, admitted May 12th (Dr. Mackey).

Prominent symptoms.—Rigors. Stabbing pains in head and small of back. Retraction of head. Vomiting. Pain on pressure over occiput and lumbar region. General hyperæsthesia. Herpes on lips. Smell, taste, and sight affected. Dilated pupils. Optic discs healthy. Delirium. Flexion of extremities. Emaciation. Subsultus tendinum. Temperature—highest, 105° F.; lowest, 97° F. Very irregular thermal curves. Morning temperature sometimes highest. Last three weeks of illness temperature remained nearly normal. Pulse—highest rate, 110; lowest, 80.

Principal treatment.—Pot. iodid.; pot. bromid. Ext. belladon. and glycerine, equal parts, rubbed on neck. T. acunit. in m. j. doses every hour diminished the pain. Blister. Morphia

hypodermically relieved pain. Leeches to temples. Quinine. Brandy, \bar{z} vj.

Duration.—Eight weeks.

Result.—Death.

Autopsy refused.

CASE IX.

E. H., æt. 2, admitted May 10th (Dr. Johnston).

Prominent symptoms.—Pains in head. Vomiting of thin greenish fluid. Retraction of head. Dilated pupils. Cough with signs of bronchial catarrh (had been in Hospital with measles a month before). Constipated bowels. Rapid emaciation. Flexion of extremities. Some days head less retracted than others. Last two days of life dysphagia. Temperature—chart appended. Pulse—average rate, 120.

Principal treatment.—Pot. iodid.; pot. bromid.; hydrarg. chlor.; ferr. bromid.; chloral hyd.; sodæ carb.; ol. morrhuæ; ferri et ammon. cit. Milk. Brandy. Eggs. Aq. calcis.

Duration.—Four weeks.

Result.—Death.

Post-mortem appearances.—Great emaciation of body. Thoracic and abdominal viscera healthy. Membranes of brain red and injected. In sub-arachnoid space at base of brain a small quantity of pink serum. Brain very firm on section.

CASE X.

E. L., æt. 23, admitted May 17th (Dr. Johnston).

Prominent symptoms.—Rigors. Aching pains in head, back, and legs. Second day of illness noticed stiffness of neck, followed by retraction of head. Herpes on lips seventh day. Great sweating. Constipated bowels. Phosphatic urine. Deafness. Slight blindness. Pupils dilated. Insomnia. Delirium. Incontinence of urine and fœces. Jerking movements of eyes. During fourth and fifth weeks the delirium and retraction of head lessened. Sight and hearing returned. Power regained over sphincters. Severe symptoms returned last few days of illness. Temperature—irregular chart. Occasionally rises of temperature in the morning. Highest temperature, 103.8° F.; lowest temperature, 98° F. Pulse—average rate, 110; highest rate, 158; lowest rate, 100.

Principal treatment.—Morphia hypodermically, $\frac{1}{2}$ -grain doses once or twice a day; always better after them. Ice-bag to spine and head relieved pain. Liq. hydrarg. perchlor. Quinine. Turpentine. Blister to neck. Digitalis. Carbonate of ammonia. Brandy, \bar{z} iv.

Duration.—Five weeks.

Result.—Death.

Autopsy refused.

CASE XI.

T. S., æt. 45, warehouseman, admitted June 7th (Dr. Johnston).

Prominent symptoms.—Delirium; was delirious for fortnight before admission; almost constantly so while in Hospital, occasionally getting out of bed, but usually muttering to himself; will sometimes answer rationally if spoken to loudly. Dilated pupils. Deafness. General hyperæsthesia. Subsultus tendinum. General rigidity at times. Slight vomiting. Swelling of knees. During delirium hands often feeling head and back of neck as if in pain. When conscious, complains of pain if right temporal region is pressed on. Masturbation. Constipated bowels. Phosphatic urine. Temperature—usually between 97° and 99° F.; lowest, 96°; highest point, 103°, four days before death. Pulse—usual rate, 100; lowest rate, 72, usually low in early part of illness; highest rate, 140, during later stages.

Principal treatment.—Morphia hypodermically. Ice-bag to neck and head relieved pain. Liq. hydrarg. perchlor.; t. cinchon.; quinine; iodide of iron; digitalis; iodide and bromide of potash; salicylic acid, seemed slightly less delirious after it. Rum, ζ iv. Milk. Strong beef-tea.

Duration.—Eight weeks.

Result.—Death.

Post-mortem appearances.—Abdominal and thoracic viscera healthy. Great emaciation. Skull—thickening of arachnoid; effusion under arachnoid; lymph in its spaces, especially near cerebellum; pallor of velum interpositum and choroid plexus; dilatation of ventricles; increased serous fluid in them.

CASE XII.

A. M., æt. 25, labourer, admitted June 14th (Dr. Johnston).

Prominent symptoms.—Rigors. Insomnia. Delirium, trying at times to get out of bed. Pains in head. Right pupil dilated more than left. Slight deafness. Sibilant sounds over upper part of chest. Pains in lower part of back on sitting up. Intermittent sickness. Constipated bowels. Phosphatic urine. Great emaciation. Temperature—ranged from 98° to 100° F. while in Hospital. Pulse—average rate, 65; highest rate, 104; lowest rate, 52.

Principal treatment.—Morphia; pot. iodid.; pot. bromid.; perchloride of iron; carbonate of ammonia; cinchona; ol. morrhuæ.

Duration.—Thirteen weeks.

Result.—Death.

Post-mortem appearances.—A slight amount of lymph at base of brain. Dilatation of ventricles.

CASE XIII.

J. D., æt. 26, labourer, admitted June 20th (Dr. Mackey).

Prominent symptoms.—Rigors. Pains in head and back of neck. Retraction of head. Insomnia. Tenderness over upper dorsal region. Delirium. Subsultus tendinum. Vomiting. Special senses not affected. Constipated bowels.

Principal treatment.—Ice-bag to head. Liq. epispasticus to neck. Ungt. sabinæ; iodide of potash; cinchona; bromide of potash; hydrate of chloral. Tincture of walnut (t. nucis juglandis) relieved sickness for a time.

Duration.—Five weeks.

Result.—Death.

CASE XIV.

A. C., æt. 17, servant, admitted July 12th (Dr. Johnston).

Prominent symptoms.—Slight rigors. Giddiness. Vomiting. Retraction of head as soon as rigors passed off. Herpes on lip. Tenderness on pressing spine. Ringing sounds in ears. General hyperæsthesia. Five days from commencement retraction of head disappeared. Temperature—highest, 104.2° F.; lowest, 98° F. Pulse—average rate, 70.

Principal treatment.—July 12th—Hydrarg. chlor., gr. iv.; pil. rhei co., gr. iv.; extract. hyos., gr. iv., statim. ℞. pot. iod., gr. x.; sodæ bicarb., gr. xv.; gnaiaçi, gr. x.; aq., $\bar{\bar{3}}$ j., 6tis horis. July 20th—Pulv. Ipecac. co.; hyd. e creta, gr. v., 6tis horis. July 25th—Ol. morrhuæ.

Duration.—Three weeks.

Result.—Cure.

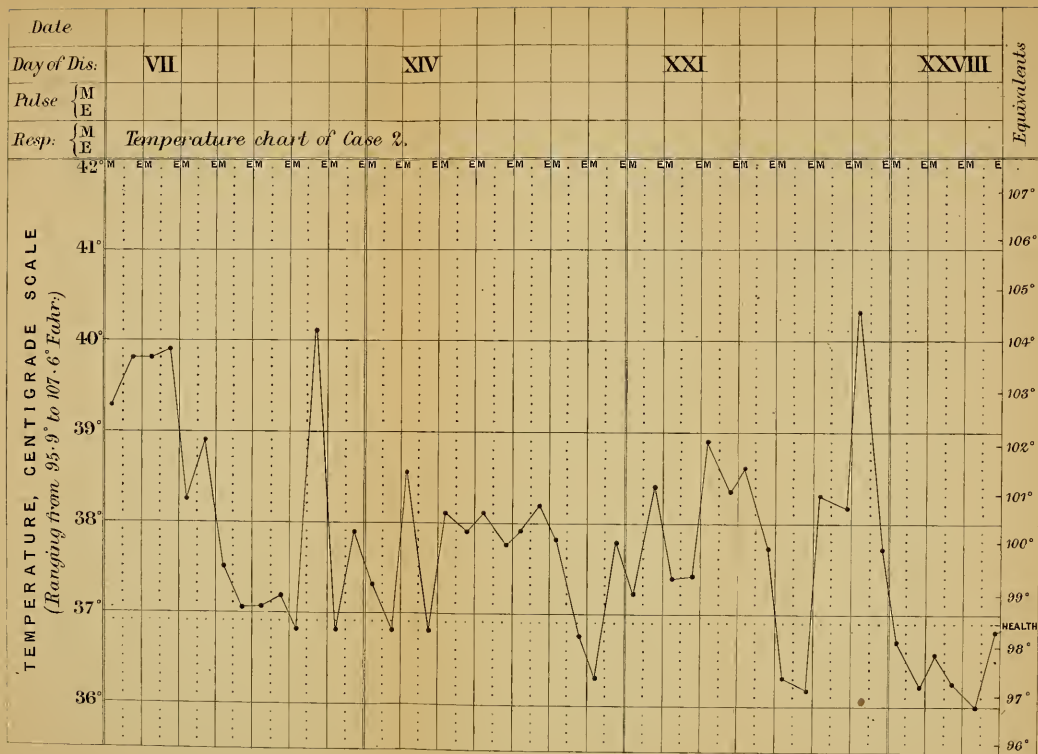
Remarks.—It will be seen that six of the above cases were under 18 years of age, the remaining eight being between 20 and 50. Also that nine of the patients died, and that five were cured. One case followed closely after measles. In some epidemics enteric fever has been prevalent; it was not so in this. At the time of the epidemic the Out-Patient department furnished an unusual number of cases of severe headache.

Symptoms.—The disease, where a clear history could be obtained, commenced with rigors, followed by great pain in the

head, often in the back also. The pain was usually unaccompanied by local tenderness, and in the head especially it was not localised. It was constant at first, but afterwards became intermittent in character. The delirium, noisy at first, became in the late stages low and muttering. In some cases pains in the legs were noticed, and after they disappeared, stiffness and hyperæsthesia. The knee and ankle joints swelled as in rheumatism. There was also abdominal pain. The vomiting was a most distressing symptom, and very constant in severe cases; it began with vomiting of food, and then of thin greenish fluid without preliminary retching. The extent to which the head was retracted bore no relation to the severity of the cases. In one case (not recorded) the retraction was so marked that a slough formed from the occiput pressing between the scapulæ. There was great restlessness in the early stages. The patients generally, if not tossing about, lay on the face; when sitting up in bed, they took great care to keep the neck, head, and shoulders fixed. Herpes on the face and neck were common in mild and severe cases alike; in three cases there were spots on the conjunctivæ; strabismus in two cases. Flushings of the face were common. Unable to bear any noise at first, they afterwards became deaf. The bowels were almost invariably constipated. The urine was passed in abundance, and was frequently phosphatic. The tongue, in the early stages moist and furred, was in the later dry and brown.

If the disease was prolonged, there was great emaciation, bed-sores, paralysis of rectum and bladder, and in a few cases of the pharynx. In one child trismus supervened. Dilated pupils were constant in the late stages, and the breathing irregular. It was not uncommon to find for several successive days as much as six degrees difference between the morning and evening temperature. A morning rise of temperature for some days was noticed in four cases. There was usually no relation between the pulse and the temperature.

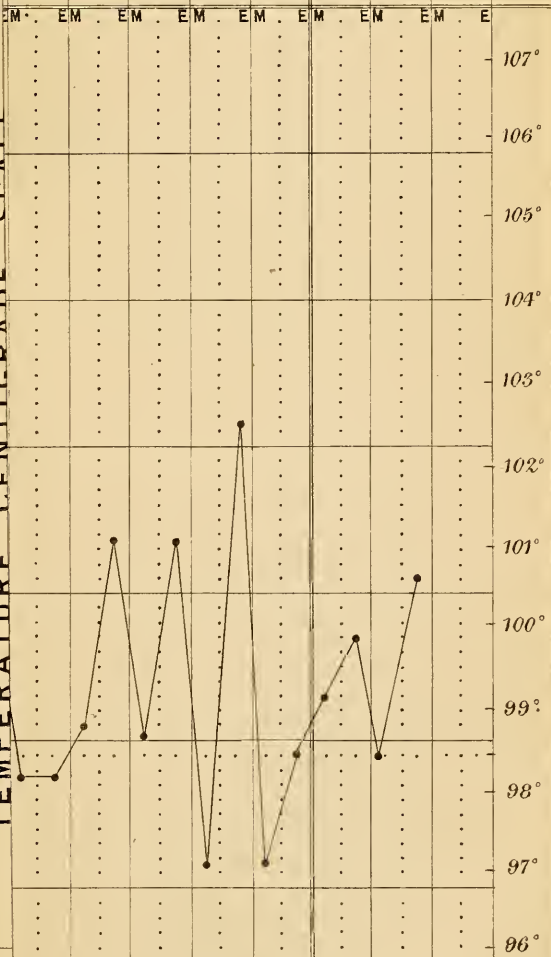
Treatment.—A very great variety of drugs were employed, often uselessly. Mercury given in the early stage until its constitutional effects were obtained (Case I.), and followed by large doses of the iodide of potassium, seemed to do good. Leeches applied to the temples greatly relieved the headache. Morphia hypodermically was of great use temporarily, but full doses were often necessary. Ice-bags to head and spine benefited some cases.



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Equivalents

TEMPERATURE CENTIGRADE SCALE



ON
CERTAIN FOREIGN BODIES
EMBEDDED IN THE TISSUES WITHOUT PRODUCING
INFLAMMATORY SYMPTOMS;
WITH REMARKS ON THE
ALLEGED TRANSIT OF NEEDLES, &c., FROM THE STOMACH
TO THE INTEGUMENT.
BY
ALBAN DORAN.

It is generally admitted that needles, pins, and other solid inorganic bodies may remain in the flesh, muscles, or even in the viscera of man, without setting up any of the various well-known pathological changes that might be expected under such circumstances. They generally enter the body by a wound in the integument through which a little atmospheric air must be admitted; this, together with the presence of an apparently irritant extraneous substance, at first sight represents a somewhat serious complication. Moreover, the wound itself is of the punctured class, by no means the mildest variety of external injury, as has long been admitted by authorities and taught in educational works on surgery.

Yet the difference between theoretical prognosis and the results of practical observation is in no branch of surgery better illustrated than in this subject, since it is well known that none of the results to be expected, in the conditions above referred to, necessarily follow the impaction of certain foreign bodies in living tissues, at least when such bodies do not interfere with the functions of any important organ.

Most of the systematic works on surgery assert, in general

terms, the immunity from inflammation observed in these injuries, without citing any particular case to the point; and while instances of hysteria, where the patient has inserted needles under the flesh, are recorded as a rule to illustrate the peculiar tendencies of hysterical subjects, examples of accidental impaction of similar substances are, on the other hand, generally related to illustrate not so much their effects, as the manner by which they have been removed through the skill of the surgeon. My object is, firstly, to consider the small amount of mischief which such bodies may inflict on the structures they pass through, illustrating the fact by actual cases collected for the purpose. Secondly, I will demonstrate, from recorded cases, that, recognised as the above fact is, it is the exception and not the rule; in other words, needles and similar foreign bodies generally induce inflammatory swelling and subsequent suppuration. Lastly, I will make some remarks on cases where certain sharp substances have been said to travel through the walls of the stomach or intestines and appear under the integuments, a phenomenon associated with the questions under discussion.

I base this memoir on a case that was under my own care a few years ago, where I closely observed the affected part, from a few hours after the injury till recovery, and failed to find a trace of any inflammatory symptom in the vicinity of the impaction of three foreign bodies of considerable size. I will not consider any of those examples where bullets and similar substances have remained in the organs or tissues of patients for years after having become encysted, as that implies at once a morbid process; most interesting, it is true, as illustrating the often-repeated fact that pathological changes are, on the whole, carried on for a salutary end, though circumstances may cause them to miss their object and terminate in disaster to the organism. The circumstances, in the above particular cases which I now dismiss, are attacks of constitutional disease or local external injuries in the neighbourhood of the cyst, which cause it to inflame in a lower type of inflammation than previously occurred when the irritation of the body produced the cyst itself. But here such conditions must be reckoned as simply a possible *result* in the class of cases to which I desire to draw attention, and as one reason why surgical interference is justifiable, though at first it might seem best to leave well alone.

My own case may be detailed as follows:—

On April 23, 1873, K. M., aged 16, a thin, ill-nourished maid-of-all-work, applied to me for relief in the Surgery of St. Bartholomew's Hospital, where I was at that period holding the appointment of house-surgeon. She complained that, in fall-

ing down a flight of stairs half an hour before, her right arm had struck a three-pronged fork, which ran deeply into the flesh. On extracting it, one prong was found entirely and the others partially broken off. I found three punctured wounds on the outer side of the patient's right arm a little below the insertion of the deltoid, but I could not at the time detect any foreign body in the wound, and water-dressing was applied over the punctures.

On May 2d the patient came a second time into the Surgery, and I found the three wounds almost healed; but on the inner side of the right arm, internal to the biceps and a little lower than the external punctures, a sharp projection could be detected under the skin. There was absolutely no tenderness nor sign of hardness or fluctuation around the foreign body which caused the projection. I cut down upon it, and extracted with a pair of dressing-forceps one of the missing prongs, $1\frac{3}{4}$ inch in length. Not a drop of pus or serum oozed from the wound, which I covered with a pad of wet lint; it healed by the first intention.

On May 6th, in my absence, Mr. Taylor removed a second prong; he could not detect any sign of inflammation in its track.

On May the 9th I saw the patient once more, and could feel the third prong projecting under the skin, in precisely the same manner as did the first and second, and with an equal absence of any signs of plastic or suppurative inflammation. This I extracted without any difficulty, and applied water-dressing as before. The last two wounds healed in a few days, like the first. The girl, contrary to my advice, had been using her arm freely in her domestic duties before the extraction of the prongs, and had never felt painful sensations of any kind. She was in a feeble state of health when under my treatment.

A case was under the care of Mr. Marrant Baker in 1872, which has already been made public.¹ A young girl injured her knee by a fall; it was poulticed, and after a day or two a needle was found protruding over the patella; in a fortnight five more were removed from the same region, and within a few months later her medical attendant, Mr. Wicksteed of Walthamstow, removed 101 needles and pins, many of large size, deeply embedded. In St. Bartholomew's Hospital, Mr. Baker and Dr. G. Bagot Ferguson removed ten needles and pins. It is a significant fact that all the latter had been deprived of their heads. Other circumstances favoured the suspicion of hysterical deception.²

¹ *Lancet*, July 20, 1872.

² In searching numerous records where hysterical women have swallowed or thrust into their skin numbers of pins and needles, I find that in most cases

I refer to the above case, because Mr. Baker has informed me that no sign of any form of inflammation could be detected along the track of any of the needles. This and the first example are the only instances that I have seen, in my own experience, where similar foreign bodies have failed to cause pathological changes.

In the Catalogue of the Pathological Series, Museum of the Royal College of Surgeons, in reference to specimen 68, we find it described as 'a hundred needles of various sizes, and most of them entire, which were extracted from the body of a Danish Jewess.' Then follows a long description of the case, which was under the care of Dr. Herholdt, and is well known in the annals of surgery. In twelve months 273 needles of different sizes were extracted, through incisions, from various parts of the body. The patient would complain of great pain, and Dr. Herholdt would forthwith separate considerable portions of the skin of the tender part from the deeper tissues and search for the needles, which he would then extract. It may be presumed that there was no inflammation around the needles, the pain being probably caused by pressure against nerves, since in the record of this same case I find that it is carefully noted that over a hundred needles were removed from the neighbourhood of a very large tumour in the axilla, and the first needle of all extracted was also surrounded by a tumour, which when cut bled without any pus escaping. These 'tumours' were evidently from inflammatory effusion.

But on searching various archives of surgery, I find that instances where it is definitely recorded that no inflammation has been set up by the presence of these bodies are very rare.

The late Professor Partridge read before the Pathological Society¹ a case where Dr. Orwin had removed from underneath the skin on the inner side of the calf of a child an entire crochet-needle $2\frac{1}{4}$ inches long. There had been no lameness, 'and neither suppuration or pain indicated its presence.' Here one may reasonably presume that simple non-suppurative inflammation was also absent, since the pressure of the needle against inflamed surrounding tissues could hardly have failed to produce pain.

A very interesting case is recorded by Dr. Noë of Ehrenfeld.² He removed from the forearm of a tradesman a piece of a knife

the patients have not only given no motives for their eccentricity, but have stoutly denied any knowledge as to how the foreign bodies entered their system. On the other hand, a Jewish girl ran a number of pins under the skin of her arm, avowedly as a self-inflicted penance after dreaming that the spectre of her grandmother had reproved her for working on the Sabbath (Hager, *Die fremden Körper im Menschen*. Vienna, 1844).

¹ Trans. Path. Soc., 1853-54.

² Berliner Klinische Wochenschrift, 1868.

measuring 6 centimetres in length by $1\frac{1}{2}$ broad; it had remained in his arm forty-six days without any sign of inflammation. 'In this case it is much to be wondered at,' justly remarks Dr. Noë, 'that this relatively very large foreign body should have excited no inflammatory phenomena during its forty-six days' occupation of the interosseous space, yet some one or other of the muscles must have been penetrated by it—the pronator quadratus and interosseous ligament were so most assuredly.'

In Hager's work¹ I find a case where a needle was extracted from under the skin of the back of a boy. 'No pus had formed around it, and nobody knew how it came there.' Campbell² removed a needle from under the skin of a woman; there was not a trace of pus, *but a painful swelling*. This latter condition puts it out of the present category.

Hager also gives an instance where an imbecile girl put seven pins under the skin of her arm through a venesection-wound. Five were dissected out. The condition of the tissues around them is not recorded, but the two remaining pins 'caused no pain, and did not hinder either the movements or the free use of the arm.' Here, then, there was presumably no inflammation.

A soldier ran a piece of glass 'the size and thickness of a groschen' into the sole of his foot. It remained twelve years there, and he could stand and march all the time without feeling any pain. At the end of that period suppuration was set up around the foreign body, after the patient had been mounting guard in wet weather.³

I cannot find any more cases where there is direct or presumable evidence of foreign bodies remaining in the tissues without causing inflammation, though the above examples afford satisfactory evidence that such *may* be the case. On the other hand, it is most certain that in the vast majority of patients foreign bodies rapidly set up inflammatory symptoms, generally of a suppurative type. This is the case even with needles and pins, in whatever way they may have entered the system.

In several of the cases already given, some out of many foreign bodies introduced into the same patient have set up tumefaction and suppuration. In one example presently to be again referred to, under Mr. (now Sir Henry) Thompson,⁴ the pins found under the skin 'were enclosed in small superficial abscesses.' Before one pin was extracted at the wrist the patient 'felt pain first of the shoulder, and traced its passage along the outer margin at

¹ *Op. jam cit.*

² Duncan's Medical Commentaries for the Year 1783-84. London, 1785.

³ Weitz, Neue Auszüge aus Dissertationen, vol. ii. p. 194. But here the glass was probably encapsuled.

⁴ Trans. Path. Soc., 1853-54.

the biceps muscle by a red slender line, although she could never feel the pin itself.'

In another instance Fischer found a needle in an *abscess* over the tibia.¹

Referring again to Hager's work, I find that in ten out of twenty cases of small foreign bodies lodging under the skin, evidence of inflammation is distinctly recorded. In six of the remainder nothing is said on the subject; in four the evidence of complete absence of inflammatory symptoms is tolerably clear. This includes Noë's, Duncan's, and Weitz's cases, and the instance where a needle was extracted from the arm of a boy, already quoted here direct from Hager's treatise. In every record of alleged travelling of pins, &c., from the alimentary canal to the surface, some at least of those bodies have been removed from superficial abscesses. Larger foreign bodies almost always excite suppuration.

I have already referred to the subject mentioned at the end of the last paragraph, and it is hardly out of place here to discuss it briefly; for many surgeons are prudently sceptical, and a collection of instances of actual transit of needles or pins to the subcutaneous tissue from the stomach or intestine may be acceptable to such truly scientific doubters. In most of these cases it is necessary to bear in mind the words of Mr. Pollock: 'The peculiar impulses of an hysterical mind occasionally induce a female to practise an amount of deception towards the medical attendant, which may induce him, on the first view of the case, to take for granted that which, upon careful investigation, would turn out to be fictitious.'²

Then, again, well-known examples are recorded where pins and needles have been swallowed, and retained for a long while in the stomach or intestines, not a single one out of their number ever appearing near the surface.

Firstly, there is Mr. Marshall's case,³ where a pound of pins were found in the duodenum and 9 ounces in the stomach of a woman who during lifetime had suffered from various symptoms of gastric irritation. Not one pin or needle was found under the integuments.

Hager⁴ records the fact that a young Polish lady carried for five months in her stomach twenty iron nails, seven iron sash-bolts, one hundred and one pins, two paternoster beads, and all sorts of rubbish besides, but not one worked its way to the surface. In Mr. Marshall's case, where, too, a great number of

¹ Virchow und Hirsch, Jahresbericht der Medecin, 1866, Fischer's case.

² Holmes' System of Surgery, 2d ed., article 'Injuries of the Abdomen.'

³ Med.-Chirurg. Trans., 1852, p. 65.

⁴ *Op. jam cit.*

pins were swallowed, the coats of the stomach were found extremely hypertrophied.¹ Possibly the same occurred in the Polish girl, and thus the transit of the pins and nails would be resisted. Instances are well known where small bodies have lodged in the cæcum and colon, and set up serious or even fatal results, without one of them reaching the abdominal parietes.

But however much we may doubt that an hysterical subject has swallowed pins and needles, there is no reason why, if such articles have once entered the stomach, they should not find their way to the surface. Accumulations in the cæcum set up irritation which often ends in abscess opening in the iliac fossa, and through it the foreign body or bodies may be discharged; or intestine irritated in the same manner may become thickened and immediately adherent to the abdominal walls,² an extraneous substance could then escape all the more readily. Now the same may reasonably be expected to occur when the foreign bodies are not fruit-stones or like indigestible portions of articles of food, but pins or similar inorganic bodies, whether lodged in the intestines or not lower down the alimentary canal than in the stomach.

By far the most authentic case of pins passing from the alimentary canal to the surface is that, already referred to, which was read before the Pathological Society in March 1854 by Sir Henry Thompson. The history is carefully recorded. There seems to have been no suspicion of hysteria; for the fact that the patient was 'aged 26, a tolerably healthy-looking servant-girl,' is in itself hardly sufficient evidence of the existence of that malady; and the coincident appearance of gastric symptoms and superficial abscesses containing pins corroborates the belief that the latter passed from the stomach or intestines to the subcutaneous cellular tissue.

The patient, being engaged in taking down linen from a clothes-line, had put a number of pins into her mouth, when a person seized her suddenly from behind 'for fun,' and she immediately bolted all the pins. 'She felt no alarm about the accident,' but, a week later, felt a pain beneath the left lower ribs which lasted twelve months, when a pin was drawn from a red swelling in her left breast; two more *headless* pins were soon afterwards removed from the same part. During the subsequent four years, nineteen more pins came to the surface, and

¹ See specimen 1141 B, Pathological Series, Royal College of Surgeons; also 1141 C and D, which consist of the pins found in the stomach, and of some more found in the patient's bed, 'bent as if prepared to be swallowed.' The pins are not headless, as in Sir Henry Thompson's and Mr. Baker's cases.

² See Path. Catalogue, Royal Coll. Surg., vol. iii.; history of spec. 1185.

she suffered all this time from severe pains in the left side ; but at the end of that period vomiting and hæmorrhage set in, and she threw up, after two months' persistence of these symptoms, *several pins' heads* on different days. The pins were removed 'in every case but one, from small superficial abscesses,' by Mr. Jones of Tenby.

This, I repeat, is decidedly the most authentic case of the kind on record, at least as far as my own researches in medical literature have extended. There can be no doubt that the patient swallowed the pins ; the accident at the time did not alarm her, but her master at once, we are told, solicited medical relief. The pain a week later about the left hypochondrium must have been due to gastric irritation, and it continued for twelve months, during which period no pins appeared under the integuments. In the meantime, it may be fairly presumed, the coats of the stomach not only became hypertrophied as in Mr. Marshall's case, but also adherent to the walls of the abdomen. The projection of one single pin's point from the anterior aspect of the stomach so as to scratch the tissues of the opposed parietes would readily set up adhesive inflammation without necessarily causing widespread peritonitis. By muscular movements the pins might be readily shifted to under the left breast, or still farther, after a lapse of time. The greater number of pins afterwards removed were found under the integuments near the left breast.

Still it must be admitted even in this case that a sceptical surgeon might assert with much reason that though the evidence of the swallowing of the pins is perfectly trustworthy, it does not follow that the pins found under the integument were the same as those which had entered the stomach. The belief that pins and needles may travel from the stomach to the surface, much as it is doubted by competent professional observers, is almost universally believed among the lower classes in this country, and the patient in this case having heard of alleged instances of this phenomenon may have desired to make herself an object of interest by secretly inserting pins under her skin to make those about her believe they had really passed there from the alimentary canal. One very suspicious point is the fact that nearly all the pins had lost their heads, as in Mr. Baker's case already recorded, as if the patient had nipped them off to facilitate the passing of the entire shank under the cutis. The suspicion is heightened when we find that the patient afterwards vomited a number of pin's heads. This looks as if she thought (supposing her to be a malingerer) that she would be less suspected if she returned some of the pins from the stomach by the mouth ; but finding that they did not return, and being afraid

of swallowing any more entire with their sharp points, she had adopted the ingenious expedient of swallowing the heads alone. Among the great number of pins found in the stomach of Mr. Marshall's case, and preserved in a glass bottle in the Museum of the Royal College of Surgeons (1141 C), I cannot find one with the head broken off, though a great number are broken through the middle of the shank, as is one which was found in the patient's bed with several others entire but bent. The angle between the side of the head of an English pin and its shank is too wide to form a deep rim in which more gastric juice could collect than over the surface of the rest of the pin, and thus eat the head off; moreover, the shank is at its thickest, and never constricted, at that point. In specimen 1141 C, alluded to above, I cannot detect one pin where the head is undermined by the action of the gastric juice, nor even so much as a trace of any such action between the head and the shank more than over the remainder of the entire foreign body, though many of these articles had been over seven years in the patient's stomach.

But the above case is much less doubtful than any other I can find. Some surgeons might adopt a middle opinion, and agree that the first pin found its way to the left breast as a '*bona-fide traveller*' from the stomach, whilst the remainder, or all those found far from the left hypochondrium, were inserted under the skin by the patient. I have failed in finding nearly so satisfactory evidence in any other case where the same conditions have been alleged to exist.

In Hager's work one instance is noted where a girl, aged 25, and addicted to somnambulism, vomited and passed per anum a number of pins. Some were found under the skin, but in such situations as the inner angle of the left eye and the nape of the neck! He then gives many similar cases, and in a few the probability that some out of many needles passed from the alimentary canal is almost as great as in Sir H. Thompson's example. As for foreign bodies other than pins and needles, there is no doubt that, lodging in the cæcum, they have set up pericæcal abscesses, through which they have been discharged externally. Hager gives cases of needles being eliminated in the same way. As for those which he asserts have been discharged, not from the right iliac region or right groin, as usual, but from the umbilical region, they probably came direct from some part of the colon rather than from the stomach. The substances most frequently so expelled are chicken-bones, fish-bones, fruit-stones, or ears of various cereals. In one case,¹ a needle, swal-

¹ Virchow und Hirsch, Jahresbericht, 1866.

lowed with a black thread attached, is alleged to have been found, thread and all, in an abscess over the tibia.

From the results, then, of clinical research into the subjects under discussion, it may be concluded—

1. That small inorganic foreign bodies *may* remain a considerable time in the tissues, more especially in connective tissue, without exciting any kind of inflammation; and hence without causing the formation of abscess, or becoming encapsuled.

2. That the above condition is nevertheless quite the exception—such small bodies exciting, as do larger ones, inflammation, almost invariably ending in the formation of abscess, or in rare cases terminating in the encapsulment of the same bodies.

3. That whilst it is admitted, on well-attested evidence, that bodies like plum-stones, ears of corn, &c., may be discharged externally from the cæcum and colon; on the other hand, satisfactory proof that articles like needles and pins have been discharged from the stomach or intestines, and travelled under the integuments far from the abdomen, is still much needed.

4. That one thing is certain—namely, the fact that several cases are known where numbers of pins or needles have been swallowed wilfully or accidentally, and have remained months or years in the stomach, exciting various serious symptoms, without one of the same bodies ever finding its way to the integument.

5. That a few cases exist—more especially one instance, already dwelt upon—where it is known that pins or needles have been swallowed, and where similar bodies have been found in abscesses or swellings as near to the stomach as the under side of the left breast, affording strong presumptive evidence that they are some of the same that have been swallowed.

6. That in cases where it is alleged that such bodies, after being swallowed, have travelled under the skin as far as to the extremities, we generally find strong proof of malingering, and not unfrequently absolutely no proof that any pins or needles have been swallowed at all; and when foreign bodies are found in situations remote from the stomach, others appearing in its immediate vicinity, as noticed in the last paragraph, it is reasonable to believe that the former have been placed under the skin by deception on the part of the patient, who has found from practical experience that some of the articles swallowed have appeared under the integument in the neighbourhood of the stomach.

The line of treatment to be adopted when foreign bodies have been detected near the surface of any part of the trunk or extremities, is quite simple, and may be found in any text-book on surgery, together with the well-known rule concerning the avoid-

ance of purgatives after the swallowing of sharp or otherwise dangerous substances. The use of emetics in the latter cases need only be named to be condemned.

When it is known that a patient has swallowed a number of pins or needles, and similar articles begin to appear subcutaneously in the epigastric, left hypochondriac, or any region more remote from the stomach, a most efficient observation of all the actions of the patient day and night will be necessary; but even then the most experienced nurse may be baffled, for in such cases the patient seems to acquire what may be called a morbidly perfect faculty of deception—just as a delirious man exerts his force so as to produce effects which would be far above his power when in full health and perfect control of his muscular system. Many surgeons will in these instances be hardly satisfied of the actual transit of needles from the stomach to the surface, until by post-mortem examination they can find an adhesion of that viscus to the parietes, or a direct communication of the latter with the former through an abscess, one or more of the foreign bodies being actually found wholly or partially in the connecting medium. Even then, as I have before noticed, this will not prove that the needles found farther from the stomach have ever been in it; unless the surgeon can be sure that he has tracked a hard, long, and thin body under the integument from the site of adhesion, day by day, till he has himself removed it from under the skin of the thorax or extremities.

When a foreign body is detected near the surface, it should decidedly be removed at once, for fear of its doing any mischief. Methods for proving the suspected metallic nature of such a body through the agency of magnetism have been devised and suggested by Smee¹ and Marshall. Such tests should be applied when a superficial abscess forms in a patient after needles have been extracted from the subcutaneous cellular tissue. If the presence of metal is indicated, but no needle found on opening the abscess, the latter should be left open for some time till the point of the needle appears within its cavity, or near it.

The usual practice is to dissect down to the point of the needle or pin, and then make use of the forceps. It is always better to adopt—especially when there are a great number of such substances to remove—the method employed by Dr. Judson B. Andrews when he had to extract 286 needles from an insane patient within five months. To use his own words:—

¹ On the Detection of Needles and other Steel Instruments impacted in the Human Body. By Alfred Smee, F.R.S. Renshaw, 1845. See also Sir B. C. Brodie's case, reported in the 'Lancet,' October 28, 1876; it was read before the Med.-Chir. Soc. on October 24th.

'They were removed in a few instances at first by cutting down upon them. This proved to be a painful and, from the movements of the needles in the tissues, a difficult process. Hæmorrhage from the small vessels at times gave some trouble. Afterwards, by manipulation, the ends of the needles were engaged between the thumb and forefinger, and the points, forced through the skin, were seized and the needles extracted with forceps. Sometimes much force was required to withdraw them.'¹

One great advantage of Dr. Judson Andrews' method is the avoidance of the formation of numerous cicatrices, which must result if the needles are dissected out. This is a great desideratum in hysterical subjects, where the presence of scars might open a fresh field for morbid contemplation on the part of the patient.

¹ American Journal of Insanity, 1872-73. 'Case of excessive hypodermic use of morphia. Three hundred needles removed from the body of an insane woman.' None of these were supposed to be swallowed or found in the stomach, and some 'produced little local irritation or trouble beyond the pricking sensation.'

RESEARCHES

MADE IN THE

PHARMACOLOGICAL LABORATORY OF ST. BARTHOLOMEW'S HOSPITAL

UNDER THE DIRECTION OF

T. LAUDER BRUNTON, M.D., F.R.S.

I. ON THE PHYSIOLOGICAL ACTION OF CASCA BARK.

By T. Lauder Brunton and Walter Pye.

The practice of subjecting persons suspected of crime or witchcraft to an ordeal by poison prevails very extensively along the western coast of Africa. The poison employed is not the same along the whole coast-line. In Calabar, which lies about the middle, the natives employ the bean of the *Physostigma venenosum*, or, as it is generally called, the Calabar bean. To the north of Calabar, in Sierra Leone, and to the south, in Angola, the favourite ordeal poison is not a fruit but a bark, which bears in different districts the names of 'doom,' 'gidu,' and 'sassy,' 'saucy,' 'cassa,' or 'casca.' This bark is obtained from the *Erythrophieum Guinense*, which, like the *Physostigma venenosum*, belongs to the natural order *Leguminosae*. The bark is of a brownish-red colour, is in pieces about 8 inches long, 4 broad, and between $\frac{1}{4}$ and $\frac{1}{2}$ thick. When treated with water it yields a brownish-red infusion.

There are two ways in which it is employed by the natives. One is to make the suspected person fast for several hours, and then to give him a few grains of rice and some infusion of the bark. If he vomits all the grains of rice and is not purged, he is said to be innocent; but if he is purged, he is pronounced guilty. The other way is to bend both ends of several boughs of trees into the ground so as to form a long archway, through which the accused walks in a stooping position after a dose of the infusion has been administered. If he is able to walk through without stumbling, he is considered to be innocent; but if he stumbles, he is said to be guilty and at once despatched.

The chief effects of the poison by which the innocence or guilt of the accused are decided, are thus vomiting, purging, and loss of muscular power or co-ordination. The effects of the poison on man, as described by some missionaries, are vomiting, redness and glazing of the eyes, and loss of the power of contracting the muscles throughout the body; so that when the poison has fairly commenced its action on the system, the sufferer is incapable of standing or walking, and the head rolls heavily about the breast and shoulders. Its action on animals was tried by Santos,¹ who says that the decoction produced alternate dilatation and contraction of the pupils, appearance of delirium, violent retching, vomiting, symptoms of tetanus, and finally death. Professor Liebreich has also investigated its physiological action, but we have not yet seen a full account of his experiments.

A small quantity of the bark having been brought from Angola by Mr. Monteiro, who had obtained it with considerable difficulty, he kindly gave it to us, and we began a minute investigation of its physiological action, so as to ascertain not only the exact manner in which death is produced by the drug, but the mode in which the various functions are affected by it, and its possible uses in medicine.

General Action of Casca.

Beginning our experiments with the simplest forms of life, and proceeding to the more complex, we found that a watery extract of the bark did not interfere in the least with the germination of seeds; it did not hinder the growth of the yeast-fungus, and ordinary mould (*Penicillium*) grows freely in it. It does not destroy full-grown bacteria nor infusoria; nor does a watery solution of the aqueous extract prevent the development of bacteria, but a watery solution of the alcoholic extract does so, a fact which seems to indicate the presence in the alcoholic extract of some principle which is absent from the aqueous extract. It has little or no action on invertebrate animals such as snails. On fishes and frogs its action, though much less than on warm-blooded animals, is nevertheless quite distinct, its administration being followed by irregular muscular movements and failure of muscular power. Birds are easily affected by it, a small dose producing violent vomiting and irregular muscular movements with difficult respiration. These symptoms are followed by loss of muscular power and death.

In cats and dogs it causes restlessness and nausea succeeded by violent and repeated vomiting. The respiration is very much quickened. The first symptom of any affection of the locomotor

¹ American Journal of Pharmacy, April 1849, p. 96.

organs in cats is a peculiar jerk of the hind limbs, as if something were sticking to the feet and the animal were trying to shake it off while walking; then the gait becomes staggering, and the animal ceases to be able to stand. Death generally occurs during a convulsion of an emprosthotonic character, apparently connected with an attempt to vomit. Consciousness seems to be preserved to the last.

The symptoms observed by us thus agree in most respects with those described by Santos, but we saw no appearance of delirium, nor any alternate contraction and dilatation of the pupil, although we looked for it carefully.

Analysis of the Symptoms produced by Casca.

Vomiting and purging.—It has already been mentioned, that while vomiting in those subjected to the ordeal by casca is regarded as a sign of innocence, purging is considered to be a proof of guilt. It is stated that the priests who prepare the infusion are able to produce either effect at will, the clear infusion being given to those whom they wish to prove innocent, while the dregs are administered to those who have offended them, or who at any rate have not propitiated them. In order to ascertain whether this was so or not we administered an infusion without the dregs to one cat, and an infusion with the dregs to another; but the result was contrary to what we expected, the one which had got the dregs recovering, while the other died. This might, however, be due to the fact that the infusion with which we operated was prepared from finely-pounded bark, which would readily yield up its active principle to water, while the infusion is probably prepared by the priests from coarsely-pounded bark, from which water would extract the poison more slowly; and if only allowed to remain a short time in contact with the bark, the infusion would be comparatively weak, while the dregs themselves would yield up their active principle in the stomach after being swallowed, and thus have a much more powerful action. The purging is due to the local effect of the poison on the intestines, for it only occurred when the poison was given by the mouth, and was never present when the drug was administered by subcutaneous injection. Vomiting, on the contrary, occurred as constantly when the casca was injected subcutaneously as when given by the mouth. The vomiting after subcutaneous injection of the drug is probably due to its being carried with the blood to the stomach, and irritating the sensory nerves of that organ in much the same way as when introduced directly into it. The reason why we believe the vomiting to be due to irritation of the nerves of the stomach rather than to the action of the drug

upon the vomiting centre in the medulla oblongata, is that when the vagi nerves were divided in the neck of one cat, a dose which would ordinarily have proved fatal produced no vomiting, nor indeed any of the usual symptoms. In other cases where vomiting occurred even after division of the vagi, it was less than usual, and it might be due to the irritation being conveyed from the stomach to the medulla by branches of the solar plexus instead of by the vagi. The purging is probably due to increased peristaltic action rather than to increased secretion, for infusion of casca introduced into a loop of intestine produced no increased secretion, as a solution of sulphate of magnesia would have done.

Muscular weakness.—Want of power to walk properly is the second symptom regarded as a proof of guilt in those subjected to the ordeal, those who stumble before they reach the end of the archway of boughs being at once executed. In attempting to ascertain the cause of this loss of power, we worked backwards thus: The motions of the limbs are due to the contraction of muscles. The contraction of muscles are due to the stimuli they receive from motor nerves. The stimuli which pass down motor nerves to muscles proceed from nerve-centres in the spinal cord or encephalon. Thus loss of muscular power may be due either to loss of power in the muscles themselves—in the motor nerves which supply them—or in the nerve-centres in the spinal cord or encephalon.

Action on muscles.—This was tested by laying one gastrocnemius of a frog in a solution of casca, and the other in an indifferent liquid, such as a 75-per-cent. solution of common salt. After some time the excitability of the two muscles by electrical stimuli was compared, and also their power to lift weights. They were found to be equal. This showed that casca was not a muscular poison, for had it been so, the muscle immersed in a solution of it would have lost its excitability before the other, and its power to lift a weight would have been lessened.

Action on motor nerves.—In order to ascertain whether the motor nerves were paralysed or not, the artery going to one leg of a frog was ligatured and the poison injected under the skin of the back. The poison was thus carried to every part of the frog except the ligatured leg. Immediately after death the excitability of the motor nerves was tested by the application of an induced electrical current from a Du-Bois Reymond's coil. It was found that the motor nerves of the leg to which the poison had been carried by the blood were not paralysed, and were quite as easily excited as those of the other leg from which the poison

had been excluded by ligature of the artery. The poison, therefore, does not paralyse the motor nerves.

Action on the spinal cord.—Some time after the injection of casca under the skin of a frog, the movements of the animal become more sluggish; are imperfectly performed; and when the toes are pinched, the foot is either moved lazily or not at all, instead of being promptly drawn up, as it normally is. The reflex activity of the cord is thus seen to be impaired, but we must not hastily conclude that this impairment is due to the direct action of the drug upon the nervous structures; for imperfect circulation of blood through the brain and spinal cord quickly deprives them of their power, and although stoppage of the circulation does not abolish the functional power of the nerve-centres so quickly in the frog as in warm-blooded animals, it nevertheless does so after a time. In order to discover whether the casca destroyed the power of the spinal cord by acting directly upon it or not, its action upon the heart was investigated, and we found that a short time after its administration it arrested the pulsations of that organ. It seemed therefore quite possible that the loss of power in the spinal cord was simply due to stoppage of the heart by the poison, but of course this was only probability, as the casca might act both on the cord and the heart. We decided this point, however, by administering casca to one frog, and waiting until the heart had stopped. The instant it had done so, we arrested the circulation in a frog of a similar kind and size by a ligature around the large vessels as they arose from the heart. In these two frogs the circulation was equally arrested, but in one of them the poison had been previously carried by the blood to the spinal cord, and could still act upon it although the flow of blood was stopped. If the casca had any paralysing action upon the nervous structures of the cord itself, reflex action ought to have ceased in the poisoned frog before doing so in the ligatured frog, but this was not the case. In both frogs reflex action ceased in almost exactly the same time. The abolition of the function of the spinal cord is therefore due to stoppage of the circulation caused by the casca, and not to the action of the drug upon the cord itself. The staggering gait, inability to stand, and paralysis which we have observed in dogs and cats, we attribute, like the paralysis in frogs, to disturbance of the circulation, and not to any special action on the nerve-centres.

Action on circulation. Action on the heart.—The first action of casca upon the heart of the frog is to cause it to beat more slowly, then the ventricle becomes irregularly contracted, some parts of it being firmly contracted and white, while here and

there other points are not contracted, and being filled with blood, look like little red pouches studding the cardiac surface. Finally, the ventricle stops altogether in a state of contraction, while the auricles continue to pulsate for some time longer. In cats also the ventricle sometimes becomes irregularly contracted before finally stopping, the lower part of the ventricle in one experiment having been contracted while the upper half was not, so that the lower half appeared to be partly pushed up into the upper in such a way as to produce a deep transverse wrinkle across the middle of the ventricle.

Action on the vagus.—It will be seen from the description thus given that the action of casca upon the heart of the frog is almost exactly like that of digitalis, as described by Messrs. Fagge and Stevenson.¹ Further experiments have shown us that casca also resembles digitalis in its action upon the vagus. A moderate dose of casca injected into the jugular vein first slows the heart, a further dose greatly quickens it, and another large dose again slows it. The first slowing is due to stimulation of the vagus roots in the medulla oblongata, for when the vagi are divided so as to cut the communication between the medulla and the heart, the pulsations again become quick. The quickening which a large dose of casca produces when the vagi are uninjured is due to its paralysing the ends of these nerves in the heart, and thus destroying the communication between this organ and the medulla quite as effectually as the division of their trunks by the knife. When the nerves, as they pass down the neck, are irritated by an induced galvanic current in their normal condition, they slow the pulsations of the heart, or stop it altogether; but after the injection of casca has paralysed their ends in the heart, no irritation of their trunks has any power to slow its pulsations. The final slowing produced by a large dose of casca must be due to the action of the drug upon the ganglionic apparatus within the heart itself, as the vagus ends are already paralysed. The extreme slowness of the heart in this stage is sometimes very remarkable, as in one experiment there were only three pulsations per minute, only one occurring in the time occupied by three or four respirations.

Action on the blood-vessels.—Casca has a most extraordinary power of contracting the blood-vessels. This is indicated by the pressure of blood within the vessels becoming high after its injection, notwithstanding the slowness of the heart's action; but it is proved most unmistakably by the fact that during the long diastolic pauses the pressure does not sink as it ordinarily does, but sometimes remains as high as 165 millimetres of mercury.

¹ Proceedings of the Royal Society, vol. xiv. p. 270.

When the arterioles are in their normal condition as regards dilatation, the blood flows readily out of the arteries into the veins, and the pressure rapidly falls in the arterial system during the cardiac diastole. When the arterioles are much contracted, however, as after the administration of *casca*, the flow of the blood out of the arterial into the venous system is impeded, the arteries remain full, and the tension of the blood within them high.

Digitalis also contracts the arterioles and causes the fall of pressure during the cardiac diastole to be slow. The mode in which *casca* and *digitalis* produce contraction of the blood-vessels, however, seems to be different. *Digitalis* causes it by stimulating the vaso-motor centre in the medulla oblongata, and this centre acts through the vaso-motor nerves upon the vessels. These nerves pass down from the medulla, through the cervical part of the spinal cord, along the splanchnics, &c., to the vessels. Consequently, when the communication between the vaso-motor centre in the medulla oblongata and the vessels is destroyed by dividing the cervical and spinal cord, the vessels dilate, and no stimulation of the vaso-motor centre has any power to cause them to contract. The contraction usually produced by *digitalis*, therefore, does not occur if the cord be divided before its injection, and is removed if the cord be divided after contraction has already taken place. This is not the case with *casca*, however, for we found that after the spinal cord had been completely divided in a cat opposite the second cervical vertebra, the blood-pressure after the injection of *casca* rose higher than in any other experiment. The *casca* must therefore act either on the blood-vessels themselves, the vaso-motor nerves, or some vaso-motor centre not contained in the medulla.

A further proof that *casca* acts on peripheral vaso-motor ganglia or nerves is afforded by an experiment in which the sympathetic nerve was divided in the neck of a rabbit, so as to sever the connection between the vaso-motor centre and the vessels of the ear on one side. On the other the nerves were left intact. After injecting a dose of *casca* the vessels in *both* ears became pale, and apparently there was no difference between them in the ear with the divided nerve and in the other.

We are rather inclined to the supposition that it does act on some such centre or centres—possibly ganglia—in or around the vessels themselves, because the local application of *casca* to a frog's foot, or its injection under the skin of the back, causes no contraction in the vessels of the web, as one would expect it to do if it acted on the vessels themselves. The arterioles begin to contract after a small dose of *casca* before any effect is produced on the vagus, so that the blood-pressure begins to rise before the pulse

becomes slow. The contraction also seems to last after the vagus is paralysed, and even after the heart has ceased to beat, so that when the animal is dying the blood-pressure falls very slowly.

The contraction of the vessels after the injection of casca is not confined to those which are under the dominion of the vaso-motor centre in the medulla. It has been shown by Ludwig and Hafiz, that while this centre can cause contraction of the vessels going to the intestines, it has little or no power over those supplying the muscles. Thus it happens that when this centre is irritated the blood-pressure does not remain high during the cardiac diastole, as one formerly supposed it would do; for although the intestinal arterioles are firmly contracted and prevent any blood from flowing into the veins, the arteries of the muscles remain uncontracted and the blood flows rapidly through them. As the blood-pressure after the injection of casca remains so high during the cardiac diastole, the arterioles in the muscles must be contracted as well as those in the intestines. In its mode of action upon the blood-vessels casca differs from digitalis, but agrees with ergot, which seems to cause contraction by acting rather upon peripheral vaso-motor nerves or ganglia, or on the muscular wall of the blood-vessels, than on the vaso-motor centre.

Action on the kidneys.—As the action of casca on the circulation so closely resembles that of digitalis, it seemed not improbable that it might also have a diuretic action, and we accordingly proceeded to try whether it had or not. The manner in which we experimented was as follows. A dog was anæsthetised with chloroform, the anæsthesia being kept up during the whole operation. A cannula was placed in one ureter so that the urine dropped from the kidney as fast as it was secreted, and the rapidity of secretion could be readily ascertained. The carotid artery was connected with a kymographion and the blood-pressure measured. On then injecting a dose of casca into the jugular vein, we found that the blood-pressure rose and the urine began to be secreted more rapidly. An additional dose raised the blood-pressure still higher, but the secretion of urine began to get slower instead of quicker, and when the blood-pressure had risen to its maximum the secretion stopped altogether. After awhile the blood-pressure began to fall, and the secretion again commenced. The explanation which we are inclined to give of these facts is that at first the casca, by causing contraction of the vessels generally, and raising the blood-pressure, increases the pressure in the glomeruli of the kidney, and thus causes the watery constituents of the blood to filter through them more quickly than usual. It thus increases the flow of urine. It next causes the vessels of the kidney to

contract more and more, so that notwithstanding the high blood-pressure in the arterial system generally, there is little blood in the kidneys. The pressure of blood in the glomeruli is consequently low and the secretion of urine scanty, and when the contraction of the renal artery becomes very great the secretion stops altogether. When the arterial spasm again relaxes, the secretion recommences at the same time that the blood-pressure falls. In this respect the action of casca agrees completely with that of digitalis.¹ It seems probable that casca will also, like digitalis, be found to have a cumulative action, should it be introduced into medicine; for the effect of any drug depends on the amount of it circulating in the blood, and this amount may be increased either by increasing the introduction of new quantities or by diminishing the excretion. It seems probable that the sudden appearance of dangerous symptoms during the administration of digitalis is due to its stopping excretion by the kidney while the drug is still taken by the mouth. The occurrence of poisoning by digitalis would thus be completely analogous to poisoning by curare in an experiment of Hermann's.² Curare produces paralysis of motor nerves when quickly introduced into the circulation by injection under the skin or into a vein, but does not usually prove poisonous when taken into the stomach. The reason of this is, that it is excreted by the kidneys as quickly as it is absorbed from the stomach, so that there is never enough of it in the blood at any one time to be injurious. When Hermann tied the renal vessels, however, so as to prevent excretion, curare taken into the stomach proved as certainly fatal as when injected into a vein. In digitalis-poisoning the drug itself causes contraction of the renal vessels, producing the same effect as the ligature applied to them by Hermann in the case of curare, and the same would probably be the case with casca.

Action on respiration.—The respiration is generally quickened by casca. This quickening appears to be due to a stimulating action of the drug upon the pulmonary branches of the vagus, as it was not observed when the vagi were divided before the casca was administered.

Action on temperature.—Casca does not appear to have any action either in lowering or raising the temperature of a healthy animal. We have not yet tried what effect it will have in fever.

Action on the eye.—When applied to the eye it has no action on the pupil, nor does it cause congestion of the conjunctiva or lachrymation.

¹ Brunton and Power, On the Diuretic Action of Digitalis. Proceedings of the Royal Society, 1874, No. 153.

² Du-Bois Reymond's und Reichert's Archiv., 1867, p. 64.

Action on the uterus.—Digitalis having been said to have a powerful action upon the uterus, an action which has been found by Dr. Dickinson to be useful in arresting menorrhagia by causing contraction of the uterus, we administered casca to a pregnant cat, but it did not produce abortion.

Probable Use of Casca in Medicine.

It is evident from the description we have given above of the physiological action of casca, that it is quite different from that of Calabar bean, the other ordeal poison employed on the African coast, which has become such a useful remedy since its physiological action was first ascertained by the admirable researches of Dr. T. R. Fraser. Unlike physostigma, it has no action on the pupil, and no special action upon the spinal cord. Its action is exerted chiefly upon the stomach, circulatory apparatus, and kidneys. Its action on the stomach seems to be rather a drawback than otherwise, as it would have been highly advantageous to have had a drug which would act like digitalis upon the heart without producing the sickness which sometimes obliges us to discontinue the use of the latter. As, like digitalis, it strengthens the heart while slowing its pulsations, it will be useful in mitral disease, and its diuretic action will prove serviceable in dropsy arising from this cause. At the same time its more powerful action on the vessels leads us to hope that it will be useful in advanced cases of cardiac dropsy when digitalis fails. This power of contracting the vessels also indicates that it will prove a useful hæmostatic more powerful than either digitalis or ergot, the virtues of which it seems to combine. Mr. Monteiro having kindly promised to obtain for us a large quantity of the bark, we trust we shall find that its action in disease corresponds to the hopes we have formed.

SECTION II.

ANTAGONISM BETWEEN STRYCHNIA AND HYDROCYANIC ACID.

By W. J. Simpson Ladell.

While attending the lectures on *Materia Medica* at the Hospital, it occurred to me that the physiological action of hydrocyanic acid indicated it as a probable antidote to strychnia. On mentioning my ideas to Dr. Brunton, he advised me to put them to experimental proof, and I accordingly made the following experiments under his direction:—

1. Two frogs of nearly equal weight had a drop of liq. strychniæ injected into dorsal lymph sac.

Frog A.—Injected at 12.43 A.M. At 12.48, tetanus. 12.49, clonic convulsion. Watched until 3 P.M., during which time it had seventy-six spasms. Only sign of life at 3 P.M. slight movement when touched. At 10 A.M. the following day was found dead.

Frog B.—12.43½ A.M., injected with liq. strychniæ. 12.47½, tetanus. Injected with two drops of prussic acid. Pupils very much contracted. Watched until 3 P.M. At 1 P.M. pupils became more dilated. When left at 3 P.M. was breathing quickly, and when touched had severe tetanus. While watched it had thirty-two spasms. 10 A.M. the following day was lively and apparently quite well.

2. Frog C.—Injected with one drop each of liq. strychniæ and prussic acid. Recovered, after severe spasms, apparently well in fourteen hours.

3. Frog D.—Injected with one drop liq. strychniæ, three drops of prussic acid. Recovered, after lying comatose some time, in twelve hours.

4. Frog E.—One drop liq. strychniæ, four of prussic acid. Died in three hours.

5. Frog F.—No strychnia and four drops of prussic acid. Death very quickly, exact time not noticed.

6. Frog G.—No strychnia, three drops prussic acid. Death in fifteen minutes.

7. Frog H.—One drop liq. strychniæ, three of prussic acid (strong). Death in one hour.

8. Frog I.—Two drops liq. strychniæ, four of prussic acid. Death in one hour. Almost no spasm.

9. Frog K.—Two drops liq. strychniæ, one of prussic acid (strong). Recovery, after spasm, in four hours.

SECTION III.

ANTAGONISM BETWEEN STRYCHNIA AND HYDROCYANIC ACID.

By J. Lauder Brunton, W. J. S. Ladell, and W. Outhwaite.

Some experiments made by Mr. Ladell in this laboratory having shown that the action of strychnia upon frogs could be modified and its poisonous effect diminished by the administration of hydrocyanic acid, we performed the following experiments in order to ascertain whether a similar antagonism prevailed in the case of warm-blooded animals.

Experiment I.

By means of a Wood's syringe 60 minims of liquor strychniæ B.P. mixed with 20 minims of dilute hydrocyanic acid B.P. were injected into the abdominal cavity of a cat. The animal died in thirty seconds without spasm. On post-mortem examination the lungs were normal. The heart contracted on irritation.

Experiment II.

Sixty minims of liquor strychniæ alone were injected into another cat in the same way. In one minute tetanic spasms occurred, and the animal died in two minutes and a half. On post-mortem examination the lungs were found congested, but the blood returning from them was not so dark as in Experiment I. The ventricles contracted on irritation.

Experiment III.

Thirty minims of liquor strychniæ mixed with fifteen minims of hydrocyanic acid were injected in a similar way. In fifteen seconds a spasm came on, which passed off in a minute and a half, but again recurred several times. In the intervals between each convulsion the breathing was spasmodic, and became more so as each convulsion approached. Respiration ceased in seven minutes after the injection. Ventricles contracted on irritation.

Experiment IV.

Thirty minims of liquor strychniæ alone were injected in a similar way. In forty-five seconds a convulsion came on, and death occurred in two minutes. The heart was found contracting. On post-mortem examination the lungs were not congested.

Experiment V.

At 12.47 P.M. fifteen minims of liquor strychniæ were injected into the peritoneal cavity of a cat weighing 8 lbs.

At 12.49 the muscles of the lower extremity began twitching, and the pupils dilating.

At 12.52 animal rushed about with its hind legs quite stiff.

At 12.55 spasm, with strong convulsion, followed by tetanus, which was especially noticed along the back, where the contraction of the muscles went on in a kind of wave from above downwards. The back was bent inwards.

At 12.57 respiration ceased, recommenced after about a minute of artificial respiration.

At 1 breathing spasmodic, and tetanic spasms followed one another at about ten to fifteen seconds' interval.

At 1.2 $\frac{1}{2}$ heart ceased.

Post-mortem.—Heart non-contractile on irritation, lungs natural, and other organs appeared normal. Blood coming from or going to heart did not look black.

Experiment VI.

At 2.25 P.M. fifteen minims of liquor strychniæ and ten minims of hydrocyanic acid (2 per cent.) were injected into the peritoneal cavity of a cat weighing 5 $\frac{1}{2}$ lbs.

At 2.27 convulsion ending in tetanic spasm, which tetanus almost immediately ceased, and at the same time the respiration did so too.

Artificial respiration was carried on, during which the cat vomited, and also passed urine.

At 2.43 the heart ceased beating.

On opening the animal a strong smell of hydrocyanic acid was perceptible, which was persistent about the lungs for some time.

The lungs were congested, as also were the intestines. Spleen was very small.

The auricles were contractile on irritation.

The blood was very black.

There was no tetanus at death.

Experiment VII.

At 3.5 P.M. fifteen minims of liquor strychniæ and five of hydrocyanic acid were injected into the peritoneal cavity of a cat weighing 3 lbs.

At 3.6 it staggered, raising itself on its hind legs and falling backwards. This it did several times. It then crawled about as though it had lost the use of its *forelegs*.

At 3.6 $\frac{1}{2}$ it vomited, and convulsions set in with very slight tetanus. It urinated, and the convulsions became violent.

At 3.7 the respiration ceased. On artificial respiration it recommenced to breathe.

At 3.11 a slight convulsion with slight tetanus. The breathing became spasmodic.

At 3.17 the heart ceased.

On opening there was a smell of hydrocyanic acid, and the organs and blood presented a similar aspect to those in the previous experiment. There was no tetanus at the time of death.

Experiment VIII.

At 3.37 P.M. fifteen minims of liquor strychniæ and five of hydrocyanic acid were injected into the peritoneal cavity of a cat weighing about 4 lbs.

At 3.40 there was a slight spasm, and the animal crawled about as though it had lost the use of its upper extremities. It was then convulsed, and the use of the upper extremities returned, but it lost the use of its lower ones. Its back then became bent, first backward and then forward. The pupils were dilated and the head bent back on its shoulders.

At 3.43 a strong tetanic spasm set in, which lasted a very short time. The animal frothed at the mouth.

At 3.47 breathing became spasmodic and then ceased. Artificial respiration was carried on for about two minutes, when the heart ceased beating.

There was no tetanus at death, and the post-mortem appearances were similar to those in the other two experiments.

Experiment IX.

At 1.23 P.M. ten minims of liquor strychniæ were injected into the peritoneal sac of a cat weighing $6\frac{1}{2}$ lbs.

At 1.25 hind legs commenced twitching.

At 1.26 had a slight spasm.

At 1.27 spasm and convulsed, ending in tetanus; forepaws being flexed and hind extremities extended; head not bent back, but back bent inwards; and the waves of muscular contraction were noticeable down the back. Began frothing at the mouth, and the breathing became spasmodic.

At 1.28 $\frac{1}{2}$ ceased breathing.

At 1.30, after artificial respiration, breathing recommenced, slight spasms continuing.

At 1.31 vomited.

At 1.40 breathing, after having been spasmodic, quite restored.

At 1.51 violent tetanic spasm, and breathing and heart ceased almost simultaneously.

Post-mortem.—Heart and lungs appeared normal, and so did other organs, liver tearing very easily, however. Blood in vessels round the heart was not black, nor were the auricles contractile on irritation. The muscles were rigid.

Experiment X.

At 1.6 P.M. ten minims of liquor strychniæ and five of hydrocyanic acid were injected into the peritoneal cavity of a cat weighing 5 lbs.

At 1.8 legs began twitching; it then fell backward, then forward, convulsed, with head bent back on shoulders, and back bent outwards. Pupils dilated. The tetanus was not great, and the wave travelling down the back was not noticed.

At 1.10 vomited and had strongish spasm, with spasmodic breathing, which became more marked as the spasms were ushered in.

At 1.14 the animal frothed from the mouth, and respiration ceased. Breathing was not re-established on artificial respiration, and the heart ceased at 1.18.

Post-mortem.—The heart was contractile on irritation; the blood in the vessels around it was very dark. The lungs were slightly congested. The muscles were not rigid, as those in the animal in the last experiment.

For convenience of reference we have arranged the results of these experiments in the following table:—

No. of Experiment.	Weight of Animal.	Quantity of Liquor Strychniæ.		Quantity of Hydrocyanic Acid.		TIME OF DEATH.		REMARKS.
		lbs.	Min.	Min.	'	"	'	
I.	...	60	20	...	0	30	...	No convulsion.
II.	...	60	2	30	...	Convulsion 1' after injection.
III.	...	30	15	...	7	0	...	Convulsion in 15".
IV.	...	30	2	0	...	Convulsion in 45".
V.	8	15	10	0	15 30	} Convulsion began in 2'; tetanus complete in 8'. Artificial respiration.
VI.	5½	15	10	...	2	0	18 0	
VII.	3	15	5	...	2	0	12 0	} Partial paralysis in 1'; convulsions in 1' 30", but slightly. Artificial respiration.
VIII.	4	15	5	...	10	0	12 0	
IX.	6½	10	28	0	28 0	Convulsion began in 2', complete in 4'.
X.	5	10	5	...	8	0	12 0	Convulsion in 2'. Artificial respiration.

From an examination of this table it appears that in Experiment I. death occurred from the effect of the hydrocyanic acid before the strychnia had begun to act. In Experiment III. the acid seemed to lessen the action of the strychnia, as death did not occur until seven minutes after the injection, although the animal was affected by the strychnia sooner than in Experiment IV., where death occurred in two minutes. In Experiment IX., however, where a smaller dose of strychnia alone was given, death did not occur nearly so rapidly as in Experiment X., where a similar dose was given along with hydrocyanic acid. In Experiment X. the dose of hydrocyanic acid was not sufficient fully to counteract the effect of the strychnia, although it greatly modified it.

From these experiments we conclude that although hydrocyanic acid may somewhat lessen the tetanic convulsions produced by strychnia, it cannot be employed as an antidote to that poison with any hope of success.

SECTION IV.

PRELIMINARY NOTES ON THE PHYSIOLOGICAL ACTION OF NITRO-GLYCERINE.

By J. Lauder Brunton and E. S. Tait.

From the observations and experiments on the physiological action and therapeutic employment of nitro-glycerine, made by numerous authorities, amongst whom may be mentioned Hering, Pelikan, Field, Thorowgood, Brady, Demme, Albers, Onsum, Eulenburg, Werber, and others, it is evident that nitro-glycerine is a powerful poison, and exerts a marked action on the nervous system when given even in exceedingly minute doses. Although many facts regarding its action have already been ascertained, it has not yet been made the subject of an elaborate investigation, and it therefore seemed to us advisable to ascertain its action more thoroughly than has yet been done. Our research is still very imperfect, but circumstances having obliged us to discontinue it for a few months, we now give the results we have already obtained, and trust to fill up the numerous deficiencies in them when we are again able to resume work together. While we confine ourselves in our present paper to a statement of the results of our own experiments, we purpose in a future one to enter into the literature of the subject, and to compare the conclusions to which our experiments have led us with those of previous observers.

General action on frogs.—A number of experiments were made by injecting a 10-per-cent. solution of nitro-glycerine in alcohol, in quantities varying from about one-tenth to four-tenths of a cubic centimetre, under the skin of the back or belly of a frog. The result was in all cases nearly the same. Immediately after the injection the animals became very restless, and the respirations became very rapid. In a minute or two the restlessness subsided and gave place to lethargy, the frogs showing a great disinclination to move, and allowing themselves to be gently pushed along the table without jumping. The respiration still continued rapid. In about two minutes more (generally three to five minutes after the injection of the poison) the frogs gave a sudden spring, and fell into tetanic convulsions.

These lasted about half a minute, and then became more or less relaxed; they soon returned, however, and continued to do so at nearly regular intervals, when the frogs were left alone, but they might also be brought on by touching the animals. When the relaxation of the muscles was imperfect, so that the legs still remained extended during the intervals, the convulsions were marked by twitchings of the toes. In some instances the mouth seemed to be the part first affected by the convulsion, as the jaws were seen to open and shut, although it is possible that this was connected with respiration rather than with the general convulsions; next the arms were affected, and lastly the legs. The arms seemed also to be more sensitive than the legs, as slight spasmodic twitches could sometimes be produced by touching or pinching the arms, when similar irritation of the legs had no effect. After continuing for some time, the convulsions became gradually weaker, and the animal died.

General action on cats.—The only warm-blooded animals on which we have as yet experimented with nitro-glycerine are cats, and although it is probable that a general similarity exists between its action on these and on other mammalia, yet it is not unlikely that there are minor differences which can only be ascertained by farther experiments. On injecting 4 cubic centimetres of a 10-per-cent. solution into the peritoneal cavity of a cat, the first symptom noticed two minutes after the injection was a stretching movement of the hind leg, as if the animal were trying to shake something off the foot. In about half an hour the cat cried as if in uneasiness or pain, and then vomited. In about half an hour more the legs seemed to fail during walking, and the animal suddenly sank down and never again rose. Vomiting again occurred once or twice, the respiration became exceedingly rapid (120 per minute), and the tongue and muscles of the lower jaw worked at each inspiration, so that the tongue lolled back and forwards like that of a dog which has been running. This action we have never observed in cats, either during health or after the administration of any other poison. The nostrils also moved with the respirations, and muscular twitching was observed over the body. The cornea was now found to be insensible, and pinching called forth no action in the limbs; but when the tail was pinched, a deep inspiration took place. Slight spasms resembling hiccough now occurred and in five minutes more the animal died, two hours and five minutes after the injection of the poison.

A large dose (10 cubic centimetres) injected in the same way almost immediately caused the respiration to become rapid (120 per minute) and the gait staggering. The animal also cried,

the tongue lolled out in the manner already described, and the third eyelids were drawn half over the eyes in the same manner as we have seen them in other cats after division of the vagi. In five minutes the respirations had reached 160 per minute, and the animal lay quite quiet. In fifteen minutes voluntary motion was quite paralysed, and reflex almost entirely so. When either the fore or hind legs were drawn out, no attempt was made to draw them up to the body, the limbs seeming quite paralysed. On tickling the inside of the ear, however, the ear was moved; and on touching the cornea, the eyelids closed.

On applying a strong induced current twenty minutes after the injection to various parts of the body and legs, muscular twitchings were produced, but no reflex movements.

In five minutes more, respirations became slow and gasping (6 per minute); the tongue ceased to loll, and in five minutes more the animal was dead, thirty minutes after the injection.

On post-mortem examination, the heart was found still pulsating, and the blood of a somewhat chocolate colour.

The principal effects produced by nitro-glycerine are thus seen to be—great acceleration of the respiration, paralysis, loss of reflex action, and apparently to a great degree of sensation, and death from stoppage of the respiration. The minor symptoms are muscular twitching and vomiting.

Action on the nervous system.—In frogs nitro-glycerine produces, as we have already mentioned, languor, tetanus, and finally paralysis. In cats there is paralysis without any tetanus, although there may be movements of a convulsive nature—such as vomiting, spasmodic respirations like hiccough, and muscular twitches—when the poison is injected into the abdominal cavity. In another experiment we found that after the injection of 1 cubic centimetre of a 10-per-cent. solution directly into the jugular vein of a cat, tetanic convulsions occurred.

In order to ascertain whether the tetanus in the frog is due to the action of the nitro-glycerine on the spinal cord, or on the nervous centres within the encephalon, the spinal cord was cut across about the middle before the poison was given. The upper part of the animal immediately became very restless, and the arms were stretched out at right angles to the body with the toes outspread. There was no alteration in the hinder part of the body and legs. The nitro-glycerine therefore does not cause tetanus by its direct action on the spinal cord, as otherwise spasms would have been observed in the hind legs. This result was confirmed by another experiment. A frog was decapitated, and after the spinal cord had recovered from the shock, and reflex movements were again observed in the limbs, nitro-glycerine

was injected under the skin. No spasm whatever was observed. Other experiments lead us to believe that the tetanus is not due to any action on the cerebral lobes, but probably to the effect of the poison on the optic lobes; but we are not yet in a position to decide this with certainty.

Action on muscle.—In order to ascertain this, two gastrocnemii of a frog were immersed in two glasses, each containing 10 cubic centimetres of a .75-per-cent. solution of common salt. To the one glass about two drops of the solution of nitro-glycerine were added. After three hours the muscle which had been lying in the pure solution of salt contracted readily on the application of an induced current, while the one which had been lying in the salt solution with nitro-glycerine was in a state of rigor mortis. Nitro-glycerine is therefore a muscle-poison, and in this particular its action agrees with that of nitrites, all of which have been found to be muscle-poisons in an unpublished research on which one of us (Brunton) in conjunction with Mr. Gresswell is at present engaged in this laboratory.

Action on motor nerves.—On ligaturing the vessels in one leg of a frog so as to prevent the circulation of poisoned blood in that limb, the nerve being left uninjured, we have found that when paralysis had begun to appear, the spasms which still could be observed were slightly more marked in the ligatured limb. On testing the irritability of the motor nerves after death, they were found to respond much more readily to an induced current in the ligatured than in the non-ligatured leg; but as the muscles of the non-ligatured leg responded but feebly to a current directly applied to them, we are at present unable to say whether the paralysis is entirely due to the action of the poison on the muscles, or whether it affects the motor nerves as well. We may possibly be able to decide this point by making farther experiments, similar to those which we have already performed, but in winter, when the muscles preserve their irritability longer than in summer, during which our present experiments were made. We also propose to repeat them with *Rana esculenta* instead of *Rana temporaria*, the muscles of these two species of frog having been shown by Schmiedeberg to be very differently affected by caffeine, a poison having an action similar in some respects to that of nitro-glycerine.

Action on the spinal cord.—The loss of reflex action both in frogs and cats, in the advanced stages of poisoning, indicates that the cord is paralysed; and the persistence of reflex action in parts supplied by cranial nerves, such as the eye and ear, after it has disappeared from other parts of the body, indicates that the cord is paralysed before the ganglia at the base of the brain.

Action on the brain.—One of the most remarkable effects of nitro-glycerine is the intense headache it produces even in infinitesimal doses. Almost all observers agree about the fact of its producing headache, but they differ regarding the nature of the headache. According to our experience, it is not always of the same kind, being sometimes frontal, sometimes occipital, sometimes affecting one side only, and at other times the whole head. In one of us (Brunton) it was several times accompanied by vomiting. It has been said by some that continued use of nitro-glycerine makes the person more sensitive, but in one of us (Tait) the contrary seemed to be the case, as the headache was only suffered from during the first week of the investigation. None of the poison was taken by the mouth, and as it is non-volatile, the amount taken in by the lungs must have been infinitesimal. It is possible that, as some writers have supposed, a little of it was absorbed by the skin, but the quantity thus taken into the system must have been excessively minute.

Action on the heart.—When the excised heart of a frog is put into 10 cubic centimetres of a .75-per-cent. salt solution, and two drops of a 10-per-cent. solution of nitro-glycerine in alcohol are added, the heart begins to beat more and more slowly, and gradually ceases altogether. A similar quantity of alcohol added to the same amount of salt solution had no action on a heart immersed in it. In one instance after the addition of nitro-glycerine, we observed a slight quickening before the beats became slow.

Two cubic centimetres of a 10-per-cent. solution of nitro-glycerine in alcohol injected into the jugular vein of a cat stopped the cardiac pulsations entirely in thirteen seconds. One cubic centimetre in another experiment greatly quickened the pulse. The power of the vagus over the heart appears to be diminished, as irritation of its trunk had less effect upon the heart after injection than before.

Action on the blood-pressure.—Nitro-glycerine diminishes the blood-pressure considerably, but its power to do so is very much less than that of nitrite of amyl.

Action on the blood.—The blood of animals poisoned by nitro-glycerine is of a chocolate colour even in the arteries. When blood is shaken up with nitro-glycerine solution, it acquires a chocolate colour, though slowly. In this respect nitro-glycerine agrees with nitrites, which also cause the blood to assume that colour.

With the spectroscope at our disposal, however, we were unable to discern any difference between the spectrum of blood from an animal poisoned with nitro-glycerine, or of normal blood shaken

up with it, and normal blood, either before or after the addition of a reducing fluid. If our observation were correct, this would constitute an important difference between nitro-glycerine-blood and nitrite-blood as described by Gamgee; but the strong similarity in colour between the two kinds of blood makes us doubtful about the correctness of our spectroscopic observation, and we hope to repeat it as soon as opportunity allows with a better instrument.

Action on oxidation.—Certain vegetable substances have the power of oxidising tincture of guaiac, and causing it to become blue. In order to ascertain whether nitro-glycerine had any power to diminish or prevent this oxidising process, a potato was pounded with water, the liquid strained off and mixed with tincture of guaiac and a small quantity of nitro-glycerine. Instead of preventing oxidation, however, it rather seemed to quicken it, the mixture assuming a blue colour more quickly and more intensely than where no nitro-glycerine was added. In this respect also its action resembles that of nitrites as described by Binz and Pick.

From our experiments it would then appear that nitro-glycerine agrees with nitrites in not lessening the oxidation of guaiac by vegetable solutions, in causing the blood of animals poisoned by it to become of a chocolate colour, in acting as a muscular poison, and in diminishing blood-pressure. Its action in this last respect is, however, much less than that of nitrite of amyl. In a future paper we hope to give a more detailed and complete account of the action of nitro-glycerine, and of the resemblances and differences between its action and that of nitrites, as well as a discussion of its possible use in medicine.

SECTION V.

ON THE EMETIC ACTION OF SULPHATE OF COPPER WHEN INJECTED INTO THE VEINS.

By T. Lauder Brunton and E. de Lancy West.

While the emetic action of tartarated antimony injected into the veins is universally acknowledged, the possession of a similar action by sulphate of copper has been the subject of dispute. When introduced directly into the stomach, sulphate of copper produces vomiting with even greater rapidity and certainty than tartarated antimony; but while some experimenters have observed emesis after its injection directly into the circulation, others have been unable to perceive any such result. It seemed

to us that one cause of the discrepancy between the observations of previous experimenters might be due to the solution of sulphate of copper forming a coagulum with the blood in the vein into which it was injected, and thus never reaching those parts of the nervous system which are concerned in the production of emesis. For the purpose both of avoiding this source of fallacy and of imitating the conditions in which sulphate of copper would be absorbed after its introduction into the stomach, we injected a neutral solution of albuminate of copper—or perhaps it would be more correct to say of cupric peptones—instead of a solution of the sulphate. This solution was made by adding sulphate of copper to white of egg and digesting the light blue coagulum thus formed with pepsin and dilute hydrochloric acid, or with pancreatin, until it was nearly dissolved. The solution was then filtered—neutralised, if necessary—and injected into the jugular vein. In order to discover whether the copper was carried by the blood to the walls of the stomach, and there acted as an irritant in somewhat the same way as if it had been swallowed, the stomach was afterwards tested for the metal.

Experiment I.

Albuminate of copper was digested with pepsin and dilute hydrochloric acid at 40° C. for twenty-four hours, filtered, and rendered very slightly alkaline with caustic soda. A cat was chloroformed and a cannula inserted into the jugular vein. When the animal had completely recovered from the anæsthetic, 10 cubic centimetres of the cupric solution were injected into the vein.

In seven minutes after the first injection, 15 cubic centimetres more were injected. There was now salivation, and the animal licked its lips. This appears to be a sign of nausea in cats.

In twenty-seven minutes, strings of viscid saliva hung from the mouth.

In thirty minutes, 20 cubic centimetres more were injected. The animal micturated and defecated.

In thirty-three minutes, respiration irregular, 90 per minute.

In thirty-four minutes, respiration irregular, 58 per minute.

In forty-nine minutes, violent retching.

In fifty-six minutes, the animal was now killed as it suffered a good deal from the retching, and the emetic action of the sulphate of copper seemed clear.

On post-mortem examination the lungs were found to be œdematous. The auricles were pulsating feebly. The animal was pregnant.

The stomach was digested with strong hydrochloric acid until

it was completely disorganised, the solution filtered, and tested for the presence of copper with sulphuretted hydrogen, ammonia, and ferrocyanide of potassium. These reagents all gave a negative result.

Experiment II.

This experiment was made like the preceding one, but the solution of cupric peptone was obtained by digestion with pancreatin and water instead of with pepsin and hydrochloric acid. The animal had a full meal four hours before the operation, and about three-quarters of an hour were allowed to elapse between the introduction of the cannula and the injection of the copper, in order to allow the effects of the chloroform to pass off.

Injected 13 cubic centimetres of the neutral solution into the jugular vein.

Six minutes after injection, vomiting. This seemed easy, and the vomited matters were solid.

Twenty minutes.—Retching.

Twenty-two minutes.—Violently sick.

Twenty-five minutes.—Died.

On post-mortem examination the auricles were vibrating rather than pulsating. There was peristaltic movement of the intestines.

On analysis of the stomach, as in the previous experiment, no traces of copper were found.

From these two experiments it seems nearly certain that cupric peptones produce vomiting when injected into the veins. It is true that vomiting not unfrequently occurs in cats recovering from the effects of chloroform; but this generally takes place, as far as our observation goes, during the process of recovery, and a sufficiently long time was allowed between the recovery from the anæsthetic and the injection of the copper to render it improbable that the vomiting which followed so closely on the injection of the copper was due to the chloroform.

Notwithstanding our failure to find copper in the walls of the stomach, it was still possible that the metal acted as an emetic by irritating the sensory nerves of the stomach or intestines, and thus stimulating the vomiting centre in the medulla oblongata reflexly, instead of acting directly on the centre itself.

For the purpose of ascertaining this we divided the sensory nerves passing up from the stomach and intestines—viz., the vagi and splanchnics.

Experiment III.

About four hours after the animal had had a full meal a

cannula was inserted into the jugular vein of a cat, as in the previous experiments. Both vagi were cut in the neck.

In five minutes after the operation the animal began to recover from the chloroform. Dyspnœa.

1.5.—Salivation. Strings of viscid saliva hang about the mouth. Respiration slow and laboured.

1.24.—Tried to inject some cupric peptones made by digestion with pancreatin, but owing to some obstruction in the cannula no fluid entered the vein.

The cat was again chloroformed and an attempt made to remove the obstruction.

1.30.—Retching. Animal recovering from chloroform.

1.38.—Do.

2.7.—The animal having now completely recovered from the chloroform, another attempt to inject cupric peptones was made, but it also failed.

2.13.—Respiration, 12 per minute.

2.17.—Salivation.

2.45.—Animal again chloroformed, and another cannula inserted.

3.5.—The animal had perfectly recovered from chloroform for about an hour. No retching had occurred. 13 cubic centimetres of the solution of cupric peptones were now injected. Almost immediately violent retching occurred.

3.16.—Death.

On post-mortem examination the stomach was found to be full.

Experiment IV.

A full meal had been given to the animal used in the experiment twenty-four hours before. A cannula was placed in the jugular vein, and the vagi cut as before.

One hour after the operation 7 cubic centimetres of a solution of cupric peptones made by digestion with pancreatin were injected into the jugular vein.

1.5.—Retching violently. No salivation or licking of lips, but mouth very frothy.

1.15.—Respiration, 8 per minute.

1.50.—Violent retching.

2.40.—Animal killed by hydrocyanic acid.

On post-mortem examination the animal was found to be pregnant. The stomach was empty. On adding some of the cupric peptones to the blood, it did not cause coagulation.

From these experiments it appears that section of the vagi does not prevent the cupric peptones from causing violent

retching either when the animal is fasting or fed, but it appears to prevent the evacuation of the stomach. Experiment III. seems to confirm Schiff's view, that vomiting occurs only when the cardiac extremity of the stomach becomes relaxed at the same moment that the expulsive motions of retching occur, and that when the vagi are cut, the co-ordination between the cardia and abdominal muscles is disturbed, so that the cardiac sphincter or lower end of œsophagus does not relax when the abdominal muscles contract, and thus the stomach is not emptied, notwithstanding the violent retching.

Experiment V.

About four hours after a full meal a cannula was inserted into the jugular vein of a cat, and both vagi were cut in the neck and the splanchnics in the abdomen. An hour and a half afterwards 20 cubic centimetres of the pancreatic cupric peptones were injected into the jugular.

In five minutes after the injection the animal died without any sign of retching.

On post-mortem examination the heart had ceased to pulsate, and clots were found in both auricles. The kidneys were anæmic and the spleen very dark in colour.

In this experiment the dose was obviously too large and killed the animal by stopping the heart.

Experiment VI.

Three hours after a full meal a cannula was inserted into the jugular vein of a cat, and the vagi and splanchnics cut as in previous experiments.

An hour afterwards 17 cubic centimetres of pancreatic cupric peptones were injected into the jugular vein.

In twenty-five minutes the animal died without any sign of retching.

On post-mortem examination the heart was found pulsating. The spleen was black.

From the foregoing experiments we conclude—

1. That cupric peptones injected into the blood cause retching and vomiting.
2. That previous section of the vagi does not prevent the retching, but interferes with the evacuation of the stomach.
3. That section of the splanchnics appears to prevent retching, but the number of our experiments is too small to decide this with any degree of certainty.
4. The absence of vomiting after section of the splanchnics indicates that copper probably exerts an emetic action by irritating

the stomach or intestines, and thus acting reflexly on the vomiting centre in the medulla oblongata, rather than by its direct action on the medulla itself.

5. We have been unable to detect copper in the walls of the stomach.

6. The conversion of metallic salts into peptones seems to offer a convenient method of introducing them directly into the circulation without producing coagula within the vessels or heart.

SECTION VI.

ON THE INFLUENCE OF QUININE AND SULPHURIC ACID UPON REFLEX ACTION.

By T. Lauder Brunton and George L. Pardington.

It was at one time supposed that diminution in reflex action indicated exclusively a diminution of the excitability of the spinal cord, so that if a frog no longer drew up its leg when its foot was irritated, its spinal cord was supposed to be more or less paralysed. In 1863, however, Setschenow showed that this view was erroneous, and that reflex excitability might appear to be nearly destroyed, while in reality the functions of the cord remained unimpaired. He found that the optic lobes in the frog, corresponding to the corpora quadrigemina in man, have the power of inhibiting reflex action; so that when they are stimulated by the application of common salt, or of an electrical current, and are thus made to act more strongly than usual, reflex action becomes slighter and slighter, and may cease altogether. When, on the contrary, their restraining influence over the cord is destroyed by cutting it across just below the medulla oblongata, reflex action becomes more vigorous than usual. The best method of estimating the activity of the spinal cord is that of Türck, and it was the one employed by us. It consists in dipping the foot of a frog into very dilute sulphuric acid, and by means of a metronome counting the number of seconds which elapse before the leg is drawn up. The less the reflex power of the cord, the longer is the time, and *vice versa*. In order to abolish sensation and voluntary motion, the cerebral hemispheres are divided across either by a single quick cut with a sharp knife through the bones of the head, or, what is preferable, by chloroforming the frog, removing the bones of the anterior part of the head along with the greatest part of the cerebral hemispheres, and then allowing the animal to recover from the anæsthetic.

The optic lobes, or that part of them which restrains reflex action, usually called, from its discoverer, Setschenow's centre, may be called into action not merely by irritants directly applied to it, but also reflexly by irritation applied to a sensory nerve, as has been shown by Nothnagel. It may also be excited by drugs injected into the body. When this centre is excited—whether by direct irritation, by reflex irritation, or by the action of drugs—the reflex excitability of the spinal cord of the frog is greatly diminished, and many seconds elapse before the foot is withdrawn from the acid. After dividing the medulla, however, and allowing a little time to elapse for the shock of the operation (whatever that may be) to pass off, it is usually found that the excitability is greatly increased, and may even rise above the normal. If the loss of excitability after the injection of a drug is due to the action of the drug on the cord itself, division of the medulla of course does not increase the reflex excitability.

The effect of quinine upon reflex action was investigated several years ago by Chaperon, who found that after injecting a solution of quinine under the skin of the back of a frog, the reflex excitability became very greatly diminished by irritation of Setschenow's centre, the diminution disappearing whenever the cord was divided close to the occiput. The stimulation of Setschenow's centre, which was thus shown to be caused by the injection of quinine under the skin of the frog's back, was attributed by Chaperon to the direct action of the quinine upon these centres after its absorption into the blood, and by its means he attempted to explain the useful effect of quinine in ague. Pfüger's theory of ague is that the malarious poison causes the spleen or liver to become enlarged, and that these organs consequently become a source of constant irritation to the sympathetic nerves with which they are supplied. This constant nervous irritation is conducted to the vasomotor centre, but instead of producing a reflex action at once, it goes on accumulating until at length it makes itself evident in a spasm of the peripheral vessels, and the other symptoms which constitute a fit of ague. The reflex spasm of the vessels is supposed by Chaperon to be arrested by the inhibitory action produced by quinine through Setschenow's centres, and thus the fit of ague prevented. It is unnecessary for us to criticise this explanation, for unless Chaperon's explanation of the action of quinine on the nervous system be correct, his explanation of its use in ague at once falls to the ground. Lately the effect of quinine upon reflex action has been made the subject of an investigation by Heubach in the laboratory of Professor Binz of Bonn, and the results arrived at in it were that quinine does lessen reflex action. Binz and

Heubach assert, however, that this diminution is not due to stimulation of Setschenow's centres, but only to the quinine weakening or stopping the heart, and thus stopping the circulation in the spinal cord, which loses its reflex power in consequence of the failure in its blood-supply.

Some years ago, however, one of us (Brunton) repeated Chaperon's experiments, and found them to be correct. In one experiment the time required for reflex action (by Türck's method) before the injection of quinine was ten seconds, after the injection 120 seconds, and after division of the cord only eight seconds. Such a result was certainly due to stimulation of Setschenow's centres, and could not possibly have been obtained had the effect of the injection of quinine been exerted merely upon the heart, and through it on the spinal cord. The experiment generally appeared to be more successful when the solution of sulphate of quinine was strong, and dissolved in water by the aid of a liberal allowance of sulphuric acid. It seemed therefore possible that the irritation of Setschenow's centres, and the consequent inhibition of reflex action, produced by the injection of quinine under the skin, might not be due to the action of the quinine itself upon these centres, but to reflex irritation. The sulphuric acid and quinine injected under the skin of the back might stimulate these centres reflexly, and diminish the reflex action which would usually occur on irritation of the foot in much the same way as Nothnagel found that irritation of one sciatic lessened the reflex which would usually have followed irritation of the other; and as Goltz had found that irritation of the sciatic nerve prevented the usual consequences of irritation of the abdominal sympathetic. In order to ascertain whether this hypothesis was correct or not, four sets of experiments were made. In the first of these, crystalline hydrochlorate of quinine, partially dissolved and partially suspended in water, was injected under the skin of the back; in the second, amorphous hydrochlorate of quinine was injected in the same way; in the third, a solution of sulphate of quinine in water and sulphuric acid was employed; and in the fourth, dilute sulphuric acid alone. The hydrochlorate of quinine was employed instead of the sulphate, as being more soluble, and thus likely to be more quickly absorbed, and to develop the specific action of quinine upon the heart and nervous system more rapidly than the sulphate. The crystalline hydrochlorate is, however, sparingly soluble; the amorphous hydrochlorate is exceedingly soluble, dissolving in about its own weight of water.

In all the experiments the time allowed to elapse between each operation and the application of each stimulus was fifteen

minutes, except in those instances where an exception is noted. Wherever a 0 is found in the tables it indicates that no reflex was observed during 120 seconds. The right and left foot of each frog was tested separately immediately after each other. The letters R and L indicate the times which elapsed before reflex action took place in the right and left foot respectively.

TABLE I.—EFFECTS OF INJECTION OF CRYSTALLINE HYDROCHLORATE OF QUININE.

Frog A.		Frog B.		Frog C.		Frog D.		Frog E.		Frog F.		Remarks.
R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	
2	4	5	3	2	2	2	3	2	2	1	1	After removal of the anterior half of the cerebral lobes.
3	5	6	0	2	2	3	4	2	2	1	2	
5	7	6	0	2	3	2	3	2	2	1	1	
5	6	50	0	2	2	6	3	3	3	2	4	After injection of 1 milligramme of crystalline hydrochlorate of quinine.
5	6	64	0	2	2	6	3	2	3	1	2	
6	12	0	0	2	2	6	3	3	3	2	3	
...	...	4	3	1	1	2	2	2	2	2	2	After division of the cord at the occiput.
0	0	0	0	3	3	1	2	2	2	2	2	
...	...	2	3	6	0	2	2	2	2	2	2	

TABLE II.—EFFECTS OF INJECTION OF AMORPHOUS HYDROCHLORATE OF QUININE.

Frog G.		Frog H.		Remarks.
R.	L.	R.	L.	
1	2	1	1	After removal of the anterior part of the cerebral lobes.
2	2	1	2	
4	18	1	2	
0	0	0	0	After injection of '05 gramme of <i>amorphous hydrochlorate of quinia</i> . These frogs all died immediately after the injection, in a state of intense tetanic spasm.
0	0	0	0	
0	0	0	0	After division of the cord at the occiput.

Frog I.		Frog K.		Frog L.		Frog M.		Frog N.		Remarks.
R.	L.	R.	L.	R.	L.	R.	L.	R.	L.	
4	3	1	2	1	1	7	10	1	1	After division of the anterior lobes of the cerebrum.
3	3	2	2	1	2	11	8	1	2	
3	4	2	2	1	2	16	8	2	3	
0	0	0	0	0	0	0	0	0	0	After injection of $\frac{1}{4}$ decigramme of <i>amorphous hydrochlorate of quinia</i> . These frogs died in a state of tetanus, with the exception of I and M., which showed some signs of life when the second stimulus was applied; but they soon succumbed. It will be observed that their reflex was lower than the others.
0	0	0	0	0	0	0	0	0	0	
0	0	0	0	0	0	0	0	0	0	After division of the cord at the occiput.

TABLE II.—Continued.

Frog O.		Frog P.		Frog Q.		Frog R.		Frog S.		Remarks.
R. L.	R. L.	R. L.	R. L.	R. L.	R. L.	R. L.	R. L.	R. L.		
1 1	1 1	1 1	1 1	0 3	1 1	After division of the anterior part of the cerebral lobes.				
1 2	3 5	2 3	0 3	1 1						
1 2	3 4	4 6	0 0	1 1						
1 1	3 3	3 3	46 48	0 0	After injection of $\frac{1}{8}$ decigramme of <i>amorphous hydrochlorate of quinia</i> . These frogs all had tetanus after the stimulus had been applied, except R and S, which had tetanus before the stimulus was applied.					
...	48 48	...						
...	48 48	...						
0 0	0 0	0 0	0 0	0 0	After division of the cord at the occiput.					

TABLE III.—EFFECTS OF INJECTION OF SULPHATE OF QUININE WITH SULPHURIC ACID.

Frog T.		Frog U.		Frog V.		Frog W.		Frog X.		Remarks.
R. L.	R. L.	R. L.	R. L.	R. L.	R. L.	R. L.	R. L.	R. L.		
2 2	6 4	6 4	3 3	5 4	After division of the anterior lobes of the cerebrum.					
2 2	2 2	5 3	4 6	3 5						
2 2	3 3	7 4	3 3	6 6						
0 0	0 0	15 0	0 12	0 0	After injection of 4 or 5 minims of <i>solutions of acid and quinia</i> —viz., quiniæ sulph., gr. ss.; acid. sulph. dil., cc. v. (gr. x. to $\bar{3}$ i.)					
...	...	0 93	8 0	...						
0 0	0 0	0 0	0 0	0 0	No tetanus in any of these.					

TABLE IV.—EFFECT OF INJECTION OF DILUTE SULPHURIC ACID.

Expt. 1.		Expt. 2.		Expt. 3.		Expt. 4.		Expt. 5.		Remarks.
R. L.	R. L.	R. L.	R. L.	R. L.	R. L.	R. L.	R. L.	R. L.		
1 1	1 4	1 1	1 1	1 1	1 2	After division of the anterior part of the cerebral lobes.				
3 2	10 10	2 4	1 1	8 0						
3 2	34 7	6 5	3 4	8 0						
3 2	24 8	8 14	12 2	0 12	After injection of 4 or 5 minims of sol. of acid. sulph., m. x.; aquæ ad $\bar{3}$ i.					
3 3	13 23	34 12	34 14	0 28						
4 4	40 18	80 34	63 41	0 102						
3 2	c 0	0 0	0 0	0 0	These frogs showed decided reflex action in grasping the finger; but did not respond to the acid stimulus after division of the cord at the occiput.					
3 4						
3 2						

TABLE IV.—Continued.

Expt. 6.		Remarks.	Expt. 7.		Remarks.	
R.	L.	Without division of cerebral anterior lobes.	R.	L.	After division of cord at occiput.	
3	4		1	1		
3	3		1	1		
3	3		2	2		
4 ¹ 3 ¹	43	After injection of acid. <i>Vide supra.</i>	3	3	Injection of acid as above.	
8	39		40	3		3
14	72		0	4		3
0	0	After division of the cord at the occiput.				

¹ The figures 4, 8, 14 refer to the minutes after the injection at which stimulus was applied.

An examination of the preceding tables shows that crystallised hydrochlorate of quinine, as might have been expected from its sparing solubility, has a comparatively slight action, and only in one case (frog B) did it greatly diminish reflex action. The soluble amorphous hydrochlorate has a most powerful action, causing in large doses tetanic spasm and death. The sulphate of quinine with sulphuric acid caused great diminution or complete abolition of reflex action, but no tetanus. The dilute acid alone also caused great diminution of reflex action—not quite so great as that occasioned by the sulphate of quinine dissolved in acid, but, like it, without tetanus. Reflex action was observed in the greater part of the frogs treated with the small dose of crystallised hydrochlorate of quinine, and in one (frog I) with dilute acid alone.

In all those cases, however, the reflex action had not been affected by the injection; and in the other experiments, where the reflex had been diminished by the injection, division of the cord at the occiput, instead of increasing it again by removal of the inhibitory action of the optic lobes, simply destroyed it entirely. The injection of dilute sulphuric acid after division of the cord below the occiput (Experiment 7) had very little effect upon reflex action. This single experiment of course requires repetition, but we have been prevented from repeating it by our inability to obtain frogs. It indicates, however, that the diminution is effected by reflex action through the nervous centres in the head.

A CASE
OF
WOUND OF THE FEMORAL VESSELS
RESULTING IN
A PERMANENT COMMUNICATION BETWEEN
THE ARTERY AND VEIN.

BY
THOMAS SMITH.

On the 9th of December 1875, a boy, aged 14, ran a sharp-pointed penknife into the anterior and inner aspect of the thigh, about midway between Poupart's ligament and the knee. There was but little bleeding at the time of the accident, but the boy was taken into the Sanatorium at Marlborough, where he remained for four days. On his leaving the Sanatorium there was still a little discharge from the wound. The very day he left, he slipped and fell, and 'sprained' his thigh badly; he was taken back to the Sanatorium with pain in the thigh and swelling about the seat of the wound.

On December the 18th he was brought to London and placed under the care of Mr. Robert Brown of Brixton, with whom I saw the boy, and as it was our opinion that the femoral artery was wounded, he was admitted into St. Bartholomew's Hospital on December the 29th.

The patient was a fair-skinned well-nourished boy, of rather delicate appearance. He lay in bed with his thigh abducted and his knee slightly flexed, in which position it did not give him any real pain, but any attempt to straighten the knee caused decided pain.

The left thigh at the centre was a quarter of an inch larger in circumference than the right; it was irregularly ecchymosed

towards the inner side, and there was a small red cicatrix just outside the femoral artery, as nearly as possible in the middle of the length of the thigh.

About the situation of the healed puncture the tissues of the limb felt firmer and thicker than natural, but there was no well-defined or circumscribed swelling.

The open hand being laid on the limb, a peculiar purring thrill was imparted to it; this was most distinct at the inner side of the scar, though it could be felt from Poupart's ligament to the termination of the course of the femoral artery—that is, an inch or two lower down than the scar.

On auscultation a murmur could be heard from Poupart's ligament to the knee, and all over the anterior and inner aspect of the thigh; this murmur consisted of two parts—a continuous hum, and a very high-pitched musical systolic murmur. The purring thrill and the murmurs could at once be put an end to by pressure on the femoral artery at any point from its commencement to just below the situation of the puncture in the skin. There was no appearance of venous distension in the limb; the foot and leg were slightly œdematous. There was no difference in the temperature of the two limbs.

On December the 30th a consultation was held, and we were unanimously of opinion that the case was one in which there was a communication between the femoral artery and vein. It was agreed to apply pressure to the whole limb by firm bandaging, and that the recumbent position should be strictly maintained. This treatment was carefully carried out, a flannel roller being employed for the purpose of compressing the limb.

January 6.—The murmur is heard as before. No difference in the size of the limbs, and no œdema of the foot and ankle. The thrill is less plainly to be felt; the murmur is very loud.

January 10.—The thrill has disappeared; the bruits are to be heard, but much fainter than heretofore.

January 14.—The thrill can be felt distinctly 2 inches to the inner side of the scar, and is only felt when light pressure is made. The bruit is now heard only just about the scar, and faintly along the vessels down to the knee and up to Poupart's ligament.

It occasionally resembles the murmurs heard formerly; but now there is seldom heard anything but the systolic bruit, the continuous hum being usually absent.

January 24.—Thrill still felt at the old place, about two inches inside the scar, and nowhere else. The bruits still are heard—about the scar, two; at knee, at Poupart's ligament, and on front and outer side of thigh, one only, the continuous hum.

To get up and to wear an elastic stocking reaching to top of thigh.

January 25.—Discharged

Was seen again by Mr. Smith on 4th or 5th February, who made the following note:—

‘No visible pulsation.

‘No pulsation or thrill perceptible to touch.

‘A musical windy murmur like the wind through telegraph-wires all over front of thigh; loudest 2 inches above and below cicatrix, and fading away above and below this to Poupart’s ligament and internal condyle.

‘At one spot, an inch to inner side of scar, there is an ordinary blowing murmur, not a continuous hum.

‘Pressure down to scar arrests murmur, difficult to say if it does so completely by pressure below. Limb natural size. Can walk easily without lameness.’

He said that if he slept a night without the stocking, he could feel pulsation in the morning.

May 1, 1876.—The limb was of a natural size, being a little smaller in all its measurements than the opposite one. There was no appearance of obstructed cutaneous circulation. He could walk easily, at least three or four miles, without any inconvenience. He still wore the elastic stocking.

A blowing murmur was to be heard along the course of the superficial femoral, except just opposite the site of wound, which latter is situated somewhat external to the artery. This murmur is synchronous with the pulse, and is not a continuous sound.

There is a windy musical hum (a continuous sound) to be heard very plainly over a space about the size of the palm on the adductor aspect of the thigh, rather higher up than the situation of the external puncture. This sound fades away above and below; it is over the situation of the saphena vein, but the latter is not visible at all in the integuments.

I fancy I can hear the same sound faintly on the posterior saphena in the upper part of the calf.

The patient says that on one occasion he had a decided return of the thrill and purring feeling in the thigh, and that, as directed, he lay in bed a day or two.

May 28, 1876.—Dr. Fergus of Marlborough, under whose care the patient was at this time, kindly made the following note of the case:—

‘I found the limb perfectly natural in its aspect, without the least sign of any abnormal condition of the circulation. The murmur is very distinctly audible throughout the course of the

vein, from the inner condyle of the femur to Poupart's ligament. There is no murmur in the popliteal space, and the pulse is perfect in that situation. I cannot believe that the artery was ever wounded, and I attribute the murmur to something inside the vein.'

July 27, 1876.—I examined the boy. He had played at cricket during the whole summer term without inconvenience. He has not worn his elastic stocking for ten weeks. There is neither pain, congestion, swelling, or pulsation. The venous murmur is heard over a more limited space. There is a continuous hum 4 inches below Poupart's ligament, to the inner side of the femoral artery; the sound becomes more blowing lower down, and ends in a musical systolic murmur just where the femoral passes into the popliteal space.

Though the sequel of this case may appear to lend some countenance to Dr. Ferguson's opinion that the femoral artery was never wounded, yet, nevertheless, I believe that the femoral vessels were wounded at the time of the accident, and that even now a small communication exists between the femoral artery and femoral vein.

I first saw the patient, three or four days before his admission to the Hospital, with Mr. Robert Brown of Brixton. There was a good deal of fluid blood among the deep muscles of the thigh. There was well-marked pulsation, a loud bruit, and a thrilling purr was perceptible to the touch. The limb was painful, stiff, and swollen. We, neither of us, entertained any doubt that a large artery, probably the femoral, had been wounded.

The patient was carefully examined subsequently by nearly all the members of the surgical staff of the Hospital in consultation, and our opinion was unanimous, that the symptoms could be accounted for only on the supposition that a communication existed between the femoral artery and vein.

The relative position of the femoral vessels at the situation of the external wound, and the manner in which the wound was inflicted, must also be taken into consideration.

The vessels, at the point referred to, lie one behind the other in such close contact that the point of a penknife thrust between them might well wound both vessels without completely transfixing either. And that this really happened is the more probable from the fact that no aneurismal sac was formed, though there was evidence of an abnormal communication between the arterial and venous currents. Now, for such an accident to occur, the knife should have entered from one side or the other, and not from the front, and this was almost certainly the direction of the wound.

At the time the wound was inflicted, the boy was cutting a piece of loose leather from the heel of his boot; the knee was flexed, the left thigh abducted and rotated outwards, and the point of the knife passed in a direction from below upwards, and from without inwards.

It is to be observed, that notwithstanding the complete recovery of the function of the limb, and the absence of any visible venous congestion, the venous murmur and the systolic bruit still persist, though they are of less intensity, and are heard over a smaller area than formerly. This freedom from serious symptoms is in accordance with the experience of others who have recorded cases of aneurismal varix.

In Mr. Cock's case,¹ where the popliteal artery communicated with the popliteal vein, and where the wound was inflicted with a dinner-knife, so that probably the communication was a free one, it was not until two years after the accident that the superficial veins became dilated, and it was eleven years before surgical interference was required. And in discussing the same subject in a paper in the *Guy's Hospital Reports* for 1851, Mr. Bransby Cooper remarks, that while varicose aneurism increases daily, becomes worse and worse, and can only be relieved by a surgical operation, aneurismal varix soon arrives at a state which becomes permanent. The only treatment employed in this case was the maintenance of continuous and firm pressure by means of a flannel roller applied to the whole limb; though commonplace, yet it had the merit of simplicity, and it was successful.

One may well hope that the circulation of the limb may never at a future time become so embarrassed as to need surgical interference, since the performance of an operation to discover the exact seat of the communication between the artery and the vein with a view to ligature both vessels would be very hazardous. The absence of an aneurismal sac would necessitate an elaborate dissection, and would require the separation of the femoral artery from its vein to an uncertain extent in order to discover the orifice of communication between the vessels.

¹ Aneurismal dilatation of the posterior tibial vein, *Med.-Chir. Trans.*, 1851.

FATAL WOUND
OF
THE ASCENDING PHARYNGEAL ARTERY
BY A TOBACCO-PIPE.

BY
W. MORRANT BAKER.

A man, 23 years old, applied at St. Bartholomew's Hospital, September 19, 1875, on account of 'sore throat.' The case was supposed, from the patient's description of his symptoms, to be medical rather than surgical, and the house-physician on duty was called to see him; but as there appeared to be an abscess in the tonsil requiring puncture, the patient was subsequently transferred to the care of the house-surgeon, who admitted him into the Hospital.

The symptoms at this time were those of acute tonsillitis—much pain and difficulty in swallowing, much redness and swelling of the left tonsil, with inflammatory oedema extending to the soft palate and its neighbourhood. The tonsil was tense and elastic as if from the presence of fluid, with a prominent spot, which looked like the pointing of an abscess; and the house surgeon on duty, Mr. Hames, accordingly punctured it with the usual precautions. To his surprise, however, only blood escaped; and although the hæmorrhage was not large, he naturally thought he might have wounded some small vessel, and that the abscess had not matured as much as was believed. This opinion seemed to be confirmed by the escape of a small quantity of blood in the course of the afternoon.

At 9 P.M. on the same day the pulse was 112, and temperature $100\cdot4^{\circ}$; and it was remarked in the notes that there was less oedema of the fauces, and that the patient could both speak and swallow better.

September 20.—Some quantity of blood, mixed, it was said, with pus, was hawked up or vomited early this morning, and

thought at the time by the nurse to be due to the bursting of the abscess in the tonsil. Yesterday's improvement in speech and deglutition remains, and the patient says he feels better. Pulse, 108. Respirations, 20. Temperature, 100.5°.

I saw the patient on this day for the first time; and, on examining the throat, found nothing more than the usual appearances of an acutely-inflamed tonsil. The small puncture made by the house surgeon was visible, and apparently quite out of the way of any important blood-vessel.

At this time the patient told me he felt very much better in every way; he could speak and swallow fairly, and there seemed no reason to doubt that all was going on well, and that, as the nurse supposed, the tonsillar abscess had burst.

The history of the case, which had been carefully taken by the dresser, Mr Pye, was as follows: Two days preceding the patient's admission into the Hospital he fell down, drunk, in the street, and grazed his throat with the end of a tobacco-pipe which he happened to be smoking at the time. There was no bleeding, and the scratch was quite healed on the following day, when he went to work as usual. He felt no ill effects, moreover—neither pain nor bleeding, nor trouble of any kind—until the morning of the day on which he was admitted into the Hospital, when his throat began to swell until, soon, he could neither swallow nor speak.

These facts were made out by careful questioning, in order to eliminate the possibility of there having been an injury by the tobacco-pipe; and as nothing seemed to point in that direction, we came to the conclusion that the fall had probably nothing to do with the tonsillitis. It seemed impossible that there could have been, in the case of a wound of any importance, no bleeding at the time of the injury, and no symptoms whatever for two days afterwards.

On the evening of the day after the patient's admission into the Hospital, it was noted that he had spat up a little blood, and was very thirsty. His throat felt better, he said, and he had slept. Pulse, 124. Temperature, 105°.

On the following morning (September 21st) his temperature had fallen to 101.6°, and the pulse was 108. He had slept fairly well in the night, but had spat up some blood. The œdema of the throat was less.

September 22.—At 4 P.M. to-day a considerable arterial hæmorrhage came suddenly from the mouth, while he was engaged in conversation with another patient, and as suddenly ceased. The house-surgeon found him immediately afterwards much blanched. Pulse, 140.

Happening to be at the time in the Hospital, I was called to the ward; and finding the site of the puncture still plainly visible, and closed only by a clot, I probed the tonsil carefully, and found what seemed to be an abscess-cavity of considerable size. I could not, however, account at all satisfactorily for the hæmorrhage; but it seemed best, on the whole, to plug the cavity with lint soaked in perchloride of iron, and to have the patient watched. I determined to open up this cavity, if the hæmorrhage recurred, to plug it, and, if necessary, to tie the common carotid artery; but scarcely expected to be obliged to resort to such extreme measures.

At 9.30, on this same evening, there had been no return of hæmorrhage. The patient was still blanched, and he could scarcely swallow. Pulse, 104. Temperature, 100°. Respirations, 20.

September 23.—This morning at 7.30, and, notwithstanding digital pressure, again at 8.30, bleeding from the mouth suddenly recurred to the extent of 8 or 10 ounces; and on each occasion suddenly stopped. It was thought by the house-surgeon and dressers who were present that the blood did not come from the site of the puncture, as the plug seemed undisturbed; but the hæmorrhage was too sudden to allow any careful examination to be made with reference to this point.

When I arrived at the Hospital soon afterwards, it was evident that the patient was in a very dangerous condition, with the usual symptoms of a well-nigh fatal hæmorrhage; and I therefore at once proceeded to carry out what I had previously determined to do in the event of the recurrence of hæmorrhage.

Ether was administered; but now, once more, on the operating-table, the hæmorrhage recurred, and nearly suffocated the patient. On his recovering from this, I proceeded as quickly as possible to explore the cavity in the tonsil, after enlarging the previous puncture sufficiently to admit my finger. At first all that could be felt was a large cavity filled with blood-clot, and intersected by fibrous or other bands, which the accumulating blood had been unable to tear across; but when about to withdraw my finger, I felt the end of a foreign body, and withdrew without difficulty, with the help of forceps, a piece of a clay tobacco-pipe, with jagged end, measuring about an inch in length.

The cavity was plugged with lint squeezed nearly dry after being dipped in a solution of perchloride of iron.

The patient's case was now desperate enough, but it seemed that any slight chance he might have, lay in his being placed in the utmost possible security against further hæmorrhage; and

the bleeding spot being out of reach, I tied the common carotid at the level of the cricoid cartilage.

The patient did not rally at all from the prostrate condition in which the last hæmorrhage had left him, and died, with no alteration in his symptoms, two or three hours after removal from the operating theatre.

For the note of the post-mortem examination I am indebted to Mr. Milner, Surgical Registrar.

'Above and behind the left tonsil was an irregular cavity, the walls of which were blackened from perchloride of iron. No fragment of tobacco-pipe was in it. The internal carotid had not been wounded. It lay $\frac{1}{16}$ — $\frac{1}{8}$ inch away from the cavity. Into the cavity itself no artery could be traced, but the ascending pharyngeal appeared to terminate abruptly just at its edge, and its coats were stained by perchloride of iron.

'There was some blood, but not very much, in the smaller bronchial tubes. No blood was in the trachea. All the large veins were full of blood. The right side of the heart was much distended.'

Cases of fatal injury of important blood-vessels in the throat, usually the internal carotid artery, by tobacco-pipes, have frequently occurred; but in these cases, so far as I am aware, severe hæmorrhage has occurred always at the moment, or on the withdrawal of the foreign body. In the present instance, on the other hand, there was an almost complete absence of hæmorrhage, and indeed of any notable symptom, for two days after the accident. The patient was intoxicated at the time of the injury, and therefore incapable of noting what blood was lost; but it may be taken for granted that had the amount been more than trifling, his attention would have been called to it subsequently, either by the state of his clothes or by the general effects of loss of blood; or surgical help would have been obtained for him at the time by others. Moreover, the sudden onset of the symptoms subsequently, which, without doubt, depended on the sudden extravasation of blood, points rather to secondary than to a continuance of even slight primary hæmorrhage.

The case seems worthy of record, not only for the interest attaching to it in itself, but for instruction's sake; inasmuch as the knowledge of the possibility of the retention of a foreign body, even an inch of jagged tobacco-pipe, in the tissues behind the pharynx, without any distress for two days, may be valuable, in leading to a right interpretation of otherwise unaccountable symptoms, and to more timely measures for their relief.

ON THE PERCEPTION OF COLOUR IN JAUNDICE.

BY

J. WICKHAM LEGG, M.D., AND VINCENT HARRIS, M.B.

In some cases of jaundice the senses seem to give wrong information ; that is to say, it is unlike that received by persons who are not jaundiced. For example, some persons complain of a bitter taste, and others that all things look yellow. Antoninus doubtless expressed the common belief when he says, 'To the jaundiced honey tastes bitter ;'¹ and Lucretius, when he wrote those lines which have passed into a proverb :

Lurida præterea fiunt quæquomque tuentur
Arquati.²

Some have thought that a mere metaphor was used in both cases ; but it is scarcely probable, for the tendency of those whose minds have not received a medical training is to seize upon those symptoms, and to note them especially, which are but little thought of by the physician.

It does not seem that Hippocrates noticed the appearance of yellow vision in jaundice, although some think that he did.³ Galen certainly speaks of it.⁴ Our English Hippocrates, Sydenham, makes it one of the leading features of jaundice.⁵ Mercurialis denied its appearance altogether.⁶ The truth lies between

¹ Antoninus, Thoughts, vi. 57. Long's Transl. London, 1869. Sec. ed. p. 130.

² Lucretius, De Rerum Natura, iv. 331.

³ Hippocrates, De Locis in Homine, cap. xvi. Littré's ed. t. vi. p. 308.]

⁴ Galen, De Symptom. Different., cap. ii. Kühn's ed. vol. vii. p. 99.

⁵ Sydenham, Processus Iutegri, in Op. Omnia. Ed. Greenhill, p. 582.

⁶ Mercurialis, Var. Lect. in Med. Script. Venetiis, 1598. Lib. vi. p. 128.

these two extremes. There can be no doubt that patients do now and then complain of seeing all things, or only white things, yellow; but that the symptom is a very uncommon one. Peter Frank, for example, says that in the course of fifty-four years amongst thousands of jaundiced patients he met with this symptom only five times;¹ and Frerichs says that he has never seen a case, although he always asked for such.²

Twelve years ago Rose recorded a very interesting and curious case in which Daltonism accompanied the jaundice.³ As the patient died, there was no opportunity of seeing if the Daltonism persisted when the jaundice went off, and the Daltonism seems at one moment at least to have been complicated by yellow vision.

No one having seen fit to verify Rose's statements, we thought it a favourable opportunity during the last autumn, when several cases of jaundice presented themselves at the Hospital, to attempt to determine if Daltonism be at all a common accompaniment of jaundice. Messrs. Elliot of the Strand made us an instrument after the plan given by Rose in his paper, to which we refer the reader for an account of its construction and method of use.⁴

Our plan of observation was as follows: A sheet of paper, upon which were painted many of the various shades of the primary colours, was presented to the patient, and he was desired to name the colours as they were pointed out. Particular attention was directed to the testing of the patient's powers of recognising blue, green, and red. In every case, save two, perfectly correct answers were given, even to minute shades of colour. One of these two cases was a woman, the first spoken of below, born in Ireland, upon whose statements little trust could be placed. The second was a man, aged 21, with simple jaundice, who persisted in stating that the colour green was yellow, a statement which cannot be explained by any known optical laws. In two of the cases, in which it was stated by the patients themselves that they saw yellow, perfectly correct answers were given when their sight was tested by the sheet of colours. With Rose's instrument no changes in vision, either with or without the quartz, were noticed in any case. The patients were also examined with the ophthalmoscope.

We are indebted to the physicians of St. Bartholomew's Hospital for the courtesy with which they allowed us to make these observations on the patients under their care.

Faith Ward.—Margaret M'G., aged 62. Simple jaundice of

¹ J. P. Frank, *De Curand. Hom. Morb. Epitome*. Viennæ, 1821. Lib. vi. pars iii. p. 307.

² Frerichs, *Klinik der Leberkrankheiten*. Braunschweig, 1858. Bd. i. p. 115.

³ Rose, *Arch. für path. Anat.*, 1864, Bd. xxx. p. 442.

⁴ Rose, *ibid.*, 1863, Bd. xxviii. p. 30.

a fortnight's duration. Skin very yellow, and signs of jaundice well marked. Urine dark, high sp. gr. No albumen or casts. Fœces light and clay coloured. Pupils dilated and equal, discs natural. Persists in saying that green is red, and the reverse. No reliable results with the quartz.

Mark Ward.—James T., aged 35. Jaundice of nine months' duration from probable fibroid enlargement of liver. Liver dullness in right mammary line quite 10 inches. Painless. Pupils small, discs natural. Discoloration of skin not very deep. Fœces light brown. Urine deep brown, containing no casts. Although it was stated in the note of the clinical clerk that there was xanthopsia, the patient was perfectly correct in the estimation of colours both with the sheet of colours and with the instrument.

Matthew Ward.—George P., aged 27, carman. Simple jaundice of about ten days' duration, slight. Urine very dark. Fœces light. Pupils and discs natural. Estimation of colours quite natural.

Luke Ward.—J. J., aged 33. Jaundice during the acute fever of pneumonia. Temperature, 104° F. Urine dark, containing one-third albumen, urates and hyaline casts. Fœces dark and hard. Pupils natural. No results with the quartz.

Luke Ward.—Henry B., aged 57. Jaundice of three months' duration. Catarrhal jaundice from a gall-stone. Clearing up during stay in Hospital. Fœces lightish, getting dark. Urine dark, but less deep than it was some time before admission. No abnormality in eyes. Can distinguish colours, and gives no abnormality with quartz.

John Ward.—Edwin John R., aged 23. Jaundice of five weeks' duration. Temperature varying from 100·2° to 98·6° Is now getting better. Cause of jaundice unknown. Urine and fœces still abnormal in colour. No abnormalities of sight either with or without the instrument.

John Ward.—Joseph K., aged 12. Jaundice coming on in the course of empyema, and gradually increasing in intensity. Quite recent (when first examined), two days. Perception of colours quite perfect.

John Ward.—William S., aged 42. Carcinoma of liver. Liver much enlarged, and bosses of cancer to be felt all over it as far as umbilicus. Jaundice very deep, of four months' duration, and getting deeper. Fœces bileless. Urine dark and porter-like. Little itching of skin. Pulse, 68 to 76; temperature, 98°. Although xanthopsia was stated in the note to be present, the patient had perfect and even delicate perception of colours, and with the instrument gave the answers exactly as he ought.

Mark Ward.—Alfred G., aged 21, brass-finisher. Simple

jaundice, getting better. Pupils dilated, discs natural. Pulse, 48 to 60; temperature, $98\cdot5^{\circ}$; respirations, 18. Jaundice not intense, and liver not much enlarged. Mucous pale, not quite white. Can see well, and can distinguish colours fairly, but persists in calling pale greens yellows. No abnormal results with instrument.

John Ward.—Edwin L., aged 26, labourer. Simple jaundice, at its worst. Very deep discoloration. Pupils small. Fæces colourless. Urine dark. No results with instrument.

John Ward.—Joseph C., aged 19. Simple jaundice, very deep, of a week's duration. No dimness of sight. Fæces colourless. Urine dark. Perfect perception of colours with the sheet and with instrument.

Besides the patients within the Hospital, four out-patients, all men, and suffering from simple jaundice, were examined in like manner, and no changes either with the sheet of colours or with the instrument were perceived.

PARACENTESIS OF THE MEMBRANA TYMPANI

FOR

MUCOUS ACCUMULATION IN THE TYMPANUM.

BY

A. E. CUMBERBATCH.

Cases of catarrhal inflammation of the tympanum are frequently met with, in which the mucus secreted neither escapes by the Eustachian tube nor bursts through the membrane, but remains within the tympanic cavity.

When the fluid is not excessive, it may be removed by the simpler methods of treatment; when it is excessive, Schwartze has revived the operation of paracentesis of the membrane for such cases.

He says, 'If the quantity of collected fluid be inconsiderable, treatment by the air douche, inhalation of sal ammoniac, or injection of astringent fluids through the tube, suffices to effect a cure; but if the fluid be so abundant as to cause a bulging or bladder-like projection of the membrane, I maintain that the removal by paracentesis of the membrane is urgently indicated.'

This plan of treatment is objected to by some surgeons, who hold that the risk of such an operation is unnecessary, since as good a result may be obtained by simpler treatment.

That some of these cases may be cured without paracentesis of the membrane is probable, but it is certain that in others simpler means fail, and according to Hinton, it is doubtful in any case whether the cure is as speedy or effectual. He states: 'The only method of giving permanent relief appears to be evacuation of the fluid by incision of the membrane, repeated as often as the necessity arises. Inflation of the tympanum with air often produces a temporary improvement, and the symptoms may so far mend under treatment as to lead to the belief that a per-

manent cure is effected, but they are constantly prone to recur. The thorough evacuation, on the other hand, results in the restoration of a perfectly healthy state.'

The belief so prevalent of the danger of incising the membrana tympani would appear to be founded on very insufficient data, as, so far as I know, there is no case of death recorded where the operation has been skilfully performed. As regards the danger of making the hearing worse, it may be stated that the operation should not be undertaken till other means have been fairly tried without success. In such cases the risk of making matters worse is very slight indeed. The two following cases are illustrative of treatment by paracentesis of the membrana tympani.

CASE I.

Thomas Darnley, æt. 12, applied October 29, 1876, at the Aural Department of St. Bartholomew's Hospital. Says he has been deaf about two months. The deafness came on after a severe cold.

On examination the right membrana tympani seems fairly normal, except that its anterior segment is rather too much depressed.

The left membrane presents a small patch of congestion at its superior anterior segment. The rest of it is of a dull white colour, with a greyish-yellow tinge posteriorly. This part bulges very distinctly, and feels elastic when touched with a probe. The handle of the malleus indistinctly seen. Air enters the left tympanum with a moist sound, but produces no appreciable alteration in the membrane. Tuning-fork heard louder on the left side.

With the right ear the watch is heard at 20 inches off; on the left side, 1 inch. After inflation with Politzer's bag, right, 40 inches; left, 6 inches.

Throat slightly congested and relaxed. Tonsils somewhat enlarged.

Ordered an alum gargle, a blister behind the left ear, and citrate of iron and quinine internally.

Under the use of the remedies, coupled with inflation of the tympanum, the hearing with the right ear was almost normal at the end of three weeks; but there was no permanent improvement in the left ear. Although the hearing was always improved immediately after inflation, as on the first occasion, the improvement did not last.

So on November 19th an incision was made in the posterior segment of the membrane with a long double-edged needle. A

small quantity of greyish fluid oozed slowly from the wound. After inflation with Politzer's bag, a further quantity, very tenacious, escaped.

Hearing power increased to 6 inches. The nozzle of a syringe was next fitted tightly into the meatus, and a warm solution of carbonate of soda was passed through the tympanum, escaping through the nose, and bringing a quantity of mucus with it.

November 20.—Has not had any pain. No signs of inflammation about the membrane. Hearing with left ear 8 inches.

The tympanum again washed out with a solution of carbonate of soda.

November 22.—Opening in the membrane smaller. Watch, 15 inches.

November 23.—Wound closed. Watch, 12 inches.

The patient, who was very nervous, declined to allow the opening in the membrane to be re-established.

He was kept under observation some time longer, and the watch could always be heard at 15 inches distance.

CASE II.

William Boon, æt. 27, applied at the Aural Department June 2, 1876.

Has been troubled with deafness about eighteen months. It commenced with a severe cold, and is increasing.

Is very subject to colds, and whenever he has one the deafness is greater.

The left membrane is of a pale yellowish-brown colour. Posteriorly it is thinned and bulges slightly, and the bulging is more distinct after inflation of the tympanum. In front of the handle of the malleus, which is distinctly seen, is another thinned spot, much depressed.

Air enters the tympanum with a faint moist sound.

Right membrane slightly thickened, of a dull white colour, and much depressed. Tubes slightly obstructed. With right ear, watch 4 inches; with left, 2 inches.

Tuning-fork heard louder on the left side. Throat relaxed.

By the use of Politzer's bag and astringents to the throat and Eustachian tubes, the hearing on the right side increased to 24 inches, but on the left side it never increased beyond 4 inches.

On July 7th the posterior part of the membrane was incised, as in the other case. No fluid escaped at first, but after inflation a small quantity of tenacious mucus escaped through the wound.

This was removed, and the tympanum washed out with a solution of carbonate of soda.

July 8.—Membrane is of a pinkish hue. Ear slightly painful; was more painful during the night. Watch heard at 6 inches.

Tympanum again washed out.

July 9.—Only very slight vascularity of the membrane. No pain. Watch, 9 inches.

July 10.—Edges of the wound adherent. No vascularity of the membrane. No pain.

After forcible inflation the edges of the wound were separated, and the tympanum again washed out. Watch, 15 inches.

July 11.—Edges of the wound firmly united. Watch, 15 inches; 20 after inflation.

Patient was last seen August 24th, and then the watch was heard with right ear 24 inches, with the left 18 inches.

A SEVERE CASE OF RHEUMATIC FEVER TREATED SUCCESSFULLY BY SPLINTS.

BY

ROBERT BRIDGES, M.B.

The inflammation of the joints, which is the characteristic and constant symptom of rheumatic fever, is the cause of such intense suffering to the patient, that even were it possible to believe that this might in all cases be neglected without danger or fear of bad consequences, yet the physician would be hardly the less bound to do all in his power to alleviate it. Unfortunately the remedies that have been tried bear witness by their number to their inefficiency; and it is common in clinical teaching to see the special assaults of this disease considered in themselves as unimportant so long as they spare the viscera, while attention is mainly directed to the heart and pericardium; and the state of the patient in whom these continue sound is held to be so satisfactory that he is merely encouraged to support his agonies, in expectation of a future and uncertain day when they will probably take their leave.

This method of treatment, if treatment it may be called, has found support in the tendency of a therapeutic fashion, that by the watchword of expectancy has not only taught prudence, but has often excused indolence and spared judgment; so that it is not a matter of wonder to the student to see some patients waiting for their pain to pass off as others do for their fever, while the knowledge that inflammation of the heart is more serious than that of the knee is sufficient erudition to divert their attention and defer their efforts.

Such considerations, coupled with the common agreement of surgeons concerning the treatment of inflamed joints, led me to ask leave to publish in this volume the account of a case of rheumatic fever of unusual severity treated by splints. The

method is not a new one, and I shall be able to give some account of its history ; but since during five years at our Hospital I had never seen it put in practice, nor even heard it mentioned, so it was not till I became responsible for the treatment of a patient who seemed to be dying of sheer pain, that being myself compelled to seek some such resource, I had an opportunity of witnessing its effects. I make no apology for having only one case to report ; it is more likely to be read, and is, in my opinion, of more value than a table of figures, and whatever experience I have persuades me that it is worth reporting.

R. L., admitted to Hope Ward, 22d May 1876. A sound, stout, healthy girl, aged 21, light complexion. Never had rheumatic fever before. The seventh day before admission slept in a house without windows, and woke with rheumatic pains and loss of voice. Has suffered severely since. On admission, voice still husky, face flushed ; restlessness and intense pain. Not much swelling of the joints, but pain in ankles, knees, shoulders, collar-bones, and a little in the hands. A slight systolic blow with the heart-sounds ; no other sign of the heart being affected (and this sign remained unchanged throughout the illness). Some rhonchus in lungs. Bowels confined. Temperature, $102^{\circ}2'$. Pulse, 100. Milk diet, with an extra pint of milk ; pil. cal. c. jal., gr. x. stat. Hst. sennæ co., \bar{z} j. post iv. horas.

Evening.—Bowels freely acted on. Temperature, 102° . Same state of restlessness. Ordered chloral hydrate, gr. xx. No sleep.

8th day.—A little sleep towards morning. Temperature at 10 A.M., 102° . Lemons ij. added to diet, and a mixture of pot. bicarb. gr. xxx., tinct. hyoscy. m. xxx., ordered, for routine, three times a day. Morphia, $\frac{1}{8}$ gr. swallowed, with some relief.

Dr. Black visited the ward in the afternoon, and pronounced the case to be one of great severity and doubtful prognosis. Temperature, $103^{\circ}8'$.

8.30 P.M.—Patient in great distress, groaning and moaning. Pulse without force or regularity ; the sounds of the heart feeble and fluttering. The temperature $104^{\circ}2'$, and at 8.35 P.M., $104^{\circ}4'$. Finding the body heat still rising, and considering her state due to her pain, I ordered morph. hyd. gr. $\frac{1}{3}$, which she had no sooner swallowed than she uttered a cry, and throwing her head back and turning up her eyes, ceased to breathe. I could scarcely feel the pulse, and the action of the heart was as feeble and irregular as I have seen it in dying persons. I aroused her by mechanical irritation and by cold, and succeeded in making her swallow an ounce of raw brandy. The pulse quickly improved, and as she became conscious she put her right hand up to her mouth as if

to remove something from it, the brandy having burnt her. This action will indicate her state, for the inflammation of the joints was such as to render conscious movement of the arm impossible. I continued now with her administering brandy and water, watching to see how it affected the body heat, which I supposed to be rising. I took the following temperatures:—

8.38.—Temperature, 104°.

8.42.—Temperature, 104.2°.

8.46.—Temperature, 103.8°.

8.50.—Temperature, 103.6°.

8.53.—Temperature, 103.2°.

8.58.—Temperature, 103°.

9.2.—Temperature, 103.2°.

The pulse was by this time steady and full, but the temperature showed no tendency to descend further, and the general state of the patient seemed to me so unsatisfactory that I determined to have splints applied to the extremities with a view of relieving the pain. Mr. Edwards undertook the surgical part, and at 9.25 P.M. the arms were fixed, and at 9.45 P.M. the leg-splints were put on. At this moment the temperature, which had not fallen before we began to apply the splints, was at 101.8°. Instantaneous, though not complete, relief was obtained, and the patient, who had not spoken readily before, volunteered expressions of gratitude and comfort. The arm-splints were bent at the elbow, and lay supported by pillows across the body; the leg-splints had a rectangular support for the foot, and, bent slightly at the knee, came half way up the thigh. The application did not cause much pain. The joints were seized firmly by the hand, moved slowly, and bound down tightly. At 11.15 P.M. the patient was sound asleep.

9th day.—Patient had slept till 1.30 A.M., and then again till morning. At daybreak she seemed again in distress, but not so great. At 9.15 A.M. in great pain. Temperature, 103°; pulse again irregular, and the heart's action uncertain, intermitting once in four beats. The bandages on the arms were readjusted; they had not been as firmly bound down as the legs. Brandy, \bar{z} v. per diem. Morph. hyd. gr. $\frac{1}{6}$ sextis horis. The routine medicine to be discontinued. The pulse improved again with the brandy and morphia, and she slept soon after taking them. Temperature, 102.6°.

11.30.—Asleep. Respirations, 28.

12.30. P.M.—Just awake. Heart's action regular. Temperature, 102°.

2.30.—Temperature, 101.3°. Heart regular.

3.30.—Temperature, 101.5°. Heart regular.

5.30.—Temperature, 102.5°. Heart regular.

8 P.M.—Temperature, 104°. Morphia, gr. $\frac{1}{8}$. Slept till 10th day, 2 A.M., when morphia repeated. Slept till 6 A.M.

10 A.M.—Pain universal. Heart regular. Temperature, 102.2°; pulse, 80. Eggs, brandy, and milk.

Noon.—Temperature, 102.2°.

2.15.—Temperature, 101.3°; pulse, 88.

6.—Temperature, 101.8°.

11th day, 9.30 A.M.—Temperature, 101.1°; pulse, 78. Slept all last night.

11.50.—Temperature, 100.2°; pulse, 78. Brandy reduced to $\bar{3}j$.

3 P.M.—Temperature, 100°. Asleep.

6.—Temperature, 101.6°. Less pain.

10.—Temperature, 101.2°; pulse, 72. Pain in chest. Heart as before. The tongue has a thick white fur; is moist. No duskiness of complexion. A good deal of restlessness and cough.

12th day, 10.30.—Temperature, 100.2°; pulse, 92.

Noon.—Temperature, 103.2°; pulse, 86. Pil. cal. c. jal., gr. x.

Afternoon.—Splints removed from her legs at her request.

6.7 P.M.—Patient in great distress again. Pulse irregular. Head 'burning.' Skin feeling cool to the hand. Body heat 106.4°. I tried the effect of brandy again, giving first half an ounce.

6.12.—Temperature, 106.2°.

6.15.—Temperature, 106.2°.

6.17.—Temperature, 106.4°. Brandy, $\bar{3}ij$.

6.22.—Temperature, 106.2°.

6.30.—Temperature, 106°. Morphia, gr. $\frac{1}{8}$.

7.30.—Temperature, 105.2°; pulse, 80.

9.15.—Temperature, 104.5°. Has slept.

10.30.—Temperature, 104.5°. Pil. cal. c. jal., gr. x.; brandy, $\bar{5}ij$; morphia, gr. $\frac{1}{8}$. Patient slept.

12.15 A.M.—Temperature, 104°.

13th day.—Had slept all night. Temperature, 104.2°. Restless. Chest sore. Drinks plenty of milk and brandy, with eggs. Heart and lungs as on admission.

3 P.M.—Temperature, 104°.

5.30.—Temperature, 104.6°.

6.30.—Temperature, 103.2°; pulse, 104.

10.30.—Temperature, 101.6°. Does not feel better. Skin moist. Severe pain across brow. Ice to head. Morphia, gr. $\frac{1}{8}$. Pulse, 98, of good character.

14th day, 10.30 A.M.—Temperature, 103°4'. Ice ordered to head had been neglected, and was now applied. Morphia, gr. $\frac{1}{4}$.

3.10 P.M.—Temperature, 102°.

4.10.—Temperature, 103°4'. Asleep.

8.15.—Temperature, 103°4'; pulse, 92. Awake.

10.30.—Delirious. Pulse, 90. Pupils equal. Heart same. Splints reapplied. The ankles were swelled a good deal, but not red. Headache bad. The pulse quickened while the splints were put on; after the splints were put on, temperature 102°8'. Morphia.

15th day.—Slept all night till 4 A.M.

10.30 A.M.—Temperature, 103°2'. Morphia, gr. $\frac{1}{8}$. Omit brandy.

1 P.M.—Temperature, 104°2'.

2.30.—Temperature, 104°.

6.—Temperature, 101°8'.

10.30.—Temperature, 101°4'.

16th day.—Great improvement in patient's general state.

10.15.—Temperature, 103°. Morphia, gr. $\frac{1}{8}$. Omit brandy.

10.45.—Temperature, 102°8'.

8 P.M.—Temperature, 103°2'. Pulse very full; respirations, 52 during sleep. Some cough.

10.30.—Temperature, 101°8'. Bowels freely opened.

17th day.—The patient seemed fairly on the way to recovery, and Dr. Shuter, who took charge of the case from this day (on which I resigned my charge as house physician), informs me that there were no further symptoms of note; that the splints were removed on the 21st day, at which time there was scarcely any pain; and that on the 26th day the pulse and temperature were registered as normal, and the patient felt quite well, but weak. She was discharged from the Hospital on July 16th, the 55th day after admission. There was no heart murmur.

As for my own notes of the case, they are little more than a transcript of what was written at the bedside, and I believe that the registrations of my thermometer may be trusted. These were all made with care, and many of them twice. I have no remarks to make but such as will occur to the reader.

When I considered the great relief which this treatment had afforded, and the successful issue of the case, which had from the first promised but ill, together with the surgical and anatomical propriety of the means used, I was very curious to discover what trial the method had had, and for what reasons it had been put aside. I found mention of a treatise by Dr. A. Goltschalk, printed at Cologne in 1845, and entitled 'Darstellung der rheumatischen Krankheiten auf anatomischer Grundlage.' I have

not seen this book, but it is stated to contain the details of three cases of acute rheumatism treated by fixing the joints at rest, according to the method advocated by Seutin, whose paper, if he wrote one, I have also been unable to procure. A passage from Goltschalk is quoted by Dr. Oehme, who refers to the book as if he considered it worthy of attention; but the subject dropped out of notice, and was taken up independently by Professor Concato of Bologna in 1869, who related a case in the March number of the '*Rivista clinica di Bologna*' of that year, in which he states, that being at his wits' end to know what to do, he hit upon the plan of using splints: '*Tornati infruttuosi i mezzi terapeutici fino allora impiegati, mi scorse in mente di sperimentare la applicazione di un apparecchio inamovibile.*' The temperature of the patient, a girl, was $39^{\circ}6$ Cent. The application seems to have been made with difficulty, and to have caused great pain; but the relief was very great, and the progress of the case, a severe one—there was a suspicion of its being blenorrhagic—was satisfactory. A year or two after this he published a pamphlet, '*Il Rheumatismo articolare acuto, e l'Apparecchio inamovibile;*' and Tamburini, who followed his plan, reported four cases. In the winter of 1870-71, Dr. Heubner, who was acting as physician to one of the war hospitals in Leipzig, hit on the same method, of the success of which a short account is appended to his interesting paper, '*Beiträge zur internen Kriegsmedizin,*' in the 12th volume of the '*Archiv der Heilkunde.*' In the same periodical, vol. xiv., there is a paper by Dr. Oehme, apparently written with the notion of settling the matter once for all; but it is rendered unreadable, and in my opinion nearly valueless, by the statistics into which he has sought to condense his experience. His figures seem to claim for the method that it shortened the duration of the pain on the average from twenty-one days to thirteen, while the fever was reduced by three or four days, and the whole duration of the illness by six or nine days. I am not sure that I understand all his figures, but I am a good enough mathematician to know that where ciphers stand for patients it is probable that patients stand for ciphers, while as a physician it appears to me ridiculous to call the indiscriminate use of any method 'giving it a fair trial.' He states that when once the splints are applied it is well not to remove them too soon, as the release of the joints is accompanied by great pain and an exacerbation of the disease, a remark which seems confirmed by the notes of my case (see the 12th day), and must be considered as demonstrative proof of the activity of the remedy.

I have found all who speak of this method give it unqualified

praise, and I will end my remarks by calling on three witnesses to speak.

Dr. Oehme says, 'By treating acute rheumatism with splints, the pain was reduced to a minimum, the duration of the fever was considerably shortened, while the course of the whole illness was actually cut short.'

Dr. Heubner says, 'The pain was less than under any other, even the narcotic, treatment; the fever passed off sooner; the sweats were less.'

Concato says, 'The application of splints in acute arthritis is the only means that up to this time has been found to produce immediate beneficial result.'

REPORT ON THE CATARACT CASES.

BY

HENRY POWER.

The number of cases of cataract that have been operated upon in the Ophthalmic Wards of St. Bartholomew's Hospital, since they were opened by the Prince and Princess of Wales in 1870, having now exceeded 100, Mr. Vernon and myself have determined to give the results of the first century, so far as they can be obtained, in a classified form, in order that they may be compared with those of other hospitals both general and special, and also with those that will be performed by future operators occupying our present position, by more or less dissimilar methods.

The following tables have been drawn up with much care by Mr. Kingston Barton, the present house-surgeon, to whose interest in his duties, knowledge of the subject, and indefatigable attention to the patients, some part at least of the good results obtained during the past twelve months is attributable. They may be relied upon as being strictly accurate. No one can regret more than the ophthalmic surgeons the not infrequent imperfection of the record in the most important point of all, the vision of the patient; but many circumstances concur to render this common, and those who are most familiar with the difficulty of completing hospital cases will be most willing to make some allowances. Cases occasionally leave the Hospital unexpectedly; some when not in a condition to have the vision taken, and under a promise, that remains unfulfilled, to return; whilst, with ever-changing house-surgeons and dressers, some cases which have been of great interest are unrecognised or passed over when they present themselves some weeks after, when the

briefest note would have completed the account and rendered it definite.

The operation is performed in the small operating-room adjoining the Ophthalmic Wards. It has a western light. The chloroformist (Mr. Mills) is always in attendance. I rather prefer operating for cataract without chloroform, though I never object to it if the patient desires it. Mr. Vernon almost invariably has it administered. There are advantages and disadvantages in its use. It no doubt keeps the patient still, and enables the operation to be performed to perfection; but I have seen eyes lost from the vomiting that has subsequently occurred, and this is more likely to occur in those who are advanced in years. If the patient be docile and steady, this danger is wholly avoided. But there are some who will bear the first part of the operation, but whose courage fails towards the close, or whose hyaloid membrane is very thin, and in whom a sudden movement leads to rupture of that membrane, escape of the vitreous, and an ultimately unsatisfactory result, a result that would not have occurred under chloroform.

We have, after fair trials, almost wholly discarded the use of ether and gas, either separately or conjoined. They both produce intense congestion of the head and vessels of the eye, and one of our cases was lost on the spot from hæmorrhage, which we attributed to this cause. Moreover, the muscular movements of the patients under ether, both of the body generally and of the respiratory muscles in particular, are often very considerable, and greatly interfere with the delicate proceeding of this operation.

It will be seen from the last table that both Mr. Vernon and myself have given a fair trial to methods of operation recommended by men of large experience, but the method we now adopt has proved so satisfactory in our hands that it will require very strong evidence I think to induce either of us to change it, and we have been led to it quite independently from observing the effects in each other's hands.

It may be well, therefore, to give a short description of this method.

The patient is always operated upon in the recumbent position. The lids are separated by a spring speculum, with screw-stop for fixing the hinge, being well curved round the temple to be out of the way of the operator. The eye is fixed with a pair of ordinary fixing-forceps, and the point of a long narrow Gräfe's knife is made to penetrate the sclerotic just outside the sclero-corneal junction, with a slight inclination downwards—at the level, in most cases, of the junction of the upper two-fifths with the lower three-fifths of the cornea. The upper section is made by

the edge of the knife being carried along the sclero-corneal junction. The iris-forceps is now introduced and a moderate-sized

portion of the iris removed.



The capsule is carefully

divided by a cystitome, and the lens delivered by gently pressing and following up the lens matter. The greatest care is taken to remove all fragments by means of a curette. The more thoroughly this part of the operation is done, in my opinion, the better the result. Great care is also taken to see that the cut edges of the iris are not engaged in the wound of the cornea. A pad and bandage with moderate pressure are then applied and the patient carried to bed. As a rule, the dressing is not touched till two days afterwards. If there has been any bleeding or discharge on the dressing, it is changed, without at all disturbing the eye, eight hours after operation, and the dressing not again touched for forty-eight hours.

The eye is then carefully looked at and redressed every day for about seven days following operation, when the patient is generally allowed to be up and have the eye uncovered for several hours during the day, a shade being worn. In from ten to fourteen days the pads are entirely left off.

General Analysis of the Cataract-Extraction Operations from October 1, 1870, to May 1876.

No. of patients operated upon	78
No. of eyes operated upon	<u>109</u>
No. of failures	20
No. of successes	89
	<u>109</u>

Methods of operation—

Gräfe's performed on	76
Liebreich's „	18
Flap „	10
Couching „	2
Linear „	1
Schuff's „	1
Spoon „	1
	<u>109</u>

Of the twenty failures—

Gräfe's operation was performed on	.	.	11
Liebreich's	"	"	3
Flap	"	"	2
Schuff's	"	"	1
Linear	"	"	1
Couching	"	"	2
			<hr/>
			20

Of the eighty-nine successes—

Gräfe's operation was performed on	.	.	65
Liebreich's	"	"	15
Flap	"	"	8
Spoon	"	"	1
			<hr/>
			89

The general percentage of successful cases since 1870 is 81.65 per cent. But taking the last fifty-two cases, which was the total number of eyes operated upon during 1874-75, and first five months of 1876, there were only five failures, or a percentage of 90.19 successes.

Analysis of each Year.

1871.—Nine patients with senile cataract. Thirteen eyes operated upon. One failure.

Gräfe's operation performed on	.	.	.	Eyes.
Flap	"	"	.	7
Spoon	"	"	.	5
			.	1
				<hr/>
				13

The failure was a Gräfe. See Table I.

1872.—Seventeen patients with senile cataract. Twenty-three eyes operated upon. Sixteen successes. Seven failures.

Gräfe's operation was performed on	.	.	.	Eyes.
Liebreich's	"	"	.	6
Linear	"	"	.	15
Schuff's	"	"	.	1
			.	1
				<hr/>
				23

Of the failures—		Eyes.
Gräfe's was done on	3
Liebreich's	„	2
Linear	„	1
Schuff's	„	1
		—
		<u>7</u>

See Table II.

1873.—Sixteen patients with cataract: twelve senile, two traumatic, one congenital, and one diabetic.

Of the twelve senile patients, nineteen eyes were operated on, there being seven failures and twelve successes.

		Eyes.
Gräfe's operation was performed on	12
Flap	„	3
Liebreich's	„	2
Couching	„	2
		—
		<u>19</u>

Of the seven failures—

Gräfe's operation was performed on	3
Liebreich's	„	1
Flap	„	1
Couching	„	2
		—
		<u>7</u>

See Table III.

The two traumatic, congenital, and diabetic were all successful.

1874.—Twenty-eight patients with cataract: sixteen senile, six congenital, and six traumatic.

Of the sixteen senile patients, twenty-one eyes were operated upon, there being two failures.

Of the twenty-one operations, twenty were the Gräfe operation, and one the old flap. The two failures were one Gräfe and the flap.

The nineteen successful operations all gave good results. Two cases, however, some months after, independent of the operation, lost a good deal of their vision through iritis. See Table IV.

Of the six congenital cataract patients, seven eyes were operated upon, four being failures.

Of the six traumatic cataracts four were successful. See Table V.

1875.—Seven patients with senile cataract. Seven eyes oper-

ated upon. One failure. Gräfe's operation was performed upon six, the flap upon one. The failure was a Gräfe, and was due to the man having a broken-down constitution, having taken calomel for years.

Of the remaining thirteen patients, fifteen eyes were operated upon, three being failures. For further particulars see Table VI.

1876.—Seventeen senile patients. Twenty-six eyes operated upon. Only two failures, those failures being Gräfe operations, and patients who had been previously broken down in health. One was an old French governess, the other an old decrepit washerwoman, both cases which would not have been touched in private practice.

	Eyes.
Gräfe's operation was performed upon . . .	25
Liebreich's " " . . .	1
	<hr style="width: 10%; margin: 0 auto;"/>
	<u>26</u>

The vision of several of these patients was very good, but could not be perfectly taken owing to the short period after operation.

Of the three congenital cataracts, five eyes were operated upon, one being successful.

The two traumatic were also successful. See Table VII.

TABLE I.—YEAR 1871.

Name.	Age.	Result.	Operator.	Nature of R. E.	Nature of L. E.	Date of Operation, R. E.	Date of Operation, L. E.	Further Operation, R. E.	Further Operation, L. E.	Date of further Operation, R. E.	Date of further Operation, L. E.	Result, R. E.	Result, L. E.
Martha Davis	63	F	H. Power	...	G.	...	2 2 71	There was total posterior synechia. Large hyphema present when patient left (very soon after operation).
Lawrence Negle	44	S	B. J. Vernon	G.	...	2 2 71	Some capsule left. V. = good. Requires slight needle operation.	
Joshua Harris	67	SS	B. J. Vernon	Flap upwards	Flap downwards.	9 2 71	9 2 71	V. with + 3.75 = $\frac{1}{8}$. Can read 1 $\frac{1}{2}$ Sn. with glasses. (Sn. 1 $\frac{1}{2}$. Note made subsequent to patient leaving Hospital.)	
William Richardson	70	S	H. Power	...	Flap	...	4 4 71	V. with + 3.75 = $\frac{2}{6}$. very soon after operation. V. = good. No further test taken.	
William Collmult.	40	S	H. Power	...	Spoon.	...	2 5 71	Had opaque cornea previous to operation. Operation otherwise quite successful.	
William Seymour	60	SS	B. J. Vernon	G.	G.	25 5 71	25 5 71	22 6 71	...	V. = good.	
Richard Moon	66	SS	B. J. Vernon	G.	G.	20 6 71	28 6 71	N.	N.	15 8 71	15 8 71	V. + 2 = No. 1 (Jäger?) + 3 = V. good for distance.	
Sarah	62	S	H. Power	...	G.	...	28 9 71	V. = good, pupil clear. + 3 reads well (does not say what).	
Margite James	71	SS	H. Power	Flap	Flap (By Mr. Savory).	17 10 71	— 68	V. = good. Slight capsule requiring needle.	

F. = Failure. S. = Success. G. = Gräfe's extraction. N. = Needle operation for capsule. V. = Vision. + 2 = Convex lens of 2-inch focus. R. E. = Right eye. L. E. = Left eye. $\frac{1}{8}$ = Snellen's type, 40 at 12 feet. Sn. = Snellen's test types.

TABLE II.—YEAR 1872.

Seventeen Patients with Senile Cataract.

Name.	Age.	Result.	Operator.	Nature of E.	Nature of E.	Date of R. E. Operation.	Date of L. E. Operation.	Further R. E. Operation.	Further L. E. Operation.	Date of further R. E. Operation.	Date of further L. E. Operation.	Result, R. E.	Result, L. E.
G. Ashton	38	F	H. Power	Linear	Linear	10 1 72	Suppurative iritis. V. = nil.	...
Martha Le Beau	59	F	B. J. Vernon	G.	G.	29 2 72	Iritis leading to occluded pupil. V. = light.	...
Susan Edwards	74	F	B. J. Vernon	Schuff's	Schuff's	26 3 72	Sloughing of wound. V. = light. In a bad state of health previous.	...
Thos. Hinton	39	SS	H. Power	G.	G.	10 4 72	11 6 72	2.5 for reading. 3.25 for distance. V. = good.	2.5 for reading. 3.25 for distance. V. = good.
Wm. Tagg	76	FF	H. Power	G.	G.	28 4 72	28 4 72	Suppurative iritis. V. = nil.	Suppurative iritis. V. = nil.
William Williamson	54	SS	H. Power	G.	G.	7 5 72	4 6 72	V. = 6½ Jäger with + 2. Patient went out too soon after operation for testing accurately.	...
Eliza Bone	65	S	H. Power	Lieb.	Lieb.	25 6 72	...	Iridectomy.	...	5 7 72	...	V. = good. Glasses not tried. Had prolapse of iris after operation. Iridectomy.	...

Agnes Johns 62	S	H. Power	...	Lieb.	...	22	8 72	V. with + 6 = very good. Too irritable to try glasses fairly.
Louisa Wright	78	SS	B. J. Vernon	Lieb.	N.	3 9	72 27 8 72	N.	5 11	72 5 11	72	V. = fair. Post. staphylocoma. (Is able to read moderate-sized print with spectacles, 22/7/76.)	
Wm. Craft	68	F	H. Power	Lieb.	26	8 72	V. = nil. Large amount of vitreous escaped from nonclosure of wound seven days after operation.	
Jas. Purnell	70	S	B. J. Vernon	Lieb.	...	24	9 72	Capsule only interfering with V. Refused further treatment. V. with 2.5 = 1 $\frac{1}{2}$ ov.	
Anna Pattison	56	SS	B. J. Vernon	Lieb.	N.	24	9 72	8 10 72	12 11	72	...	V. = Sn. 2 $\frac{1}{2}$ with 2.5. Both eyes together, V. = Sn. 2.	
Caleb Rickettes	48	S	H. Power	Lieb.	...	22	10 72	V. with + 3.5 = 4 $\frac{3}{8}$. V. = 5 $\frac{1}{2}$ o.	
John Evans	43	S	B. J. Vernon	G.	...	22	10 72	V. with 2.5 = Sn. 4 $\frac{1}{2}$. 3.5 V. = 2 $\frac{9}{16}$.	
Amos Poulson	62	S	H. Power	Lieb.	Tapped.	9 9	72 29 10 72	Iridectomy.	16 1 73	25 3 73	...	Suppurative iritis, shrunken globe.	
M. Stanton	56	S	H. Power	Lieb.	N.	29	10 72	...	4 12	72	...	+ 2.75 = Sn. 1 $\frac{1}{2}$. + 4 = 3 $\frac{1}{2}$ o.	
Grace Hurlstone	72	S	B. J. Vernon	Lieb.	N.	12	4 72	...	29 10	74	...	Count fingers 3 feet. + 2 = good vision.	

F. = Failure, S. = Success, Lieb. = Liebreich's extraction. N. = Needle operation for opaque capsule. Sn. = Snellen's test types, $\frac{3}{8}$ = Snellen's type, 70 at 3 feet. R. E. = Right eye. L. E. = Left eye. V. = Vision. + 25 = Convex lens of 2.5-inch focus. 5" = Five inches distant.

TABLE III.—YEAR 1873.

Twelve Patients with Senile Cataract.

Name.	Age.	Result.	Operator.	Nature of R. E.	Nature of L. E.	Date of R. E. Operation.	Date of L. E. Operation.	Further R. E. Operation.	Further L. E. Operation.	Date of further R. E. Operation.	Date of further L. E. Operation.	Result, R. E.	Result, L. E.
George Cole	31	F	H. Power	...	Lieb.	...	7 1 73	...	Excision of globe	...	2 3 76	...	Adhesive iritis leading to sympathetic ophthalmia.
Elizabeth Ramsey	60	FF	B. J. Vernon	G.	G.	— 72	7 1 73	Iridectomy N.	N.	17 12 73 18 2 73	2 73	V. = fingers at 1'.	V. = light. Recurrent iritis in both eyes.
John Chapman	54	S	B. J. Vernon	Lieb.	...	7 1 73	V. = good, $\frac{3}{8}$. (Can see to read with spectacles, 26/7/76.)	
Wm. Dicker	70	F	H. Power	G.	...	25 2 73	Escape of vitreous and hemorrhage (globe became shrunken and squared).	
Harriet Dearing	71	SS	H. Power	Flap	Flap	4 3 73	1 4 73	V. = good. Not given in ex-actitude.	V. = good.
Charles Chierwood	74	FF	H. Power	Conchling	Conchling	25 3 73	25 3 73	Capsule	Capsule	22 4 73	22 4 73	V. = not much. Opaque capsules.	V. = not much.
Joseph Tomsett	66	S	H. Power	G.	...	25 3 73	V. with + 2.75 = 1½ Sn. at 9".	
Sarah Birch	49	SS	H. Power	G.	G.	30 9 73	15 10 73	V. with + 4.25 = 20.	V. with 4.5 = 20.
Mary Glynn	57	SS	H. Power	G.	G.	7 10 73	30 10 73	N.	11 11 73	V. with 3.25 = 20.	V. with 3.25 = 20. 3.75 glasses ordered.

Frederick Hinin	63	SF	H. Power	G.	Flap	23 10 73	14 10 70	V. = good, but particulars not given. Left Hospital soon.	Irido-choroiditis. Shrunken globe.
William Newland	57	S	B. J. Vernon	G.	...	18 11 73	V. = fair. Patient went out very soon after the operation.	
John Ashford	53	SS	H. Power	G.	G.	26 11 73	30 12 73	V. with + 4.5 = $\frac{20}{20}$, and with + 3 = Sn. 4 at 1'.	V. with + 4.5 = $\frac{20}{20}$, and with + 3 = Sn. 4 at 1'.

Two Patients with Traumatic Cataract.

Eliza Hollis	5	S	H. Power	...	N.	...	28 10 73	...	N.	Injury caused by a piece of wood in chopping wood flying off. V. = fair.
William Horswill	34	S	H. Power	...	Capsule	...	18 11 73	Had a blow in the eye when a child. Displaced pupil and opaque lens. V. = $\frac{20}{20}$.

One Case of Congenital Cataract.

Mary Ann White	4	S	H. Power	...	N.	...	24 9 73	...	N.	Curette	...	30 9 73	4 11 73	...	V. = good. + 3 ordered.
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One Case of Diabetic Cataract.

Margaret M. Leager	15	S	H. Power	N.	N.	13 3 73	13 3 73	Curette	...	29 3 73	...	V. with 4.25 = $\frac{20}{20}$, and 3½ Jäger with + 2.75.	Not given.
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F. = Failure. S. = Success. G. = Gräfe's operation. N. = Needle operation. Lieb. = Liebreich's operation. V. = Vision. 1' = One foot. 9" = Nine inches. R. E. = Right eye. L. E. = Left eye. Sn. = Snellen's test types. $\frac{70}{70}$ = Snellen's type, 70 at 20 feet. + 3 = Convex lens of 3-inch focus.

TABLE IV.—YEAR 1874.

Sixteen Patients with Senile Cataract.

Name.	Age.	Result.	Operator.	Nature of E.	Nature of L. E.	Date of E. Operation, R. E.	Date of E. Operation, L. E.	Further Operation, R. E.	Further Operation, L. E.	Date of further Operation, R. E.	Date of further Operation, L. E.	Result, R. E.	Result, L. E.
Henry Biney	71	SS	B. J. Vernon	G.	G.	19 8 73	18 9 73	N.	N.	15 1 74	15 1 74	Sn. 2½ with + 2. Spectacles 2½ and 3½.	Sn. 2½ + 2. 4½ + 4 25. Spectacles 2½ and 3½. Some months after, vision much diminished from occluded pupil.
John Hill	63	SS	B. J. Vernon	G.	G.	21 10 73	30 12 73	N. and iridectomy.	...	6 2 74	...	V. = 1½ with glasses.	...
Philip Knebone	66	S	H. Power	G.	...	18 3 74	3 75 = 28. Very well satisfied with result.	...
Jas. Jennings	70	S	H. Power	G.	...	28 4 74	3.5 distance, 2.25 can do ordinary reading.	...
Job Webb	64	S	H. Power	...	G.	...	2 6 74	4½. No statement except vision good.
Flora Reid	63	F	H. Power	Flap	...	9 6 74	Sloughing of wound. Small occluded pupil. V. = light.	...
William Densley	48	SS	B. J. Vernon	G.	G.	30 6 74	13 10 74	V. = 4½ with 3.25. Sn. 1½ with + 2.25.	Sn. 3½ with + 2.25. Many months afterwards iritis did damage.
Frances Murphiss	69	F	H. Power	G.	...	7 7 74	Sloughing of wound. V. = light.	V. = 168. No further result given.
John Morley	29	S	H. Power	...	G.	...	— 72
Geo. Miles	78	S	B. J. Vernon	G.	...	11 8 74	V. = good. Ordered + 4. Went out on account of family affairs.	...
John Richards	68	S	H. Power	...	G.	...	3 9 74
M. Chappell	70	S	H. Power	...	G.	...	22 9 74
E. Rosenberg	54	S	H. Power	G.	G.	6 10 74	6 10 74
Jos. Cheatie	55	S	H. Power	G.	...	6 10 74	...	N.	26 1 75	Fingers at 1'. + 3 = 1½. Left globe excised 25/1, 75.
Bliza Powell	67	SS	H. Power	G.	N.	20 10 74	V. with + 4 25 = 1½. V. with 3.5 = 4½. + 2.5 = Sn. 4.
Wm. Greene	65	SS	H. Power	G.	G.	17 11 74	1 12 74	V. with 3.5 = 4½. + 2.5 = Sn. 4. Did not know her letters.

F. = Failure. S. = Success. G. = Griffe's operation. N. = Needle operation. Sn. = Snellen's test types. 1½ = Snellen's type, 70 at 12 feet. V. = Vision. R. E. = Right eye. L. E. = Left eye. V' = One foot, + 2.5 = Convex lens of 2.5-inch focus.

Six Patients with Congenital Cataract.

Name.	Age	Result.	Operator.	Nature of Operation, R. E.	Nature of Operation, L. E.	Date of Operation, R. E.	Date of Operation, L. E.	Further Operation, R. E.	Further Operation, L. E.	Date of further Operation, R. E.	Date of further Operation, L. E.	Result, R. E.	Result, L. E.
S. Burton	10	F	B. J. Vernon	..	G.	18 6 74	26 6 74	Irideremia of R. E. Went out before absorption. Atrophy of choroid and cresent.	V. = not much. Nystagmus present.
Ada Jeffreys	16	F	H. Power	N.	..	7 7 74	Capsule left behind. V. = light. Cataract of this eye complete.	Posterior synechia. Cataract of this eye was zonular.
Emily Doswell	9	F	B. J. Vernon	N.	V. = light. Irido-cyclitis (syphilis)
Eliza Price	21	F	B. J. Vernon	..	N.	..	4 8 74	Curette	V. with 4.25 = 2/3, and with 2.75 = 1/2 Sn.
R. Walker	35	SS	H. Power	N.	N.	15 2 75	3 9 74	Curette	V. = 1.00. Count fingers at 1'.
S. Hackett	14	S	B. J. Vernon	..	N.	..	27 10 74	Curette
<i>Six Patients with Traumatic Cataract.</i>													
Shadrack Morgan	48	S	B. J. Vernon	G.	..	27 1 74	..	N.	..	3 3 74	Reads his newspaper well with + 2. Injury caused by hot cinder.
Wm. Sapsed	11	S	H. Power	Curette	..	31 3 74	V. = fairly good, 2/80. Injury caused by awl being driven in eye.
Robt. Block	20	S	B. J. Vernon	..	Extraction.	..	— 69	..	Capsule	..	3 4 74
Joseph French	11	F	H. Power	N.	..	28 4 74	V. = light. Injury caused by rupture of cornea and sclerotic. Prolapse iris from a handle of whip.
Jas. Mullen	32	S	B. J. Vernon	..	G.	..	28 4 74	+ 3.5 = good vision. A blow on eye ten years ago.
Walter Sayers	30	F	B. J. Vernon	..	Gibson's linear extraction.	..	16 12 74	Excision	Traumatic cataract from iridectomy for retino-choroiditis (at Guy's). + 2 = Sn. V. at 6". Injury. Gunshot wound, No. 6 shot. Sympathetic irritation of R. E.

F. = Failure. S. = Success. N. = Needle operation. G. = Gräfe's operation. R. E. = Right eye. L. E. = Left eye. V. = Vision. Sn. = Shellen's test types. 2/3 = Shellen's type, 20 at 20 feet. + 2 = Convex lens of 2-inch focus. 1' = One foot. 6" = Six inches.

TABLE VI.—YEAR 1875.
Seven Patients with Senile Cataract.

Name.	Age.	Result.	Operator.	Nature of Operation, R. E.	Nature of Operation, L. E.	Date of Operation, R. E.	Date of Operation, L. E.	Further Operation, R. E.	Further Operation, L. E.	Date of further Operation, R. E.	Date of further Operation, L. E.	Result, R. E.	Result, L. E.
Jas. Crosby	67	S	H. Power	...	Flap.	...	3 4 75	Not operated on. Immature cataract.	V. = $\frac{1}{16}$ in. + 3 5 = $\frac{4}{5}$. Fingers at 3'. Some capsule left behind. Patient would not return for further operation.
Thomas Warren	67	F	H. Power	...	Gibson's lin. ext.	...	4 5 75	Sloughing of cornea. Patient had taken calomel for years.
Sarah Thompson	73	S	B. J. Vernon	G.	...	20 7 75	...	N.	...	10 8 76	...	V. + 3 5 = $\frac{5}{8}$. + 3 = Sn. 5 at 8'.	
James Hotchkiss	43	S	B. J. Vernon	...	G.	...	27 7 75	Operated upon for traumatic cataract, May 1874.	V. = $\frac{5}{16}$ in. Left-eye cataract incomplete. V. + 3 2 3 = $\frac{15}{16}$. + 2 5 = Sn. 1 1/2 at 12'. Does his work well without glasses.
T. Ramsden	60	S	H. Power	...	G.	...	21 9 75	V. = $\frac{4}{20}$. No vision taken. Some capsule said to be in pupil.
Pat. Tighe	72	S	B. J. Vernon	G.	...	6 11 75	
Joseph Cheetham	48	S	B. J. Vernon	G.	...	7 12 75	V. = fingers at 6'. Did not return for glasses. Incomplete cataract.	

Five Patients with Congenital Cataract.

Wm. Wisby	4	SS	B. J. Vernon	N.	N.	10 3 75	13 4 75	Curette.	27 4 75	27 4 75	4 75	With + 3-25 distinguishes objects at 12'.	Recognizes objects at 12' with + 3 25. Sees to eat his food.
Jas. Lemon	12	SF	H. Power	N.	N.	20 4 75	20 4 75	V. = $\frac{4}{16}$ with glass (not stated what No.)	Pupil occluded by adhesion, &c. Numerous operations on both eyes before satisfactory result could be obtained.
William T. Hawkins	2	S	H. Power	N.	...	20 7 75	No result given, but pupil said to be quite clear.	
Samuel Brown	23	S	B. J. Vernon	N.	...	16 11 75	...	Curette.	...	23 11 75	...	V. = fingers at 12" and $\frac{5}{16}$. Eye too irritable for glasses. Left eye normal.	
Isabella Irvine	28	S	B. J. Vernon	N.	...	10 8 75	...	Linear extraction.	...	14 8 75	...	No result given except a clear pupil. Patient to return, but did not.	

Eight Patients with Traumatic Cataract.

Geo. Pullen	23	S	H. Power	N.	...	19	1 75	V. = fingers at 2'. Would not have capsule touched again, being quite content with his good condition. V. L. E. = $\frac{20}{80}$. Injury caused by piece of steel flying off from a chisel. Lens and iris injured.
Henry Jones	4	S	H. Power	...	N.	...	2 3 75	...	N. Cannula forceps	...	15 3 75	6 4 75	V. = fingers at 1 1/2'. Injury from a plate thrown at him. Lens matter opaque. Posterior synechia.
W. Backman	12	F	B. J. Vernon	...	Suction.	...	23 3 75	Injury caused by a fork entering eye. Eye went to bad from irido choroiditis. V. = perception of light.
Jas. Turner	23	S	B. J. Vernon	...	Gibson's linear extraction.	...	25 4 75	Injury caused by a chip of iron striking eye. Wound of cornea and lens. V. L. E. not given. Some capsule in centre of pupil. Patient refused to have a needle operation performed.
Archibald Palmer	10	S	B. J. Vernon	...	N. and curette.	...	24 8 75	Injury from a thorn entering eye four months previous. V. = $\frac{30}{80}$. + 3 = $\frac{20}{80}$.
Charles Claridge	15	F	B. J. Vernon	...	Capsule operation.	...	22 12 75	Old injury from stick striking eye. Prolapse of iris day after operation. Ultimately excision on account of sympathetic ophthalmia.
Mercy Sheather	80	S	B. J. Vernon	...	Extracted by curette.	...	21 12 75	...	N.	...	18 1 76	4 1 76	A traumatic cataract from iridectomy for choroiditis. V. = fingers at 2'. + 3 75 = $\frac{20}{80}$. + 2 75 = Sn. 5 at 12".
Harriet Brisendon	19	S	B. J. Vernon	Suction.	...	21 12 75	...	N. N.	4 1 76	18 1 76	V. + 2 75 = $\frac{20}{80}$. + 2 25 Sn. 3 at 10". Injury caused by scissors entering eye.

F. = Failure. S. = Success. N. = Needle operation. G. = Gräfe's operation. R. E. = Right eye. L. E. = Left eye. Sn. = Snellen's test types. V. = Vision. 10' = Ten inches. 2' = Two feet. x 3 75 = Convex lens of 3 75-inch focus.

TABLE VII.—YEAR 1876.
Seventeen Patients with Senile Cataract.

Name.	Age.	Result.	Operator.	Nature of R. E.	Nature of L. E.	Date of R. E. Operation.	Date of L. E. Operation.	Further R. E. Operation.	Further L. E. Operation.	Date of further R. E. Operation.	Date of further L. E. Operation.	Result, R. E.	Result, L. E.
Chas. Catten	61	S S	H. Power	G.	G.	14 3 76	— 8 75	Eye too irritable for vision.	V. = Sn. 1½ with + 2 75 at 13½°.
M. Wolsh	65	S S	H. Power	G.	G.	23 3 76	— 7 75	V. = ⅓ with + 3.5. Sn. 2½ with 2.5.	V. = ⅓ with + 3.5. Sn. 2½ with 2.5.
M. Hubbard	63	F	B. J. Vernon	...	G.	...	4 4 76	Suppurative iridis.
Mary Clapperton	60	S S	H. Power	G.	G.	11 4 76	6 6 76	V. = Large objects.	V. = Large objects. Too irritable to try glasses.
M. Smith	72	S	B. J. Vernon	G.	...	11 4 76	With 3.5 does fine work. V. = ⅔ with + 3 75.	V. = ⅔ with + 3 75.
M. Levett	48	S S	H. Power	G. (downwards.)	G.	18 4 76	— 10 74	V. = with + 2.5. Fingers at 18°.	Sn. 3½ at 8° with 2.5.
R. Smith	54	S S	H. Power	G.	G.	20 4 76	30 5 76	V. = ⅔.	V. = ⅔.
Win. Nudds	63	S S	H. Power	G.	G.	20 4 76	30 5 76	Fingers at 18°, Sn. 3½ with 2.5. Cornea opaque from old lime injury.	Sn. 4 with 2.25. (Sn. 1½ with 2.5; ⅔ with 3.5.) 25/7/76.
Jane Tookey	65	S S	H. Power	G.	G.	— 1 76	0 11 75	4 5 76	4 5 76	V. = ⅓ with + 3.75.	V. = ⅓ with + 3.75. + 2.5 = Sn. 4.
Eliz. Owen	54	S	H. Power	...	G.	...	11 5 76	V. very good.
Justino Sylvestre	56	F	H. Power	...	G.	...	11 5 76	V. = Bare perception of light. Suppurative iridis.

Mary Ann Page	59	S	H. Power	G.	...	11	5	76	Large objects with + 3.5. Eye irritable (Iris).	...	V. = $\frac{3}{8}$ with + 3.5. Reads Sn. 2 at 12" with + 2.5. (Sn. 1 $\frac{1}{2}$ with 2.5. 6/76)
T. Thomas	60	S S	H. Power	G.	Lieb.	11	5	76	—	72	...	V. = $\frac{3}{8}$ with + 3.5. Reads Sn. 1 $\frac{1}{2}$ at 12" with + 2.5.	...	V. = $\frac{3}{8}$ with + 2.75. V. very good. (Sn. 1 $\frac{1}{2}$ + 2.75. 11/7/76.)
M. Belville	64	S	H. Power	...	G.	16	5	76	V. good. Eyes irritable.
H. Shulver	55	S S	H. Power	G.	G.	16	5	76	—	75	...	16	5	76
Jane Moore	78	S	H. Power	...	G.	21	3	76	...	16	5	76
E. Rowley	56	S	B. J. Vernon	...	G.	16	5	76	Count fingers at 1'. Reads Sn. 3 $\frac{1}{2}$ at 10" with + 2.5. Went out early.

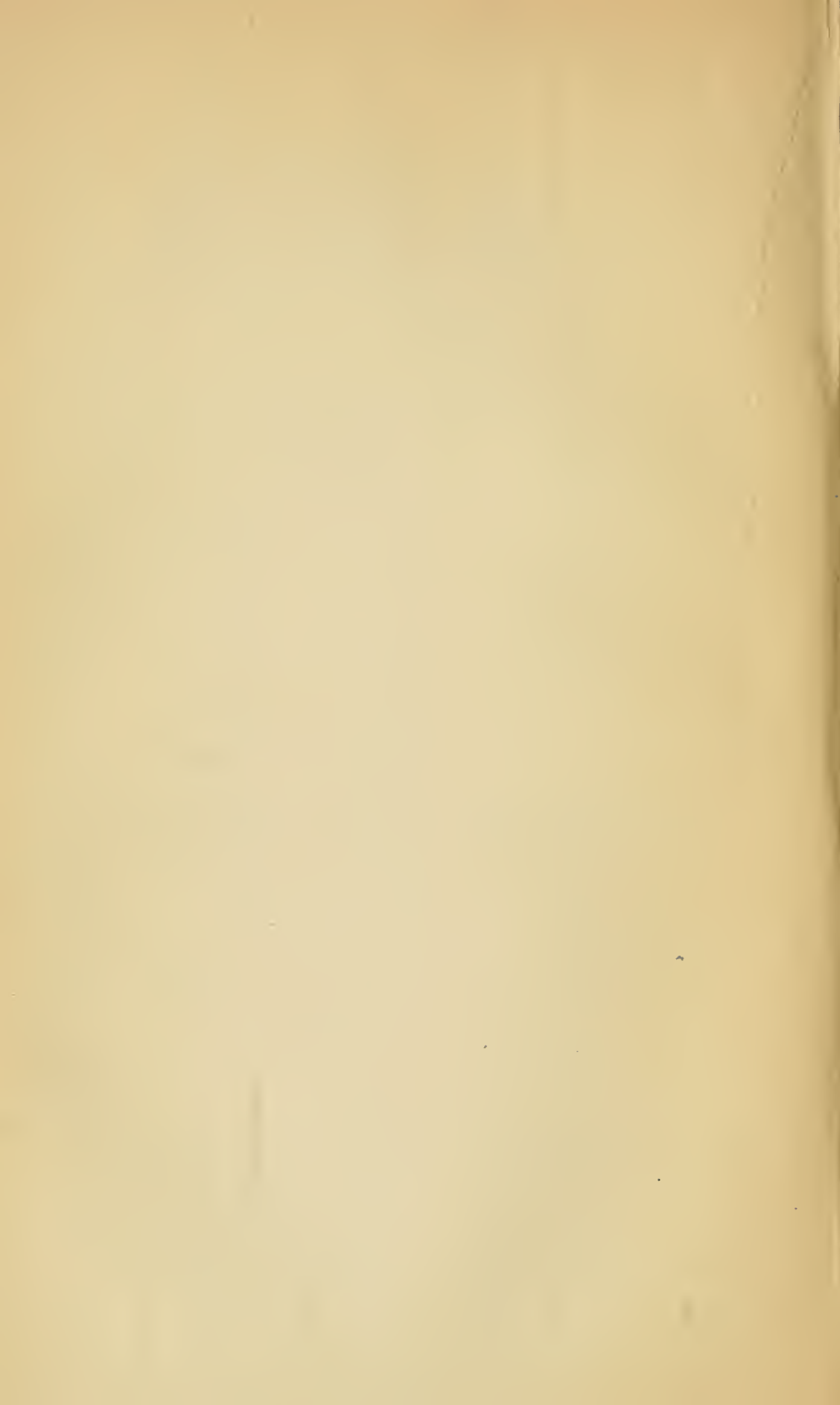
Three Patients with Congenital Cataract.

Kate Green	2 $\frac{1}{2}$	S S	B. J. Vernon	N.	N.	18	1	76	18	1	76	27	1	76	27	1	76	V. improved.	V. much improved.	
Wm. Judge	13	S	H. Power	...	C.	...	29	2	76	Suction	...	14	3	76	V. = fingers at 10".	V. Sn. 3 $\frac{1}{2}$ at 9" with + 2.75 ; with + 3.75 = $\frac{3}{8}$.	
T. Burton	7	S S	H. Power	N.	N.	—	2	76	—	2	76	...	11	5	76	11	5	76	V. very good.	V. (not able to read) very good.

Two Patients with Traumatic Cataract.

John White	18	S	H. Power	...	N.	...	28	3	76	Linear extraction.	7	3	76	7	3	76	V. = $\frac{3}{8}$ with + 3.5. Sn. 3 $\frac{1}{2}$ at 8" with + 3. Injury caused by a piece of wood flying off when cutting wood.
Emma Wells	10	S	H. Power	...	N.	...	21	3	76	11	4	76	V. = $\frac{1}{16}$, with + 3.5 = $\frac{3}{8}$. Caused by child running a pen into her eye.	

F. = Failure, S. = Success, G. = Gräfe's modified linear operation, L. = Liebreich's, N. = Needle operation for cataract. L. E. = Left eye, V. = Vision, Sn. = Snellen's test types, 10" = Ten feet, 10' = Ten inches, R. E. = Right eye.



MEDICAL OPHTHALMOSCOPY.

BY

JAMES KINGSTON BARTON.

In spite of all that has been written and lectured, the ophthalmoscope is still almost entirely left in the hands of ophthalmic surgeons, whereas there is not the slightest doubt but that every physician, and surgeon too, ought to be well acquainted with its practice and use. It is not that the ophthalmoscope is a marvel for diagnosis without other aid, but that it is a most essential help to diagnosis in numerous diseases, intracranial and general. It is useful also in those not infrequent cases of loss of sight, total or partial, after injuries to the head. The great mistake in learning the use of the ophthalmoscope, as indeed of all the scopes and meters, is that it is put off to the end of the student's career. He then begins to practise it in the medical wards on diseased conditions, instead of having become an adept, by overcoming its little difficulties on healthy eyes first. One difficulty in the ophthalmoscope is the bringing the retina and fundus into exact focus, which is only done by an unconscious working together of the examiner's hand and head, and this harmony of movement is only learned by some weeks' constant practice. If healthy eyes were always examined at first, the appearances of the normal fundus would become so engrafted upon the mind that deviations from the normal standard would be at once recognised. Again, the two fundi are alike, but all those differences which are not morbid but congenital would be noted. For instance, a man would not jump to the conclusion that because there was a patch of pigment on one of the borders of the optic disc, there must necessarily have been some previous inflammation, for it is found that in almost all eyes there is some pigment around the disc, either as a ring or as an arc, generally at outer side. One of the chief signs of past neuritis

is disturbance of pigment around the disc ; but in these cases there are always the other signs of this affection—viz., pale disc depressed from surface, arteries attenuated, irregular margins, and the vision of the patient is sure to be affected to a more or less degree.

For examining patients it is always advisable to use the same light, as one can then better appreciate differences in colour and tint of the disc and fundus ; for the same disc may look pale by one light and hyperæmic by another. It is also advisable to examine at first (with reference to one accustomed to the ophthalmoscope) without atropine, and if one is then led to think that there may be some changes in the periphery of fundus, it is necessary to drop in some weak solution of atropine (one which will dilate the pupil without paralysing the power of accommodation, as atrop. sulph. gr. ss., aquæ ad ʒij.) ; for if a stronger solution is used, the patient is left unable to use his eye for reading for several days. Before examining any fundus, the eye should be examined with the oblique light—*i.e.*, the light is held to the side of the eye and a 2-inch lens is placed between the light and the eye, thus brilliantly illuminating the anterior media of the eye. Thus any opacities in cornea or lens which might interfere with the examination of fundus are detected. The right eye should always examine the right eye of patient, and the left the left eye. Before beginning to use the ophthalmoscope it is just as well to get up the theoretical optics of the subject, as this simplifies the practice very much. The optics are written upon at length in most text-books.

In the medical wards of a large hospital like St. Bartholomew's there is an unlimited scope for ophthalmoscopic practice. The diseases in which it is really most useful for diagnosis are many.

First the large class of cerebral affections. Changes in the optic nerve are more often occasioned by the coarse diseases of the brain rather than the fine. For instance, in cerebral tumours of all kinds, or anything that may alter the intracranial blood-pressure, the anatomy of the optic nerve at once reveals how such small alterations in pressure of fluids within the cranium will affect its structure. The nerve almost entirely fills up the bony canal in the sphenoid, through which it passes. A sheath of arachnoid encircles the nerve down to its exit from the canal. From the first principles of hydrostatics it is easy to understand how the nerve may be compressed within this bony canal by any cause which lessens the arachnoid cavity, such as any intracranial tumour, whether solid or fluid. Again, in inflammation of the meninges of the brain, the inflammation may spread down the sheath of the nerve, thus causing pressure. The effects of

this pressure are to produce squeezing of the vessels in the optic nerve, and of the ophthalmic artery itself—that is, stagnation of blood in the veins, the blood not returning quick enough, and a total or partial obliteration of the arteries, sufficient blood not passing through them. Exudation next follows; the nerve-tubules are then squeezed, and if the morbid condition causing all this does not cease, the final result will be atrophy of these tubules, and consequent impairment of the sense of vision. The changes that the ophthalmoscope reveals are—the optic disc is hazy and indistinct; its margins shade off imperceptibly; its surface is raised, woolly in aspect, pink in colour. The retinal veins are enlarged and tortuous. The arteries are small. (The greater the amount of pressure, the smaller the arteries.) There is an increase in the number of small vessels which spring from around the margins of disc. The retina may become affected, and if so, it is found to be hazy, with striations radiating from the disc. The vision of the patient is generally impaired, sometimes to a considerable extent; but at other times, if the extent of the neuritis is only small, the patient does not complain of any loss of vision.

Another cause of similar changes in the optic nerve is anything pressing upon the cavernous sinus, and thus interfering with the return of blood from the retinal and other ophthalmic veins.

If the cause of the neuritis be only temporary, the condition of the nerve recedes; and if it has not been of any great extent, the nerve-tubules may have escaped any serious pressure, and consequently no atrophy or loss of vision ensues. It leaves its marks, however, which can be recognised by the ophthalmoscope. There is a slight irregularity of margins of disc, with disturbances of pigment here and there around it. The surface of disc will be a little paler than natural. The arteries will look rather small. For all ordinary purposes the patient will not complain of any loss of vision, but if the vision is carefully tested with some types, it will be found to be slightly deficient. If, on the other hand, the neuritis has been severe, or its cause is permanent, the nerve-tubules become pressed upon, and gradually atrophy. Connective tissue is developed from the exudation, which also, in contracting, injures the nerve-tissue. The atrophy of the optic nerve thus produced is at once recognised under the ophthalmoscope. The disc becomes quite white (after a long period it presents a bluish-grey colour), due to the lamina cribrosa showing the perforations where nerve-tubules had passed through. The surface of disc is depressed. The arteries (retinal) are mere threads, hardly recognisable. The veins are

also small, but look large as compared with the arteries. Vision is impaired often to a very great extent. The margins of the disc are very irregular, with great disturbances of pigment around it. The choroid will be generally found atrophied. The pupil is usually dilated. If there is complete blindness in one eye, but partial vision in the other, when light is thrown into the good eye, the pupil of blind eye will contract in harmony with the other (due to reflex action).

There is another form of atrophy of the optic nerve, which goes by the name of 'primary white atrophy.' Its ophthalmoscopic appearances are—the disc is quite white and small, but its margins are clean cut, and there is no particular disturbance of pigment around it. The vessels are extremely small. From the appearances of this disc it is at once evident that no inflammatory changes have taken place around it. It is usually found in some general cerebral disease, such as is found in drunkards and excessive smokers, or after a blow upon the head. In fact, this form of atrophy is that which follows the finer morbid changes in brain-tissue.

With regard to those cases of atrophy following a blow on the head, they are most interesting. Not infrequently a man receives a blow on the head, and after the immediate effect has passed off he complains of nothing. However, some days or weeks after, he begins to notice that his vision is misty. If the optic discs are examined when he first complains, they are noticed to be hyperæmic or slightly woolly. Later on they become pale. The changes seen in the eye are not of a very active kind; as, for instance, is seen in the congested disc from cerebral tumour. There were two interesting cases in Rahere Ward lately. One man received a blow on the head, and suffered from symptoms of concussion for several days. His discs, a week after, were hyperæmic, with slightly indistinct surfaces and margins, but sufficiently distinct to be recognised. Another man came into Rahere the same week, having fallen down in a fit and struck his head. His optic discs presented the signs of active neuritis. The man died a few days after, but no actual cause for the neuritis could be found.

It is very difficult to make out the cause of this neuritis after injuries to the head. The blow may cause a hæmorrhage somewhere along the optic tract which would cause pressure, or a hæmorrhage into the optic canal in the sphenoid. Again, the blow may have some effect on the sympathetic centre, causing paralysis of some of the branches supplying the blood-vessels of the optic nerve, causing dilatation of these small vessels. Post mortems up to the present have not revealed much, but there is no doubt that

the examination of the brain, and especially the optic tract, commissure, and optic canal, should be most carefully made to see if any clots or lesion could be found.

If a patient were suffering from symptoms which pointed to a probability of cerebral tumour, and on examining with the ophthalmoscope changes in the optic nerve were found as described above, an additional evidence would be found in favour of a tumour. If the history of the patient revealed syphilis, or, what is of more use, if there were any signs of syphilis about the body, the probability would be that there was a syphilitic gumma within the cranium. There is perhaps no part of the body which shows past traces of syphilis more positively than the eye. Syphilis leaves its marks either in the form of adhesions of the iris to lens, from past iritis, generally in both eyes, or a hazy vitreous from choroiditis, or changes in the choroid showing old choroiditis, such as patches of disturbed pigment in various parts, and very often about the region of the yellow spot. If the choroiditis has been excessive, it would lead to atrophy, and patches of white sclerotic would show through. If retinitis or neuritis had occurred, their signs would be apparent. A case particularly illustrating this was in Luke Ward lately. A young man had hemiplegia. He had no morbus cordis, or any signs of diseased vessels. He at first denied having had syphilis. On examining his eyes, old adhesions of the iris were seen in both eyes. The vitreous was hazy, so hazy that details of fundus could not be at all clearly seen. From his description of the mode of attack of inflammation (one eye affected several weeks after the other, and not much pain) in his eye, and from the appearances of the eye at present, it could be safely asserted that he was suffering from syphilis. He afterwards confessed to having had syphilis. His hemiplegia might thus be put down to some syphilitic lesion. In any case where one cannot get a syphilitic history, a careful examination of the eyes should be made, as some of its marks may very likely be there. Of course the eyes do not become affected in every case of syphilis.

Cases of tubercular meningitis supply another class of most interesting examinations. Neuritis often supervenes, and, curiously, it seems to do so at two different periods. In some cases the neuritis comes on and leads to white atrophy of optic nerve before the cerebral symptoms appear. In others the neuritis only shows itself just previous to the breaking out of the cerebral symptoms, or just after their appearance. There have been several interesting cases of this kind in the wards. Two children—one (a boy) who was in Pitcairn, and one (a girl)

in Stanley—both had disease of spine and affections of joints. The boy became gradually blind, and on examination white atrophy of both optic discs was seen; he eventually died of tubercular meningitis. The girl was not examined till about twenty-four hours after the appearance of cerebral symptoms, but both her discs were found to be atrophied, showing previous changes. Two other children were in Hope Ward lately. One was brought in with cerebral symptoms, but no disease of joints or spine. The pupils were dilated and fixed, which is usually the case in the later stages of this disease; the optic nerves were both swollen and hyperæmic, presenting active changes. The child died, diagnosis correct, and the eyes were saved for microscopical section. No tubercles were found in the tunics of the eye. Another child, at present in Hope, has tubercular disease of the lung. Lately she has been getting drowsy, and on examining the eyes the optic discs were found to present the signs of neuritis. The prognosis is bad, and it remains to be seen whether the child will die of a cerebral affection.

Another set of cases in which changes in the eye are often found are patients suffering from Bright's disease. Changes are more often seen in those who have the small granular kidney. Patches of effusion occur along the course of the vessels in the retina, generally near the disc; retinitis follows at these spots, to be seen as white shining patches of effusion.

Several of these glistening patches are seen along the course of the vessels in the fundus. In patients with the large pale kidney, retinitis with larger amount of effusion occurs. There has been a very interesting case of this kind in Faith Ward during the last few months. The woman in the course of a short time became quite blind (she was suffering from albuminuria, the diagnosis being a large pale kidney). On examining with the ophthalmoscope in the direct method, a silver-grey reflex instead of the ordinary red colour. This was seen to be due to detachment of the retina, which was bulging forward quite up to posterior part of lens. The detachment could be seen to be chiefly of the lower part of retina in both eyes. She had no pain. Vision was nil. Blisters were applied to the temples. She gradually began to recover vision after a few weeks' total blindness. In about a month afterwards the detachment of retina had considerably diminished, it being chiefly confined to lower part of fundus. It was plain also that it was the bulging up of lower part that obstructed the view of upper portion of retina. If the whole retina had been detached in the manner that the lower part was, vision would have probably never been restored. The lower portion will no doubt be

seriously damaged for vision, but the upper is apparently not much affected. When the fundus could be illuminated, it could be seen that there had been general retinitis and neuritis. The effusions have steadily diminished, and vision improved so much that she can read. It is not often one sees so much effusion in albuminuric patients, as it is generally a neuro-retinitis with effusions at various parts.

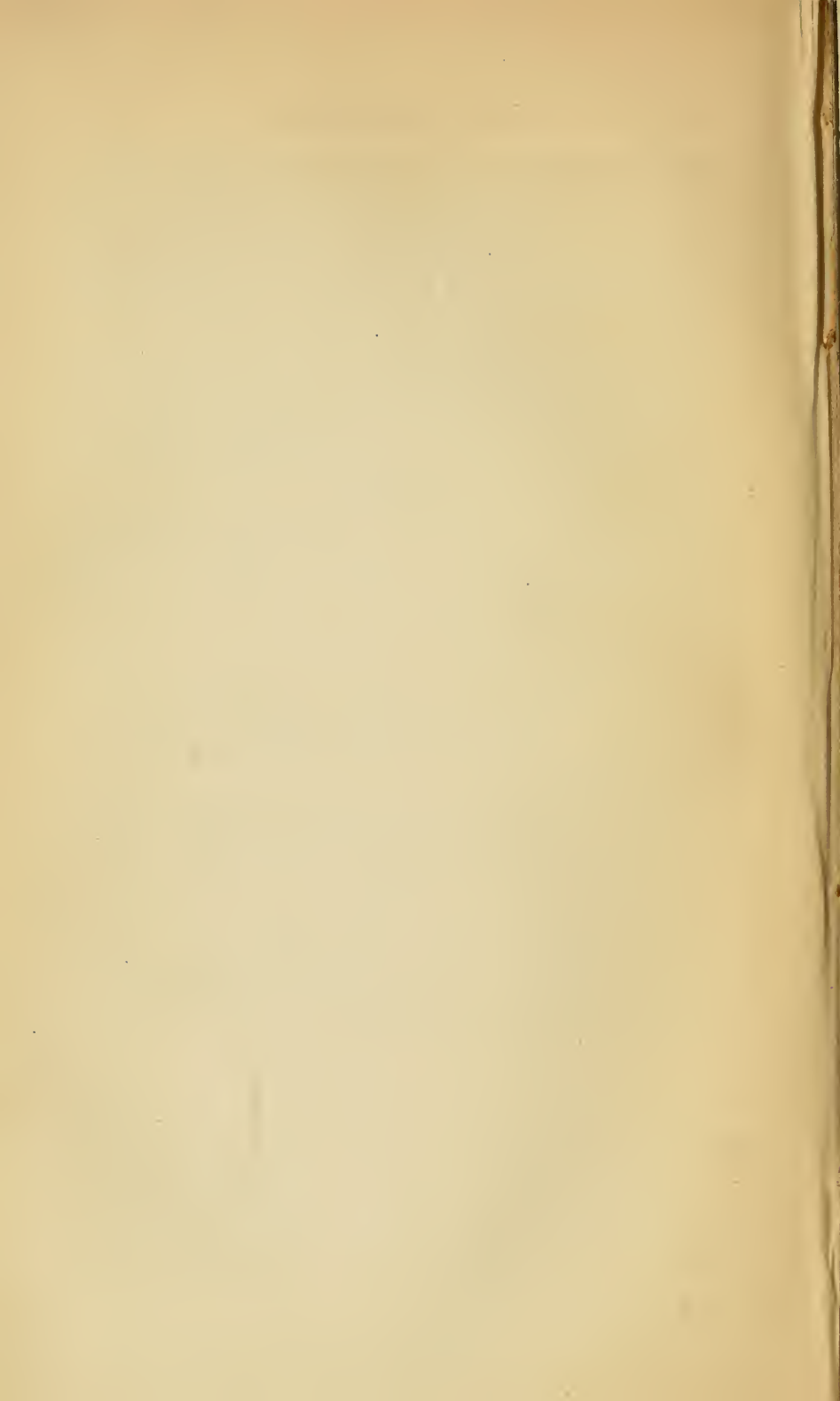
There is a patient at present in Matthew Ward with albuminuria (large pale kidney) who is nearly blind of one eye. With the ophthalmoscope it is seen that there has been past neuritis and retinitis. There have been similar changes in the apparently sound eye, but not to such an extent. The small white patches scattered about fundus are the most characteristic changes left by retinitis in albuminuria.

In locomotor ataxy changes in the eye are often amongst the earliest symptoms. The ocular muscles are often the first to suffer, at least they become irregular in their action. The patients complain of double images, caused by the muscles of the eyes not acting together. There was a man in Luke lately with locomotor ataxy. On examining him one day, he had external squint of the left eye; a few days after, he had external squint of both eyes. Afterwards the eyes recovered their proper position. Any very marked changes of the optic nerves are not often seen in this disease. The disc often becomes slightly hyperæmic, and eventually pale. There have been several cases in the Hospital showing the latter change.

Amongst the other general diseases in which changes in the eye are said to occur may be mentioned leucocythæmia. There have been two cases in the Hospital lately, but neither presented any alterations from the normal fundus.

There has been a case of Addison's disease in Mark Ward; the choroid presented extreme pigmentation.

Amongst the insane would be found another large class of interesting cases for the ophthalmoscope. To any one who has once taken up the ophthalmoscope, it is always a source of great interest as well as of great use.



ABDOMINAL SECTION IN A CASE OF RUPTURED BLADDER.

BY

ALFRED WILLETT.

T. F., aged 48, admitted into St. Bartholomew's Hospital at 1.30 P.M. on Sunday, June 11, 1876, with symptoms pointing to ruptured bladder.

History.—Was in perfect health until 9.30 A.M., when a man with whom he had quarrelled came to the house where he lived, aroused him, and challenged him to go down and fight him. Simply pulling on his trousers, he at once went down, and the contest began. His antagonist being the better man, quickly threw him, and whilst on the ground he was kicked over the pubes. Instantly being seized with acute pain, he was helped up and taken into his house. He now became very faint, and had urgent desire to pass water; but although he made repeated attempts, he failed to pass any. His pain, distress, and collapse increasing, he was brought to the Hospital four and a half hours after the injury.

On admission he complained of intense pain in the hypogastrium, but the whole abdomen was tender on pressure. His skin was cold, countenance anxious, breathing hurried and thoracic, pulse small and feeble, tongue dry and brown. There was no bruise or mark of injury visible. Replying to questions, he stated most positively that the last time he passed urine was at eleven o'clock the previous night, and that he knew his bladder was full when he went down to fight, but that, being enraged, he hurried, and did not stop to empty it. He also said that when he was kicked he felt something give way in his abdomen.

The house-surgeon, Mr. Edwards, at once introduced a catheter,

and drew off about 8 ounces of blood and urine, the last 2 ounces consisting almost entirely of bright arterial blood.

At 3.30 P.M. I saw the patient, and arrived at the conclusion he had sustained rupture of the fundus of the bladder, including the peritoneal coat, and consequent effusion of urine into the general cavity of the peritoneum.

Regarding the issue, if the patient were left alone, as inevitably a fatal one, I resolved to wait twenty-four hours; and should the train of symptoms tend to confirm my opinion, with the concurrence of my colleagues I would propose to the patient an operation as the only chance of saving his life.

I left directions that the patient should be given Ext. Opii Liq. m. x. every two hours, and that his urine should be drawn off every few hours.

6 P.M.—Pain increasing; abdomen becoming more tense and tympanitic. Four ounces of urine and blood drawn off. Temperature, 99.5° . Respirations, 42. Pulse, 96.

11 P.M.—Has not been sick, and is in less pain. Says he wants to go to stool; nothing has passed. About same quantity of urine drawn off, but with only a trace of blood. There were no drops of blood at the end, as had been noticed before.

June 12, 5 A.M.—Has slept for less than one hour. Complains of more pain in abdomen, and since 3 P.M. sickness and retching have come on. His abdomen is more distended. No flatus has been passed. Four ounces of urine quite free from blood drawn off.

9 A.M.—Symptoms of intestinal obstruction increasing. Temperature, 99.8° . Respirations, 28, sighing. Pulse, 88, full and regular. Urine drawn off; remains quite clear.

1.30 P.M.—He was now seen in consultation by several members of the staff. An examination per rectum was made, but no tumour, or indeed anything abnormal, discovered. A full-sized metallic catheter was introduced cautiously, and the interior of the bladder explored with its point; but beyond that it passed at once up to the hilt, and its point moved readily in all directions, no direct evidence of injury to the bladder was obtained. Certainly no distinct sensation was felt of its passing through a rent in the bladder. When, however, the urine was drawn off, it was found to be of a uniform deep chocolate colour, and it was observed to flow with a marked interrupted stream as the patient breathed. Ten to twelve ounces at least of this fluid were drawn off, and more might have been withdrawn. All his symptoms had been hourly increasing in intensity. My colleagues almost unanimously agreed that the patient's history and symptoms pointed conclusively to his having sustained a rupture of the

bladder, with escape of urine into the peritoneum ; that general peritonitis had set in ; and that the symptoms of obstruction were due probably to paralysis of the muscular coat of the intestine, and consequent arrest of peristaltic action, but might be dependent upon a coil of intestine slipping into the rent of the bladder and becoming strangulated. An opinion as unanimous was expressed that the patient must die if left alone, whilst a majority agreed that an operation, laying open the abdomen, sewing up the rent in the bladder, and cleansing the peritoneum of the bloody urine, afforded the patient the only chance of recovery. On informing him of his condition and prospects, he willingly assented to its performance, and at about 2.30 P.M. I commenced the following operation : An incision, some 5 to 6 inches in length from the umbilicus to the pubes, was made in the mesial line, and carried through the parietes. All bleeding points having been secured, the peritoneum was opened. At once several ounces of dull brownish fluid, with strong urinous odour, escaped. The intestines were greatly distended, and instantly bulged out through the wound. The peritoneal coat of the intestine, as well as the peritoneum generally, was highly injected, and adjacent surfaces were glued together. Passing my hand into the pelvis, I detected a laceration of the bladder. The coils of gut were only slightly more adherent here than in the abdomen proper, whilst I satisfied myself there was no protrusion of bowel into the lacerated bladder. The omentum was raised from off the intestines, and so much of the latter as lay in the pelvis were drawn up, laid upon the upper part of the patient's abdomen, and protected from harm and chill by flannels rung out of moderately hot water. There was about half a pint of the bloody urinous fluid in the pelvis, and when this had been sponged away, a rent of the bladder some $3\frac{1}{2}$ inches in extent was exposed ; it extended diagonally across the fundus, having a direction from before backwards, and from right to left. The appearance was that of a nearly straight tear through all the coats of the bladder, except at its most dependent part, where it was jagged and uneven. The bladder was flaccid, but of course quite empty, and at the site of rupture its walls were fully half an inch in thickness. I brought the torn edges easily into apposition, and united them by eight interrupted sutures of fine Chinese silk. The sutures were placed at intervals of rather less than half an inch, and seemed to close the rent completely. Before returning the intestines, I cleaned out the abdomen as thoroughly as I was able ; but the mesentery of the gut lying outside the abdomen, acted as a transverse diaphragm, and I was much disappointed to find, on replacing these coils,

that some of the fluid had been pent up above it. Owing to gaseous distension, very considerable difficulty was experienced in replacing all the intestines within the abdomen, and I was quite unable to introduce my hand and cleanse the upper part of the peritoneal cavity so satisfactorily as I could have wished; but the patient's shoulders were raised, in order to make the pelvis more dependent, and all fluid that found its way there removed. The intestines that had been lying out of the abdomen during the operation were sponged over with warm water and carefully cleansed before returning them. So extreme was their distension, that to enable me to introduce sutures and close the external wound, Mr. Langton, who assisted me, was obliged to spread out his hand and restrain the bowels from forcing their way through the wound, withdrawing his hand gradually as the successive sutures, also of Chinese silk, were tightened. Through the lower angle of the abdominal wound I passed a carbolised drainage-tube into the pelvis, securing it to the edge of the external wound, which was then dressed precisely as after ovariotomy. A Thompson's catheter was introduced and retained in the bladder. On being replaced in bed, hot bottles were placed beside him, and he was well covered up.

6 P.M.—Free from pain, and in a profuse perspiration. No urine as yet passed. Has been given an enema of Ext. Opii Liq. ʒss. in starch. Temperature, 99.2°. Pulse, 120. Respirations, 23. Directed to suck ice, and take iced milk in small quantities.

10 P.M.—Skin warm, and freely perspiring; no sickness; expression tranquil. One and a half ounces of blood-stained urine escaped through catheter, but has not got rid of any flatus. Says he feels much better than before the operation. Temperature, 104°. Pulse, 128. Respirations, 27, and more deeply drawn. Tongue moist, coated. To have Ext. Opii Liq. m. xx. per rectum.

June 13, 8 P.M.—Has vomited twice, bringing up in all about a pint of darkish watery fluid. Slept at intervals for nearly three hours. About one ounce of clearer urine has come through the catheter, but complains that he wants to make water. Has passed flatus per rectum. Says he is in pain over the abdomen, and feels sick. Is getting restless. He was given Ext. Opii Liq. m. xx. twice during night. Temperature, 104.2°. Pulse, 120, regular, volume good. Respirations, 24, more shallow. Skin perspiring, but the surface is less warm to touch.

11.30 A.M.—Is rather drowsy and face dusky. One and a half ounces of much clearer urine passed, and some slight discharge from drainage-tube. Temperature, 105°. Pulse, 156, small volume and very weak. Respirations, 36, thoracic. Skin clammy. Brandy ordered to be given freely.

1 P.M.—Died rather suddenly after vomiting a large quantity of dark fluid.

The following is Mr. Butlin's report of the post-mortem examination:—

Post Mortem.

The wound in the abdominal parietes was adherent almost along its whole line; not much swelling of abdomen.

Wound.—The intestines immediately behind the wound were adherent to it.

Intestines.—All the coils of intestine in the lower half of abdomen were adherent to each other, and to the peritoneal lining of abdomen, by recent lymph. The intestines in contact with the bladder were adherent to it.

There were about 2 ounces of bloody fluid at the back of the peritoneal cavity, about an ounce of this lay just above the bladder.

Bladder.—The opening in the bladder was everywhere well closed except between the posterior two stitches, where there was an orifice through which water injected per urethram escaped very freely. Even here there appeared to be an attempt at repair. Elsewhere the edges of the wound were adherent. There was very little sign of inflammation in the interior of the viscus.

Other organs normal.

Reviewing the case, it appears to me now, as indeed it did at the time I first saw the patient, that the success of such an operation as I performed would in great measure depend upon the promptness with which it was done; but I did not feel myself justified in attempting an operation of so hazardous a character without the concurrence of my colleagues in my views of the nature of the injury, the hopeless condition of the patient as he was, and the possibility of saving his life by operative interference. It was a source of deep regret to me to find I had not effectually closed the bladder wound. At the time all who witnessed the operation agreed in the apparent complete apposition of the edges of the rent. If it should fall to my lot to perform this operation again, I would distend the bladder before closing the abdominal wound, and I would wash out the abdominal cavity more thoroughly. It is worth pointing out the marked contrast in the behaviour of the parietes and intestines that is found to exist in abdominal section when performed in such a case as this and in ovariotomy. Here I found the parietes firm, almost unyielding and so great gaseous distension of the intestines, as to cause

them not only to burst through the wound, but to render it a matter of much difficulty to replace them when I closed it. Under these circumstances it is quite impossible to introduce one's hand and sponge all parts of the abdomen where fluid may be suspected to lie, as is invariably done in ovariectomy. It would, I feel sure, be better to let a stream of warm water run into the abdomen, and trust to a thorough abluition by this means of the deeper parts. It can be scarcely necessary to insist on the necessity of dealing with these cases at once—that is to say, before peritonitis is established—if the patient is to be given the best chance of recovery. This position, however, assumes not only that the operation may be regarded as a legitimate surgical enterprise, but also that an exact diagnosis in these cases can be sometimes formed at once; that the cases are fatal when left alone; and that no other measures have been suggested for dealing with them by a procedure sufficiently comprehensive to warrant the expectation of its proving successful. This case, I contend, proves the possibility by abdominal section of restoring the integrity of the bladder, the operation involving not only exposure of the injured viscus, and the closure by sutures of the torn aperture in it, but also the effectual removal and thorough cleansing of the peritoneal cavity from blood and urine effused into it.

I may be asked whether I consider the patient's life to have been prolonged by the operation. Unhesitatingly I reply no; my opinion would be rather that it was shortened by some twenty-four or more hours, for urine is not the most irritating fluid that may escape into the peritoneum. Neither can I account for the rapidity with which the patient succumbed, unless it were the shock of the operation on a man advanced in years, and the subject of acute peritonitis at the time of operation; for the post mortem showed that the peritonitis was less marked than when the operation was performed.

In an article in vol. ix. of these Reports, headed 'On some Points in the Surgical Treatment of Intra-Peritoneal Injuries,' Mr. Thomas Smith drew attention to the knowledge which we now possess of the behaviour of the peritoneum when surgically interfered with, as the result of the experience obtained in the varying circumstances under which ovariectomy has been performed, and performed with so great an amount of success. Mr. Smith drew from the consideration of these facts the legitimate and almost inevitable deduction, that for the future the impression that to wound surgically the peritoneum is a mortal offence to its inviolability, to be resented swiftly and surely by the death of the patient, must be abandoned, and that in place of the suspicion and dread which shrouded the contemplation of

any surgical procedure that involved the mere mention of the peritoneum, we should treat it rationally, feeling sure it would often prove grateful for timely interference; or, speaking less enigmatically, that bearing in mind that fatal peritonitis is the inevitable result in certain cases of injury to the viscera contained within the abdominal cavity when left alone, it becomes the surgeon's clear duty to interfere, by opening the abdomen with the object if possible of repairing the lesion, and that if this were but successfully accomplished, there would always be a prospect of the patient's recovery; or even when it was found impracticable to secure the desired end, the patient's condition would not be made more hopeless; and lastly, that whenever death ensued, yet still valuable information might be obtained, by ascertaining the cause of failure, or showing how a more exact diagnosis might have been formed. Lastly, Mr. Smith enumerated a number of intra-peritoneal injuries which he considered justified the exploratory operation of abdominal section; and although rupture of the bladder does not appear in the list, yet I claim that it falls within the general description of the class of injuries which are said to warrant the performance of this operation, and I feel I may justly invoke the authority of the arguments contained in Mr. Smith's article in favour of the operation I performed.

But here I may be met with the question, Can it be said that symptoms of ruptured bladder are so defined and constant in their character that we can be so sure what we shall find, as to justify us in undertaking so grave a procedure as abdominal section? All will admit that in speaking of ruptured or lacerated bladder, it is necessary to draw a distinction between cases of which the one I have narrated is an instance, and those in which the bladder is torn in the neighbourhood of the neck, where it is uncovered by peritoneum, and when generally the lesion is complicated by other injuries, such as fractured pelvis. Insisting on this distinction, and desiring to confine my remarks to the former class alone, it seems well to begin with an inquiry as to whether it is possible to arrive at a diagnosis in uncomplicated cases of rupture of the bladder at its fundus, in all of which cases the peritoneal covering is torn, the lesion having as its necessary result effusion of urine into the general cavity of the peritoneum. I believe it is. In all the instances I have seen, the patient has sustained a severe blow or squeeze over the lower part of the abdomen at a time when the bladder was distended, and has at once become unable to pass urine, although having urgent strangury, if anything only a few drops of bloods escaping. If a catheter be introduced shortly

after the occurrence, only blood has been evacuated; although when again used after a few hours, both blood and urine have flowed; and in some cases, after a further interval, clear urine has been drawn off. Whilst these symptoms are referable directly to the bladder, other features in the case will be found pointing as directly to the patient having sustained lesion of an abdominal viscus—viz., collapse, intense pain in the supra-pubic region, general tenderness over the abdomen, and upon this state peritonitis supervenes, commonly fatal within three or four days.

The conjunction of these two sets of symptoms marks unmistakably this injury. In my opinion, evidence of the bladder being distended is the most important fact to ascertain, for it seems impossible that (under the conditions of the cases we are considering) an empty bladder could be ruptured by any violence applied to the abdomen, lying, as it then does, completely protected by the pelvis.

The prognosis in ruptured bladder may be shortly stated to be almost invariably fatal, and of rupture across the fundus I am not aware of the recovery of a single unequivocal case, unless one I shall have occasion to refer to further on deserves to be placed in this category. Mr. Birkett, in the article on injuries to the bladder in Holmes's 'System of Surgery,' records fifty examples, all of which, with the exception of three, terminated fatally; and of the three recoveries, only one had symptoms of extravasation of urine into the peritoneal cavity. Again, Dr. Stephen Smith¹ records seventy-eight cases, with five recoveries; but of these five only three were cases of rupture due to external violence, three were cases of urine effused into the cellular tissue around the neck of the bladder, one was a case of partial rupture, and one was stated to be a case of extravasation into the peritoneal cavity. Professor Gross remarks:² 'Not much can be said about treatment of this lesion. It is obvious no measures, however well directed, will in general be of any avail in saving life.' And again: 'All the mischief that can be done is done in the first instance by the escape of urine into the peritoneal cavity, from which it will be out of the power of the surgeon to remove it, or to prevent its pernicious effects.'

Under the absolutely hopeless circumstances in which surgeons are called upon to treat these cases, we are bound to put into practice any procedure that offers a reasonable chance of success; but before considering whether abdominal

¹ Professor Stephen Smith on 'Rupture of the Bladder,' New York Jour. Med., 1851, vol. vi. p. 374.

² Gross's Surgery, Dis. and Inj. Bladder, 2d ed. 1855, p. 146.

section offers such a prospect, I feel it incumbent upon me to notice a line of treatment first suggested by Dr. Stephen Smith in 1863, which has since been twice put into practice in the United States, with recovery in both cases. The operation is that of lateral lithotomy, as usually performed for stone in the bladder. The first case was under the care of Dr. W. J. Walker of Boston, U.S. The case was one of fracture of the pelvis at the pubic symphysis, with—as it was diagnosed—laceration anteriorly and external to the peritoneum. A large tumour was observed extending from the whole line of Poupart's ligament nearly to the umbilicus; it was elevated $1\frac{1}{2}$ inches above the surface. After a catheter had been introduced and several ounces of urine withdrawn, the tumour referred to disappeared. The bladder was then opened as in the lateral operation for stone; immediate relief followed, and in fifty-five days from the operation the patient resumed his occupation. This case is mentioned by the author of the second case, Dr. Erskine Mason.¹ The facts of Dr. Mason's case were as follows: M. B., 26, Irish, admitted into Roosvelt Hospital, December 26, 1871. He was unable to micturate, but had urgent desire to do so. He had fallen downstairs the previous day. There were small bruises and hurts about face, arms, and legs. A No. 10 catheter was passed, and there was no impediment to its passage, but only a few drops of urine streaked with blood flowed. On December 27, catheter was again passed, and 4 ounces of urine 'with some blood' drawn off. More tenderness over abdomen. 4 P.M., patient voided urine. Dr. Mason says that at this time the diagnosis was not clear between ruptured bladder and injury to kidneys, and it was decided to watch the case. December 28.—A little bloody urine drawn off. Abdomen tympanitic. Legs drawn up. Hiccough and vomiting. Now the diagnosis of rupture of the bladder and general peritonitis was arrived at, and lateral lithotomy performed. Dr. Mason, who performed it, states, 'Under ether I examined him thoroughly per rectum, and convinced myself there was no laceration of the urethra or neck of the bladder around the prostate. Posterior to the prostate, and to the left, a decided tumour was felt, which to my finger communicated the sense of fluctuation. There was no thickening or induration of the tissues anterior to the neck of the bladder. A large-sized staff was now passed into the bladder with the greatest facility, and I laid open the bladder as in the lateral operation for stone.' Bloody urine escaped in quantity. 'Passing my

¹ New York Med. Jour., August 1872, vol. xvi. 1872, July to Dec., p. 113, art. i., 'A Case of Rup. Urin. Blad.; with Remarks on Treat. of this Injury.' By E. Mason, Adjunct Prof., Surgery Univ., New York.

finger,' the author goes on to say, 'into the bladder so as to enlarge the opening, I felt confident I detected a rent in the posterior wall of the viscus; but I did not examine this opening thoroughly, as I feared I might do injury did I pursue investigations further in that direction.' Two facts were observed—one, that the interior of the bladder was sensibly cooler than the surface; the other, the disappearance of the tumour felt through the rectum. The diagnosis now made was '*the rupture had taken place through the posterior wall of the bladder, that the rent had extended through the peritoneal covering, and that the urine had extravasated into the pelvic cavity, but rested chiefly in the posterior cul-de-sac.*' Owing to venous hæmorrhage the wound was tamponed with lint for two days. As regards the after-progress of the case, it was most satisfactory. On second day it was noted '*a brown discoloration is now observed over the inguinal, hypogastric, and peritoneal regions and down the thighs,*' which on third day '*present a hard and indurated feeling,*' and '*patient is bathed in profuse perspiration, which has a strong urinous odour.*' On fourth day it was noted '*discoloration fading, and considerable perspiration, which is of the same strong urinous odour.*' On fifteenth day he was almost convalescent, and his recovery was uninterrupted. Opium was given for a week, yet the bowels acted on the first day, and almost daily. Patient was discharged cured on the thirty-seventh day after the operation. A sound detecting nothing abnormal in the bladder.

Commenting on the success of these two cases, Dr. Mason discusses four operations which have been suggested in cases of ruptured bladder: 1. Opening the bladder above the pubes, as in the high operation for stone, or tapping that viscus here. 2. Tapping the cavity of the pelvis either above the pubes or at the pelvic cul-de-sac per rectum. 3. Peritoneal section. 4. Lateral lithotomy. Dr. Mason considers the first three of the above can only be occasionally serviceable, depending upon the situation and extent of the lesion; the fourth he strongly urges. It was advocated in 1863 by Professor Stephen Smith, in an article on ruptured bladder, who says of it that it seems the most rational plan yet pursued. Whilst Dr. Mason is convinced '*it is one which more fully than any other meets the urgent requirements of the case—viz., the giving vent to extravasated urine, and preventing recurrence of the same—that it is of equal service whether the rupture has taken place either at the anterior or posterior portion of the bladder, both Dr. Walker's and my own fully testify.*' Again, in both cases in which it was tried it was successful; and this is more than can be said of any

other treatment, especially when so complicated as each of these was—one from fracture of the pelvis, and the other from general peritonitis, with extravasation of urine into the pelvic cavity’—the author’s being the only case on record, ‘in which laceration was evidently in the posterior portion of the organ, with extravasation into the pelvic cavity, which recovered through the means of operative interference.’

Probably this procedure has been adopted in similar cases in the States, although I do not find the records of any. But I have never heard of its being practised on this side of the Atlantic; indeed I do not know that it has been recognised. This would be the more singular if the operation had maintained its position, because, without any question, in a condition so wellnigh hopeless as rupture of the bladder, the performance of lateral lithotomy would be looked upon as a very simple affair, if the good results Dr. Mason anticipated in these cases had been confirmed by a more extended experience. In justice to the operation in question, I felt bound to quote Dr. Walker’s case, although from its nature it is one that I had not intended to discuss. With regard to the proposal to open the bladder by the lateral operation in cases of laceration of the bladder about its neck causing extravasation of urine into the pelvic cellular tissue, in connection with fractured pelvis, such as Dr. Walker’s, I am quite ready to admit it possesses at first sight claims for regarding it favourably, yet I must say I see no reason why efficient catheterism, with prompt incisions into the collections of effused urine, should not be equally or more serviceable, especially when, as in Dr. Mason’s case, it may be necessary to tampon the wound for some days; neither can one forget that this particular condition, effusion of urine into the pelvic cellular tissue, is the special fatal risk connected with lateral lithotomy in cases of calculus. With regard to Dr. Mason’s case, I do not consider he has proved his claim to call it a case of rupture of all the coats of the bladder, including the peritoneal, and consequent extravasation of urine into the general peritoneal cavity. For the purpose of producing all the evidence in favour of Dr. Mason’s view of the case, I have reported the case at a length quite unnecessary for my own ends; but feeling I could not acquiesce in Dr. Mason’s view, it seemed but just to give the salient features of the case as nearly as possible in Dr. Mason’s own words.

The grounds of my challenging Dr. Mason’s position are these: 1. The absence of any evidence of direct injury to the supra pubic region, and of any reference to the presumed state of the bladder as regards distension of that viscus at the time of the accident. These may be only omissions, but I have stated

above that I deem decisive evidence upon them essential to form a diagnosis. 2. I note the symptoms did not warrant an exact diagnosis until seventy hours after the occurrence, when symptoms of peritonitis supervened, and I ask, Would blood and urine have lain that time in the peritoneum without exciting that action at a much earlier period? 3. With regard to the tumour felt in the rectum, which so immediately disappeared upon opening the bladder; the position of the rent, which Dr. Mason was so fearful of exploring; and the appearance of the brown¹ discoloration some days or so after the operation, I would ask whether these symptoms do not all point to the more limited extent of the effusion. 4. Besides these, there are not wanting some indications that Dr. Mason himself is in some doubt whether the case quite accords with the diagnosis he has claimed to have established. It will have been noted that he, from prudential motives, did not explore the rent which he felt in the bladder; but if Dr. Mason had felt quite sure bloody urine had collected in the abdomen, would he not have thought it essential to make quite certain he had established a vent for it? Again, Dr. Mason seems to infer the urine was lying only in the pelvic cul-de-sac of the peritoneal cavity, and had not passed into the general cavity of the abdomen; but I hold there is no authority for considering it possible that when the peritoneal covering is torn in cases of rupture of the fundus of the bladder, urine will find its way into the pelvic portion of the peritoneal cavity, and will not invade it universally.

I have now indicated the points which lead me to conclude that in Dr. Mason's case the peritoneum was not injured. That a laceration occurred immediately posterior to the prostate is, I think, almost certain, as also that when the effused urine encroached upon the pelvic peritoneum, local peritonitis was excited. Equally I regard it as quite clear the patient owed his life to Dr. Mason's decisive operation, and the timely performance of it.

Yet the point remains, May not, after all, the lateral operation be the most judicious proceeding in this lesion, and afford the patient the best chance of recovery? I have given Dr. Stephen Smith's and Dr. Mason's arguments in favour of this view. I cannot, however, share their opinion, for I do not see what lithotomy can do to remove the blood and urine which is lying in the abdominal cavity, or in arresting its injurious effects upon the peritoneum, beyond what catheterism can accomplish. I say this from observation of a fact which I have observed in these cases—viz., that within a limited period—

¹ Gross, *loc. cit.*

in my patient within twelve hours—the urine that is drawn off is found to be clear, whereas there remains all the while a quantity of deeply blood-stained urine locked up in the abdomen. The conclusion I draw from this fact is that the intestines lying over the rent quickly adhere over and around it, and so close it up. Indeed, in one case a hernia of the intestine occurred, producing symptoms of acute intestinal obstruction, which so masked the case that the original and correct diagnosis of rupture of the fundus of the bladder was set aside in favour of ruptured intestine, the urine that was drawn off after the lapse of twenty-four hours from the accident being clear. At the post mortem a large rent was found in the fundus of the bladder, into which a coil of intestine had slipped and become strangulated. There was also much bloody urinous fluid within the abdomen, and general peritonitis. If, then, nature succeeds commonly in closing over the rent in the bladder so completely that the bladder can again become a receptacle for urine, and thus forms an effectual barrier against subsequent extravasation into the peritoneum, the demands of surgery, so far as regards the bladder, would be met by steps being taken to ensure the withdrawal of the urine as quickly as it was received into the bladder; but it is not the urgency of the bladder symptoms so much which threaten death as peritonitis dependent upon the presence of an irritating fluid, and these are the symptoms which appeal most directly to the surgeon for relief, and I am justified in stating that abdominal section appears to meet the urgent requirements of these cases, which involve the removal of the effused blood and urine and the cleansing of the peritoneal cavity. As the performance of this would necessarily reopen the bladder wound, the operation is completed by effectually closing the rent in the bladder by sutures. Nothing but an enlarged experience can decide whether this operation offers such a reasonable chance of recovery as to make one justified in pressing it upon a patient whom the surgeon can only regard as the subject of a mortal injury. It appears that so long ago as 1840, Dr. Blundell,¹ an American surgeon, suggested an operation very similar to this, only that instead of stitching up the rent with silk, he proposed to bring it forward, and fasten its edges to the margin of the abdominal wound. Dr. Blundell experimented upon four rabbits, three of which recovered; but it does not appear that he had an opportunity of putting his operation to the test in any case of ruptured bladder in the human subject. It would, if it were possible of accomplishment, be treating the lesion as the pedicle is dealt with in ovariectomy; but I should imagine the cases in which it was

¹ Dunglison's *Amer. Med. Liby.*, Philadelphia, 1840, p. 54.

possible to drag forward the edges of the rupture to the surface of the abdominal wound, and retain them there, would be quite exceptional, still I regard the suggestion as a step in the right direction, and as worthy of being put into practice where practicable.

Lastly, I would urge the point, whether the injury may not be promptly recognised, and the propriety of surgical interference determined with the least possible delay, as then the operation might be performed before the worst features of peritonitis had appeared.

SOME REMARKS
ON THE
INTRODUCTION OF THE WHOLE HAND
INTO THE RECTUM.

BY

W. J. WALSHAM.

These remarks are founded principally on four cases in which during the past year I introduced my whole hand into the rectum for the purpose of diagnosis. Three of these cases occurred in the wards of St. Bartholomew's Hospital, and one in the practice of Dr. Pottle of Bunhill Row. Although the introduction of the whole hand into the rectum has frequently been resorted to in Germany, the late Professor Simon of Heidelberg having especially turned his attention to the subject,¹ it has seldom been practised in this country; the only recorded cases I have come across being those published in the 'Lancet' a few years back by Messrs. Maunder, Heslop, and Batteson. And although I am informed that Mr. Bryant has often adopted this method of examination, I have been unable to find any account of his cases.

As palpation by the hand in the rectum, then, has seldom been practised by British surgeons, it seemed to me that the experience gained in the four cases just referred to, more especially as it confirms Professor Simon's statement that this method of examination is without risk, together with some experiments which I performed on the dead body, were worth recording in the Hospital Reports for the year.

¹ Professor Simon's papers will be found in the 'Arch. für Klin. Chir.,' vol. xv., and 'Göschel's Deutsche Klinik,' Band xxiv.; abstracts of which are published in the 'British Medical Record' for 1873.

Having been asked by Mr. Smith to examine through the rectum one of his patients supposed to be suffering from stone in the kidney, I thought it advisable before doing so to practise the operation on the dead subject. Ten or twelve experiments were accordingly made, a detailed account of which will be found at the end of these remarks.

In *all* these experiments, whether on the male or female subject, the hand, which measures a little less than $7\frac{1}{2}$ inches at its widest part, *could be readily* passed into the rectum. This was a point I was anxious to determine, as Mr. Maunder in his communication to the 'Lancet' of 1868 states that he was unable to introduce his whole hand (the size of which he does not state) into the male rectum, although he had succeeded in doing so in the case of a female; and Professor Simon says that in some of his cases he has had to incise the sphincter before he could overcome the resistance of this muscle.

After the conclusion of the examination a careful dissection was in all cases made, but no rupture of the sphincter or other disturbance of the cellular or vascular connections of the rectum could be discovered; in the majority of cases not even the integumentary fold immediately surrounding the anus was torn, although considerable force was used in many instances.

The hand, when once through the sphincter, could be readily carried up the rectum, and a thorough examination of the pelvic cavity and lower part of the abdomen and their contents made.¹

In the first two experiments I found little difficulty in passing through the rectum into what, from the distance my hand had penetrated, I imagined to be the sigmoid flexure of the colon; but that the hand really reached this portion of the gut could not be absolutely determined, as the bodies being required for the purpose of dissection, the abdomens were not opened at the time. In the fifth case, the abdomen having been previously opened, I was able to ascertain by actual inspection that my hand had passed into the sigmoid flexure, and by a little manipulation with the other hand I was enabled to put it into the lower part of the descending colon without rupturing the peritoneum.

In the 3d, 6th, and 9th experiments it felt as if arrested a little above the middle piece of the rectum, and this was found to be the case when the abdomen was opened. The cause of the constriction was then discovered to be a narrowing of the

¹ A detailed account of what can be made out in the *healthy* subject will be found in a short article which I contributed to Mr. Holden's 'Medical and Surgical Landmarks.'

gut where the peritoneum is reflected from the rectum to the walls of the pelvis.

Professor Simon fixes the junction of the first and second pieces of the rectum as the limit beyond which it is unsafe to introduce the whole hand; but in this opinion I only partially agree, as in Experiments I., II., V., X., and XII., I was able to pass a considerable distance beyond this point without causing any rupture of the peritoneal and other coats of the bowel, or disturbing to any serious extent the vascular and cellular connection of the bowel. In the remainder of the cases, however, the gut became suddenly smaller where it received its complete covering of peritoneum, and there can be no doubt that in similar cases Professor Simon is quite right in affirming that it would be highly dangerous to pass the hand into this narrowed portion; for, wishing to ascertain whether it would be practicable to overcome this constriction, I used considerable force, but was unable to succeed until the peritoneal coat of the gut had been ruptured. This narrowing of the gut is detected by the tight constricting sensation which it gives to the hand, and once felt, can be readily recognised again, and distinguished with little difficulty from the simple contraction of the muscular coat, which in the living ordinarily grasps the fingers as they are pushed onwards. The number of cases at present examined is hardly sufficient to fix the percentage in which a rapid narrowing of the gut exists.

The highest point in the abdomen I was able to reach was the lower end of the kidney and the cartilage of the last rib. When the hand is arrested in the rectum the loose connections of the gut still allow of very free movement, so that even in this case parts above the brim of the pelvis can be examined.

In commenting on the cases in which the operation was performed upon patients, only a short summary of the symptoms will be given. A full account of the cases, however, taken principally from the Hospital books, will be found at the end of this paper.

W. P. was admitted into St. Bartholomew's Hospital, January 1876, under the care of Mr. Smith, supposed to be suffering either from stone in the kidney or from some tuberculous affection of the genito-urinary mucous membrane. His symptoms were—

Painful and very frequent micturition. Urine high coloured, containing abundance of pus and blood on movement. Slight swelling at the lower and back part of either testicle. He was sounded under gas, and the absence of stone in the bladder ascertained.

On examination his prostate was found fairly healthy. There was slight swelling and œdema of the left foot and leg. After a few days' rest in bed, the œdema and the blood in the urine disappeared, the latter being again observed when he was allowed to get up and walk about. January 14th, he was examined by the rectum. The patient, who was a big, powerful, and well-nourished man, having been brought deeply under the influence of chloroform, was placed on his right side, with his thighs well drawn up. The left hand being the smaller, was well greased with fresh butter, and the introduction commenced. The sphincter ani was dilated very gradually, first two, then four fingers, and finally the thumb being passed through the anus, and the dilation slowly and steadily continued. On attempting to pass the whole hand, considerable resistance was experienced, but was at length overcome by steady pressure and additional force applied by Mr. Smith to my elbow. The hand now slipped into the rectum, and although moderately grasped by the muscular coat, could explore the pelvic cavity without much trouble. By this means the normal condition of the prostate and the absence of stone in the bladder were confirmed. The parts seeming fairly large, the hand was carried with considerable facility into the sigmoid flexure, no constriction being experienced in the upper part of the rectum; but there was some little difficulty in following the course of the bowel, as the loose folds of the sigmoid flexure hung about the fingers, and care had to be taken, in finding the passage, not to perforate the gut. On passing the brim of the pelvis, the pulsation of the lower part of the abdominal aorta and of the iliac arteries was plainly felt. The hand being now pushed further up the gut, the fingers could be distinguished through the abdominal walls near the last rib; and on being pressed backwards in the region of the left kidney, nothing that gave the sensation of a stone could be detected.

The operation having lasted full twenty minutes, the hand was withdrawn. The patient experienced no inconvenience from the manipulation. There was no incontinence of feces, no rupture of the sphincter or integumentary fold round the anus; in fact, the man stated that he would not have known anything had been done to him had it not been for a feeling of slight soreness about the anus for a few hours after the operation. He was discharged from the Hospital.

Whether a stone in the kidney can be detected by the hand in the bowel, is a point, I fear, not settled by this case.

A stone was certainly not felt, and the hand as certainly reached the kidney; but as the patient is still living, it is of course impossible positively to affirm that one was not present.

The second patient into whose rectum the hand was passed was a female under the care of Mr. Holden. She was admitted on January 14th with symptoms of intestinal obstruction of a somewhat doubtful nature. After various remedies had been tried and failed, I was asked to see if I could detect a stricture in the upper part of the rectum. A long tube had been previously passed a foot and a half up the gut, and a pint and a half of fluid injected, the greater part of which had been retained. The patient having been deeply narcotised, the left hand was introduced with the same precautions as in the former case; but on reaching the upper portion of the gut, it was arrested by a tight constriction, through which only the fingers could be passed. As there could be little doubt, from the experience gained in the dead body, that this constriction was due to the rapid narrowing of the gut at the point where the peritoneum is reflected to the pelvic wall, it was deemed imprudent to proceed farther with the investigation, and the hand was accordingly withdrawn. No inconvenience *of any kind* was experienced by the patient from the manipulation. On the 17th, 20th, and 26th the bowels were opened, and average motions passed; but after the 26th they again ceased to act, and the patient gradually sank, and died on the 5th of March. At the post-mortem examination a small epitheliomatous stricture was found in the upper part of the sigmoid flexure. Had the narrowing of the gut not existed, and the parts been as capacious as in Mr. Smith's case, the hand would have probably detected the cause of the obstruction; but as it here failed to do so, the introduction of the hand was possibly productive of harm, as the non-detection of the stricture may have deterred Mr. Holden from performing colotomy—an operation which, as the post mortem shows, would have been successful so far as opening the bowel above the stricture was concerned. But at the same time it must be remembered that this was one of those doubtful cases in which it was next to impossible to come to a correct diagnosis of the situation of the obstruction, for, as before stated, a tube had been passed a foot and a half up the bowel; so, even if the hand had not been introduced, it is not at all certain that any operation would have been undertaken.

The third patient was a female under the care of Dr. Pottle. She also was suffering from internal obstruction of a doubtful nature. Chloroform having been administered, the hand readily passed through the sphincter, and the parts being very capacious, a thorough examination was accomplished. Much fecal matter was discovered in the upper part of the rectum and sigmoid flexure, the hand easily passing into this portion of the intestine, as the constriction so often referred to was not present in this

instance. That the hand really passed into the sigmoid flexure, as above stated, I feel convinced, for the forearm almost as far as the elbow was forced through the anus. Much fecal matter was removed by the aid of the hand and a long injection-tube, several feet of which were directed by the fingers into the bowel. No bad effects followed this operation, although the hand was withdrawn and reintroduced several times; the sphincter was not ruptured, and there was no incontinence. The patient rallied a few days after the operation, and several motions were passed. She died, however, some eleven days after I saw her, from an attack of severe diarrhoea. A post mortem could not be obtained, but I think I can safely affirm that in this patient no stricture of the rectum or sigmoid flexure existed, and in this opinion I was supported by Dr. Andrew, who also saw her.

I examined the fourth case at the request of Mr. Savory, likewise for the purpose of ascertaining the presence of a stricture; but here, unfortunately, as in Case II., the hand was prevented by the narrowing of the upper part of the rectum from reaching far enough, and no stricture could be discovered. A slight thickening, however, of what seemed to be a neighbouring portion of intestine through the walls of the gut was detected by the hand, though I was unable to give an opinion as to its nature. No rupture of the sphincter ensued. The patient, who was very much exhausted before the operation, died the same evening, and at the post mortem a small stricture near the commencement of the sigmoid flexure was discovered. Before the parts were disturbed the hand was again introduced into the rectum, and an attempt made to reach the stricture; this was found to be quite impossible, the fingers being fully 6 inches from the disease when pressed onwards with a force nearly sufficient to rupture the gut. The resistance to the further passage of the hand was clearly seen, as diagnosed during life, to be due to the sudden narrowing of the rectum a little below the sigmoid flexure, where the peritoneum is reflected to the pelvis. On allowing the diseased portion of the sigmoid flexure to sink into the pelvis, it was at once perceived that it was this that had been felt during life as the indistinct thickening through the walls of the rectum; but even with the light of this experience, it does not seem probable that in a similar case such a condition could be diagnosed in the living patient, for it must be remembered that the fingers, when they reach the higher part of the rectum, are held tightly together in the form of a cone by the surrounding bowel, and cannot be separated so as to grasp any structure situated outside the bowel sufficiently well to deter-

mine its nature. The only manner in which such a part can be here examined is by pressing upon and carrying the tips of the fingers over it.

Although in the three cases of intestinal obstruction just related, the introduction of the hand was not so successful from a diagnostic point of view as could be wished, a fact of some importance has at any rate been established—viz., that a stricture below the descending colon may exist without the hand being able to detect it. But whether this should deter one from repeating the operation in like cases in future appears at present an open question. On the whole, however, the weight of argument I think tends in the direction of a repetition, for should the hand, the careful introduction of which has been shown to be without risk, detect a stricture, all doubts as to the proper mode of treatment will be removed; but should it fail, no harm need accrue if the surgeon bears in mind that a stricture may still exist though undetected, as in the latter instance he will be no worse off for the introduction of the hand, and can act according to his judgment as if this mode of investigation had never been undertaken.

The whole gist of the matter, however, seems to lie in this—whether in the *majority of cases* in which a stricture really exists the hand will be able to detect it. To settle this point a large number of observations will be necessary, and many experiments on the dead body must be undertaken to determine accurately the percentage of cases in which the hand can be passed into the sigmoid flexure without impediment from the band-like constriction already referred to. I have already in a considerable number of bodies taken accurate measurements of the rectum and sigmoid flexure at definite distances from the anus, but I am unwilling to make use of these until a still larger number have been accumulated, and shall here content myself with stating that the circumference of the bowel is subject to great variation.

In the three cases of intestinal obstruction, it will be seen that the hand in all failed to detect a stricture; notwithstanding which, in two cases one was discovered after death, and in the third no post-mortem examination was permitted. Setting aside, then, the third case as non-proven—although from the thorough examination obtainable, and the subsequent history of the patient, there seems little doubt to my mind that the absence of a stricture was clearly established—Mr. Holden's and Mr. Savory's cases may be briefly considered. In the former, the stricture being in the upper part of the sigmoid flexure, did not descend into the pelvis, and the narrowing of the upper part of the rectum prevented the hand reaching it, so that by no means could it have been detected.

with safety. In the latter case, although the hand was unable to reach the disease from the inside of the bowel, still the stricture might have been possibly discovered through the gut-walls; its non-discovery perhaps being due to the inexperience and want of education of the operator's hand, rather than to any fault in the method of investigation.

Palpation by the rectum, however, is probably of more service in the diagnosis of several other diseases of the pelvis and abdomen than in these in which I have as yet had an opportunity of practising it. Professor Simon, indeed, states that he has found it of considerable help in determining the nature of many doubtful affections of the bladder, uterus, and ovaries, and in forming a correct estimate of the extent of the various pelvic deformities. But besides the uses to which this method of examination has been put by Professor Simon, I venture to think that there are several other affections of these regions in which it will be found of considerable service; for, passing my hand over the brim of the pelvis, I was much struck by the facility with which the pulsation in the large branches of the abdominal aorta could be traced to their exit from the pelvis; and it seems to me that in case of aneurism of any of these vessels, a very accurate idea of the extent and conditions could by these means be made out. In gluteal aneurism, also, a careful exploration of the margins of the greater sacro-ischiatic foramen through the walls of the rectum would probably determine whether or no the intra-pelvic portion of the vessel was encroached upon by the sac, a point, as it is well known, of some importance in deciding on the treatment of this affection. In diagnosing between cancer and aneurism of the pelvic arteries it may also prove useful. Although it is obvious that any conclusion based on the limited experience of this line of investigation as yet obtainable must be necessarily imperfect and liable to modification, still I have ventured to draw up the following, which I think are in accordance with what is at present known of the subject:—

1. That the hand, if small, can be introduced into the rectum of both male and female without fear of rupture of the sphincter or incontinence of fœces.

2. That the dilatation of the sphincter should be very gradual, five minutes at least being allowed for its accomplishment.

3. That no pain or inconvenience is experienced by the patient as an after-result of the operation.

4. That when once through the sphincter, the windings of the gut should be followed very cautiously by a semi-rotatory

movement of the hand, and by alternate semi-flexing and extending the fingers.

5. That in many cases the hand can be passed into the sigmoid flexure, and possibly, in rare instances, into the descending colon.

6. That should the hand meet with a feeling of constriction about the junction of the first and second pieces of the rectum, no force on any account should be used to overcome it, as this can only be accomplished by rupturing the peritoneum, which is here reflected from the intestine.

7. That this method of investigation is of use in detecting a stricture high up the rectum or in the sigmoid flexure of the colon, but that a stricture below the descending colon may exist although the hand may be unable to discover it.

CASE I.

W. P., æt. 31, admitted into Henry Ward, under the care of Mr. Smith, December 10, 1875.

History.—Two years ago he was first taken with slight pain in passing his water. The pain came on suddenly whilst he was ploughing, taking him in the left side of the belly. The testicle did not swell for about three months after the first attack of pain. After the pain he could not pass his water well, nor get it all away, and required to strain very much. Required to pass his water rather more often than before. At the end of a week or ten days he passed some pure blood after the water. The testicles swelled very much, especially the left, and seemed very tender. When the swelling subsided, a small lump remained at the lower part of each. The trouble with his urine got slowly worse. The urine became thick, and stained often with blood. Was sounded in the Plymouth Hospital; no stone found; said to have inflammation of the bladder. Has been getting worse and worse ever since, until he has been obliged to pass water every few minutes. General health has nevertheless been fairly good. There was no history of gonorrhœa, of cough, or of hæmoptysis, nor was there any family history of tubercle or scrofula. He appeared on the 11th as a well-nourished man, walking about the wards apparently in perfect health, but obliged to pass water every few minutes night and day. There was pain in micturition. Urine bright red, containing large quantities of sediment, and loaded with albumen, containing also a large quantity of pus and blood. Just below the left testicle, and attached to it, is a tolerably firm swelling connected to the globus minor. In the globus minor of the right is a

similar, but smaller, lump. He was sounded under gas, but no stone found.

'December 13.—He was examined by the finger in rectum, and small lump felt in prostate.

'December 17.—Swelling and œdema of foot and leg.

'January 13.—Has been in bed for several days. Œdema of foot and leg has passed off. When he gets up and walks about the ward or square, blood appears in urine; when he keeps quiet in bed, the urine is almost free from blood.'

January 14.—Examined through the rectum by Mr. Walsham. The patient having been brought deeply under chloroform, was placed on his right side, with his thighs well drawn up. The left hand being the smaller, was well anointed with fresh butter, and the introduction commenced. The sphincter ani was dilated very gradually, first two, then four fingers, and finally the thumb being passed slowly through the anus, and the dilatation gradually and steadily continued. On attempting to pass the whole hand, considerable resistance was experienced, but was at length overcome by steady pressure and additional force applied to the elbow. The hand now slipped into the rectum, and though moderately grasped by the muscular coat, could explore the pelvic cavity without much trouble. The condition of the prostate and the absence of stone were confirmed. The parts seeming fairly large, the hand was carried with considerable facility into the sigmoid flexure, no constriction being met with in the rectum; but there was some little difficulty in following the course of the bowel, as the loose folds of the mucous membrane hung about the fingers, and care had to be taken, in finding the passage, not to perforate the gut. On passing the brim of the pelvis, the pulsation of the lower part of the abdominal aorta and of the iliac arteries was plainly felt. The hand being now pushed farther up the gut, the fingers could be distinguished through the abdominal walls near the last rib. On being pressed backwards in the region of the kidney, nothing giving the sensation of a stone could be detected. The operation having lasted full twenty minutes, the hand was withdrawn. The patient experienced no inconvenience from the manipulation. There was no incontinence of fœces, no rupture of the sphincter or integumentary fold round the anus; in fact, the man stated he would not have known anything had been done to him had it not been for a feeling of slight soreness about the anus for a few hours after the operation.

'March 17.—Sent to the Convalescent Hospital.

'April 18.—Readmitted. Condition much as before. Nothing definite discovered.

'May 5.—Discharged.'

CASE II.

'Jane D., æt. 47, was admitted on January 21, 1876, into President Ward, under the care of Mr. Holden.

'On January 14th she first felt a slight pain in the abdomen. On January 17th, the pain having continued, she was sick, and has vomited ever since. On January 19th she was so much worse that she went to bed. The abdomen was then swollen. The bowels acted very slightly on January 18th, but have not done so since that date—when this note was taken, on January 24th.

'She lies in bed with her shoulders raised, complaining of pain in abdomen, chiefly about umbilicus, which comes on about every twenty minutes. The abdomen is much distended, quite soft; not tender, except very slightly in the middle line just above and below the umbilicus, and in the right lumbar region.'

'The abdomen is tympanitic. No hernia detected. The patient is weak; her face pinched; tongue dry, central streak. Pulse, 96, soft, regular, and very small; temperature, 99.1°.

'January 21.—A long tube was passed up the rectum for about a foot and a half, and three pints of olive oil and gruel were thrown up. Some of the injection came away at the same time, and the rest remained for about half an hour.'

'Ordered—Turpentine stupes. Ext. opii gr. $\frac{1}{4}$ every two hours. D. L. brandy, \bar{z} iv. Morphia injection, gr. $\frac{1}{4}$, at bed-time.

'January 22.—Another injection of olive oil, which, like the last, returned. No action of bowels. Pulse, 86; temperature, 100°. In great pain.

'January 23.—No action of the bowels.

'January 24.—No action of the bowels. Temperature, 98.6°; pulse, 104. No sickness.

'January 28.—Has changed little this day. No action of the bowels. She has constant pain, though not severe. The temperature 98.4°. Tongue furred and dry. Abdomen the same. Constant eructations.

'January 30.—A very small and solid motion passed. The treatment as before.

'February 2.—No further motion. Pulse, 80, weak.'

'February 8.—Mr. Walsham was asked to examine by the rectum. The patient was brought under chloroform, and placed on the right side, with the legs well drawn up. The left hand and forearm having been well buttered, two fingers were passed into the anus; and after the sphincter had been somewhat dilated,

the ring and little finger were introduced. Firm pressure was now kept up by the fingers for some minutes; the thumb was then passed. Much force was now gradually and firmly applied, the sphincter slowly dilating; but the hand could not be passed until assisted by pressure at the elbow. When well within the rectum, the pelvic cavity was thoroughly explored; but on reaching the upper portion of the gut a tight constriction was felt, which was diagnosed as due to the narrowing of the gut at the point where the peritoneum is reflected to the pelvic wall. The fingers only could be passed through this constriction, and no stricture was discovered; the hand was accordingly withdrawn. The sphincter was not ruptured, and no inconvenience from the operation was experienced by the patient.

'February 10.—Feels about the same. No motion. Slight pain in rectum.

'February 11.—A little better. No motion. Rectum less painful. Passes wind downwards.

'February 12, 13, 14, 15, 16.—No motion. No pain in rectum.

'February 17.—Has had a very good motion. Motion hard, and about one inch in diameter. Has vomited.

'February 19.—Motion this morning about the same as that of the 17th.

'February 21.—A very good motion on Sunday. Feels much better in herself.

'February 24.—Passed a slight motion.

'February 25.—In a good deal of pain. No motion. No sickness.

'February 26.—Another motion.

'February 28.—Bowels open on Sunday.

'March 1.—Not so well. Bowels not open since February 27. Abdomen much distended; highly tympanitic. Tongue dry. Pulse fair.

'March 2.—No motion. Unable to keep anything on the stomach. In great pain.

'March 5.—Bowels not open. Constant sickness. Death.

'*Post mortem*.—Epitheliomatous stricture of the upper part of sigmoid flexure.'

CASE III.

For these notes I am indebted to my friend Mr. Capon.

'Mrs. B., æt. 50, came under the care of Dr. Pottle, March 7, 1876, with symptoms of intestinal obstruction. She had been suffering from diarrhœa some weeks before she saw Dr. Pottle.

She was a fairly-nourished woman. Her abdomen was distended. Slight pain in right hypogastric region. Occasional sickness and vomiting.

'She was treated by enemata and opium, after purgatives had been used, but her bowels were not relieved.'

'On March 17th Mr. Walsham was asked to pass his hand up the rectum for the purpose of ascertaining the existence of a stricture. Much fœcal matter was removed, with considerable relief to the patient. The hand being introduced into the rectum, a tube was passed several feet, and much fœcal matter brought away. There was complete power over the sphincter next day, and no rupture.'

'She rallied for seven days, but died March 28th with diarrhœa.

'No post mortem was obtainable.'

CASE IV.

Emily B., æt. 46, was admitted into President Ward, April 12, 1876, under the care of Mr. Savory.

'She stated that four years ago she suffered severe pain in the hypogastrium, and passed some blood from the bowels. From this time she experienced some difficulty in passing her motions, but this trouble was quite insignificant for the first four or six months after her attack of hæmorrhage from the bowels. For three years her motions had been small and passed with difficulty, the bowels seldom being moved without injections. For the last three weeks she had no action of the bowels, many injections having been given, which have returned mixed with fœcal matter in very small quantities. For fourteen days vomiting has occurred, and for some days has been very offensive. On the evening after admission she vomited about a pint and a half of fœcal matter. The patient was fairly nourished. Her abdomen was greatly distended and tympanitic. She was exceedingly faint and exhausted.'

April 13.—Mr. Walsham was asked to pass his hand into the rectum. The patient having been deeply narcotised with gas and ether, the hand was readily introduced. Much force was necessary to overcome the sphincter, a gentle push on the elbow being required to aid the passage of the hand. A tight constriction was felt about the junction of the second and third pieces of the rectum, which prevented the hand reaching the sigmoid flexure. No stricture was detected. As the patient was faint and exhausted, an injection-tube was passed about a foot and a half up the gut, and a pint or more of gruel with brandy was

injected. Some fœcal matter was in this way brought away, but no stricture discovered. The patient died the same evening.

‘At the post mortem the intestines were found greatly distended. As low down as to the beginning of the sigmoid flexure, the muscular coat of the intestines was hypertrophied. At the upper end of the sigmoid flexure was a carcinomatous stricture 1 inch in length and about $2\frac{1}{2}$ in breadth, involving all the coats and the whole circumference. Other organs were healthy. After the abdominal cavity had been opened, and before the parts were disturbed, Mr. Walsham pushed his hand into the rectum and endeavoured to reach the stricture, but was prevented from doing so by the narrowing of the gut some 8 inches from the anus. His fingers, when pressed onwards with much force, were still some distance from the stricture. On allowing the sigmoid flexure with the stricture to drop into the pelvis, the diseased portion of the gut could be touched through the walls of the rectum by the hand.’

Experiment I.

Subject, an adult male of large proportions. The body placed on its back. Hand introduced by first passing two fingers, then four, and finally the thumb. The whole hand passed with knuckles towards the curve of the sacrum.

The hand seemed to be in a fairly capacious bag when once through the sphincter. The symphysis, pubic arch, spine of the ischium, and outlines of the sacro-ischiatic foramen distinguishable. The curve of the sacrum was readily followed, and the promontory easily made out. Some difficulty was experienced in finding the way up the bowel, as the folds of mucous membrane hung about the hand. This difficulty was overcome by semi-rotatory movement of the hand, and by searching for the canal of the gut with the tips of the fingers. Fingers and hand seemed to travel into sigmoid flexure.

Something in the situation of, and feeling like, the kidney was touched on pressing back the hand in the lumbar region.

Abdomen not opened at time of experiment, but when the parts were examined some days afterwards, the sphincter was not ruptured, and the cellular and vascular connections of the rectum were not injured. The coats of the bowel normal. Sigmoid flexure and upper part of rectum capacious.

Experiment II.

Adult male subject. Hand introduced as in Experiment I., with the same result.

Experiment III.

Adult female of fair proportions. Abdomen opened before the experiment began. The hand easily introduced as in Experiments I. and II., but arrested near the upper part of the rectum by the reflexion of the peritoneum forming the mesorectum, the gut suddenly narrowing at this point.

Experiment IV.

Adult male subject. Hand introduced as before, but arrested near the middle of rectum. Abdomen cut open whilst the hand was in the bowel. The passage of hand prevented by the reflexion of the peritoneum.

Considerable force exerted, and hand only passed after rupture of peritoneal coat.

Experiment V.

Full-grown female of large proportions. Abdomen opened. Hand easily introduced, and carried by a semi-rotatory movement into the sigmoid flexure. Connections of the bowel very free, allowing the hand and gut to be moved easily about the pelvis and lower part of the abdomen. Left kidney felt. By a little manipulation with the disengaged hand, the hand in the bowel was pressed into the lower end of the descending colon, and was here tightly embraced by the gut, but no rupture of the gut-walls was produced.

Experiment VI.

Full-grown male subject. The abdomen was opened before the hand was placed in the rectum. Some difficulty in getting the hand through the sphincter, as the ischio-rectal fossæ had been dissected. The hand was arrested at upper part of rectum, where the peritoneum forms the meso-rectal fold. The hand was forced into this narrow part of the gut, but in doing so the peritoneal coat was ruptured in form of a longitudinal rent. With the parts thus exposed, the pelvis was explored by the hand through the wall of the gut, the loose connections of the rectum and sigmoid flexure allowing the hand to be carried very freely round the walls of the pelvis. The pubic arch could be made out, and the ischiatic foramina and foramen ovale distinguished.

Experiment VII.

Adult male subject. Hand passed with some difficulty. Ischio-rectal fossæ had been dissected. Sigmoid flexure and upper part of rectum tightly contracted. Hand arrested as in Experiment VI. Sigmoid flexure afterwards stretched; found very

small, with no possibility of the hand being introduced into it. Meso-rectum somewhat short.

Experiment VIII.

Adult female. Small proportions. Abdomen opened. Hand could be passed only a short distance up the rectum. Sigmoid flexure and upper part of rectum small.

Experiment IX.

Subject, adult male. Pelvis rather small. Hand introduced with some difficulty, as the ischio-rectal fossæ had been dissected, allowing the anus to recede before the hand. As in some previous cases, it was arrested by the narrowing of the upper part of the rectum, where the peritoneum is reflected.

Experiment X.

Adult female. Fair proportions. Pelvis very capacious. Hand passed readily into the rectum, and carried easily into the sigmoid flexure, which measured $7\frac{3}{4}$ inches when fully distended, at a distance of 12 inches from the anus. Only half the sigmoid flexure remained in this case, the rest of it having been taken away with the arch of the colon. The hand was forced rather quickly through the upper part of the gut, and notwithstanding the fact that the gut when distended measured half an inch more than the circumference of the hand, a longitudinal rent was made extending 2 inches from the divided end.¹ The meso-rectum was very long, allowing very free movement of the gut.

Experiment XI.

Abdomen open. Ischio-rectal fossæ dissected. Hand introduced with difficulty, from the bowel receding before the hand. Narrowing of the gut prevented hand reaching far up the rectum. Rectum moveable. Could reach above the pelvic brim. Pelvis explored as before.

Experiment XII.

Adult male. Hand readily passed into the lower part of the sigmoid flexure.

¹ This rent was due to the weakening of the gut from the coats having been divided, the splitting beginning at the cut end.

REPORT
FROM
THE POST-MORTEM ROOM.

BY

J. WICKHAM LEGG, M.D., AND J. A. ORMEROD, M.B.

We venture to place on record in the Hospital Reports some of the more interesting cases which have come under our notice in the post-mortem room during the last twelve months. We wish to express our thanks to Dr. Black, Dr. Andrew, Dr. Southey, Dr. Church, Dr. Gee, and Dr. Brunton for their courtesy in allowing us to publish cases formerly under their care in the Hospital.

Aneurysm of the Anterior Communicating Cerebral Artery.

Five years ago Dr. Gee put on record¹ a case of aneurysm of the anterior communicating artery so like our specimen that the two might almost be described in the same words. Dr. Gee tells us that Lebert in 1866 could find only two cases of this aneurysm on record.² Since then Dr. M'Dowall has recorded a case in which the aneurysm did not burst,³ and Dr. Greenfield one in which the rupture seems to have been caused by a fall from a scaffold.⁴

The original observations from which the following account is taken were made by Dr. Wharry.

George Orme, aged 41, a van-driver, was admitted into Mark Ward, under Dr. Andrew, on June 16, 1876.

The account given of him was this: Six weeks ago, while pulling hard at a rope, he felt something give way in his head, and fell down insensible. This morning he was unusually well and in good spirits, and ate heartily at breakfast. Before going to work he left his watch with his wife, saying he might not come back. He had done no heavy work, when suddenly, about 7.15 A.M., he said he felt very queer, leaned against a post, and vomited; then said he would lie down, and fell insensible on to some hay in the stable.

¹ Gee, St. Bartholomew's Hospital Reports, 1871, vol. vii. p. 147.

² Lebert, Berliner klin. Wochenschrift, 1866, p. 345.

³ M'Dowall Lancet, 1875, vol. ii. p. 202.

⁴ Greenfield, Trans. of the Path. Soc. of Lond., 1876, vol. xxvii. p. 2.

He was admitted about 9.15 A.M., and is then described as a well-nourished and muscular man, lying on back, completely comatose, face dusky, eyelids almost closed, pupils very contracted, axes of eyeballs divergent, eyeballs occasionally rolling. Respiration slow, deep, irregular, with occasional sighing and stertor. Mouth somewhat open. Arms and legs motionless, and partially relaxed. Pulse, 48; full, irregular. Arteries not hard nor tortuous. No cardiac murmur. Temperature, 96.8°. Surface cold, showing goose-skin.

At 10.30 the limbs were rigid. There was occasional relaxation of the buccinators. There were general muscular twitchings, with clonic movements of the lower jaw.

At 11.30 the skin was warm and moist. The temperature had risen to 99.5°.

At 2.15 P.M. the bowels had been open (after an enema and purgative given in the morning), and it was thought that his condition was somewhat improved, but the coma continued. The skin was hot and bathed in sweat. The pupils much less contracted. The right arm seemed more powerless than the left.

He died the next day.

Examination twenty-seven hours after death.—Body moderately fat. Marks of blistering on nape of neck and legs. Calvaria rather thick, sinuses natural. There is an effusion of blood under the meninges, covering the lower part of anterior lobes and the part which turns upwards; also of the cerebellum. On dividing the two anterior lobes from one another, a large clot of blood is seen between the two, the brain substance around being much softened. This clot depends from an aneurysm, the size of a small marble, of the anterior communicating cerebral, which has a large rent in the lower aspect. The blood has found its way through the corpus callosum on the right side into the ventricles, where large clots are found, and has passed out by the velum interpositum on to the upper surface of the cerebellum. The fourth ventricle is filled with blood. No hæmorrhages in the pons. The rest of the cerebral arteries seem free from atheroma. The right retina shows numerous small hæmorrhages; there are likewise hæmorrhages in the sheath of the optic nerve.

Lungs natural; congested at bases.

Heart natural; valves much stained with blood. No thrombi in heart, nor granulations on valves.

Aorta in its length almost free from atheroma.

Viscera in belly quite natural. No disease of kidneys.

The aneurysm is preserved in the Museum.

ANEURYSMS OF THE HEART.

The readers of these Reports will remember that in the volume of last year two cases of aneurysm of the mitral valve were recorded. There are now two cases of aneurysm of the heart brought forward: the first is an example of a rather rare disease, an aneurysm of the septum of the heart, apparently of congenital origin; the second, another case of aneurysm of the mitral valve.

Aneurysm of the Septum of the Heart—Phthisis—Contracted Kidneys—General Atheroma, with Aneurysm of the Left Internal Iliac Artery.

William Headlam, 60 years of age, was admitted on September 27, 1875, into Matthew's Ward, under the care of Dr. Black.

For the following clinical notes we are indebted to Dr. Bridges, the house-physician.

The patient, an old man, with considerable ascites, and some anasarca of feet, is a plumber and painter by trade. He is very thin and wasted; has a sallow complexion, and thin hair. There is no blue line on the gums.

In the beginning of the year he had 'rheumatism' and swelled legs, and, for the first time, a cough came on in the summer; but at the same time he was fairly well until about three months before admission, when he slowly lost strength and appetite, and suffered from constipation.

Physical examination.—Motions contain bile, and are apparently healthy. Urine is cloudy from urates, but contains no albumen, sugar, nor indican. The feet and ankles are anasarcaous. There is considerable ascites and anasarca of the abdominal walls. The liver dulness reaches as high as the lower border of the fifth rib. No sign of enlargement below. Percussion over chest normal, save bases behind, which are absolutely dull, and where no respiratory murmur may be heard. The apex of heart beats in the nipple-line, and much below it. Sounds are normal, but intermittent. A loud diastolic (not præsystolic) murmur is heard occasionally at apex. When the heart's sounds intermit, the next beat does not carry the murmur. Pulse, 84; small and weak. Arteries tortuous.

October 6.—Both ascites and anasarca seem to be increasing, the latter invading the legs, and becoming extreme.

October 21.—Friction-sound heard all over right lung.

November 1.—He began to grow weaker.

November 5.—He was delirious, gradually losing his senses, and dying exhausted on November 8.

Examination twenty-four hours after death.—Chest and arms very thin. Belly much swollen. Legs oedematous, particularly the right.

The friends forbade the head to be opened.

Both pleuræ adherent at apices. The pleuræ greatly puckered and opaque in places, corresponding to hardness scattered through the lungs. These places being cut into, show in nearly all cheesy spots from peas to nuts in size, surrounded by yellowish, round, solid bodies of the size of mustard-seeds. These occur in groups, and at the apex are surrounded by highly-pigmented lung-tissue. No miliary tubercles seen. The bases of both lungs emphysematous.

The pericardium natural. The heart of natural size, the coronary arteries dilated, atheromatous, and tortuous. The heart-muscle is of a brown-red colour. There are no clots in the heart. The tricuspid valve much thickened at its edges, and opaque. The left side shows no signs of an acute endocarditis, past or present. The aortic and mitral valves are distinctly atheromatous, the aortic sigmoids having calcareous plates at their attachment to the aorta, and are much stiffened. At the base of the left ventricle, between two aortic valves, where the large flap of the mitral is attached, in fact, in the undefended space, is a pouch which would take about half a small marble. It approaches a hemisphere in shape, the mouth being the widest part and nearly circular, the diameter varying from 17 to 20 millimetres. The walls of the pouch are formed of a thin semi-transparent tissue, and are crossed by bands of membrane arranged like the *musculi pectinati* of the right auricle. The convex surface of the pouch on the right side of the heart is formed by the attached part of the tricuspid valve, and part of the wall of the auricle adjoining. The pouch is not perforated, and there is no roughening or opacity of the endocardium of the left side around the pouch.

The peritonæum holds a very great quantity of a dark but clear fluid. The bowels are not adherent to each other; there are no false membranes. The peritonæum seems opaque and macerated looking, but it is not thickened. In many places, especially over the cæcum, it is highly pigmented in small patches. There are also numerous ecchymoses, and the larger of the small vessels seem full of blood. Over the liver is a thin layer of blood in substance, and between the coils of the bowels are some small clots. There is a particularly large ecchymosis, the size of half-a-crown, over the mesocolon of the ascending colon. The mucous membrane of the stomach and intestines is quite sound.

The spleen very small and wasted.

The portal vein contains only fluid blood. The gall-ducts natural. The liver, though small, looks quite natural in appearance.

The supra-renal bodies natural. The kidneys small, the capsule tearing off in layers, or leaving a rough surface; in many places cystic. On section the kidney is red, having a cortex wasted to a few millimetres in breadth, very confused in structure, and tough. Bladder empty.

The aorta much dilated; there is extreme atheroma, so that an elastic shining piece, or one not thickened, cannot be found. The same may be said of the iliac arteries, common, internal and external. There is an aneurysm the size of a walnut on the left internal iliac artery. This aneurysm presses upon the accompanying vein, and at that spot begins a thrombus, adherent to the back part of the vein, grey in colour, but not filling the vessel. This extends up into the inferior vena cava, the mouth of the iliac being covered by a large thrombus as big as a walnut adherent to the vein about the opening of the iliac, but not elsewhere. It sends a prolongation down into the right iliac vein, which passes through the common into the internal and external iliacs, but not into the femoral vein. In the right internal and external iliac veins the thrombus is adherent to the walls, filling the vessels, and of a grey colour.

Pelvet states that aneurysms of the septum are always preceded by endocarditis.¹ We think this statement somewhat too broad, as the present case seems to show that aneurysms of the septum may be seen without preceding endocarditis. There are two like cases, apparently of congenital origin, recorded—one by Dr. Peacock,² the other by Dr. Hare.³

Aneurysm of the Mitral Valve.

John M'Neill, aged 37, was admitted into John's Ward, under the care of Dr. Brunton, on August 11, 1875.

For the clinical notes we are indebted to Mr. Macready and Mr. Bott, the house-physicians.

He is a cheesemonger by trade, and has not been a drinker. Five weeks before admission, while coming home from Kilburn, he was very sick. The feet were then swollen. Feet and legs now very cedematous, and belly swollen. Pulse, 82; fair volume, slow. Urine faintly smoky; specific gravity, 1018; highly albuminous. Under microscope, numerous hyaline, medium-sized casts, granular in part, showing also epithelium-cells far advanced

¹ Pelvet, *Des Anévrysmes du Cœur*, Paris, 1867, p. 52.

² Peacock, *Trans. of the Pathological Society of London*, 1846, vol. i. p. 61.

³ Hare, *ibid.*, 1865, vol. xvi. p. 80. *Cf.* Reinhard, *Arch. f. path. Anat.*, 1857, Bd. xii. p. 139.

in fatty degeneration. Many free epithelium-cells also far advanced in fatty degeneration. Blood-corpuscles are scanty. At the heart there is a loud systolic mitral murmur. During the month of September the urine remained very scanty, scarcely ever more than a pint in the twenty-four hours, and highly albuminous, usually one-half or three-quarters of the volume of the urine being filled with the coagulated albumen, and sometimes three-quarters. The anasarca was general. At the end of the month a double murmur developed itself at the apex of the heart. On October 7 the presence of sibilant and sonorous rhonchi over both sides of chest was noted; also dulness at both bases. The urine remained highly albuminous, but more in quantity, until the man died with an increase of bronchial symptoms on November 11, 1875.

Examination forty-five hours after death.—Belly and legs much swollen.

The right pleura holds about two pints of a clear fluid; the left about three. The peritonæum holds nearly a gallon of a fluid which is clear when first escaping, but afterwards becomes mixed with shreds of lymph.

The left lung collapsed throughout save at apex. The right lung less so; the lower lobe only being solid.

The pericardium is adherent to the heart at the back part near the apex for a space the size of a shilling, and there are also adhesions between the inferior and superior cavæ. There are two white patches on the front surface of the heart. The heart is much larger than natural; apparently more enlarged on the left than the right side. The aortic valves let water through readily. There is nothing remarkable on the right side; very slight dilatation of the tricuspid orifice. The appendix on the left side is free from clot. The endocardium of the auricle over the small flap of the mitral is studded with vegetations, none bigger than a mustard-seed, for a space which might be covered by a florin. On the large flap of the mitral are seen two bulgings—one at the edge of the valve close to the apex, rather flattened, and not bigger than a split pea; the other, close to the first, and seeming to run into it, but it is placed more towards the centre of the valve, and is of the size of half a hemp-seed or bigger. A chorda tendinea passing into the apex of the valve has been torn loose from its attachment to the musculus papillaris; another, close to its attachment to the valve, shows a spindle-shaped enlargement the size of a hemp-seed; it is hard, and seems to be made up of lime salts. The edge of the mitral valve shows small granulations: these are much more marked on the small flap. On the ventricular surface of the large flap are two openings leading into the substance of the valve. One is in the

middle of the valve, and corresponds to the smaller bulging on the auricular surface. The opening is surrounded by vegetations of the size of mustard-seeds. A probe can be passed into the smaller bulging through the opening. The other opening is close to the edge of the valve, but is not surrounded by vegetations; it leads into the larger cavity. The aortic valves are stiff, having many granulations along their edges; the substance of the valves is opaque.

The liver small, soft, and flabby, taking the print of the fingers. On section, the surface is coarsely marked, few red marks in comparison with the amount of yellow present.

Spleen large; it shows two infarcts, which are triangular and of pale straw colour.

The stomach holds a quantity of altered blood.

Kidneys of natural size; the surface somewhat granular, pale, studded with points of white, and a few red ramifying vessels; several cysts and scars of numerous infarcts. On section, the cortex is seen to be very pale, opaque yellow, narrowed, and showing a great contrast to the pyramids, which are of a pale purple. All trace of striation of the cortex lost.

Lympho-sarcoma of the Mediastinum.

William John Ross, aged 52, a coachmaker, was admitted into Mark Ward, under Dr. Andrew, on December 30, 1875.

The clinical observations of this case were made by the house-physician, Dr. Harris.

He gave the following history: He had had a cough for twelve months, having been previously quite healthy. Six months ago he noticed a lump just above the left collar-bone, which was followed by other lumps in the neck and axillæ. Rather more than a month ago both legs began to swell, and soon afterwards the left hand and arm. He had lost flesh rapidly for the last six months. Used to vomit his food two months ago. Had had occasional dyspnœa and dysphagia. He is described as somewhat pale and thin; lying on back or left side; respiration quiet; sleep disturbed by cough; sputum bronchitic in character. Tongue furred, appetite fair, bowels regular. Temperature, 101·8°. Pulse, 120.

There is on both sides of the neck, but especially the left side, a considerable enlargement, due to enlarged glands, in places solitary, in places matted together, and comprising the sub-maxillary, supra-clavicular, and the glands in front of and behind the sterno-mastoid. These glands are elastic, hardish, painless, and the skin over them non-adherent. There are large lumps in the left axilla and smaller ones in the right. There are no changes in the eye. The blood contains excess of white cells.

The thorax shows increased venation on the left shoulder, and

at the lower and front part. The walls are œdematous, and the chest itself barrel-shaped. There is visible epigastric pulsation. There is impairment of resonance, in front, down to both nipples; entire dulness down the sternum, and at the apices behind. Over the whole chest, but especially at right base, there is rough inspiration and loud rhonchus; laryngeal stridor; between the scapulæ tracheal breathing.

The heart-sounds are indistinct. Abdomen apparently natural.

There is œdema of the legs and the hands.

No enlargement of inguinal glands.

On January 9th he was taken suddenly with pain in the left side, increased by inspiration, and with shortness of breath, and at 12.30 A.M. on the 10th he died.

Examination thirty-four hours after death.—Body pale. Face and neck swollen. Glands of neck to be felt, easily movable under skin. The axillary and inguinal glands do not seem to be enlarged. There are no hæmorrhages apparent anywhere.

The notch in the liver reaches to the umbilicus. There is some slight general peritonitis; the fluid not being in excess, but holding large clots. The glandulæ concatenatæ of the neck are all enlarged from the angle of the jaw to the sternum. They are large, white, hard, and give no juice on scraping. They show no hæmorrhages. The second piece of the sternum shows a tumour the size of a nut, which is seen on raising the sternum to communicate through the bone with a large mediastinal tumour. This tumour is very closely adherent to the upper part of the sternum, and extends above the sternum into the base of the neck.

Both pleuræ are filled with fluid, more on the right side. There are large clots in the fluid, and the pleuræ are greatly roughened. The apices of the lungs cannot be separated from the mediastinal tumour which grows into them. The rest of both lungs collapsed.

There seems to be no pericarditis. There are enlarged lymphatic glands where the pericardium is attached to the diaphragm, and the tumour descends over the pericardium so as to form a thick cover to the upper part of the pericardium. The new growth can be seen projecting into the pericardium by the side of the vessels at the base of the heart. The heart itself small; muscular walls of a brown colour. The superior vena cava as it enters the pericardium almost blocked by a new growth into it. The clots in the heart very white, the serum very pale. No disease of the valves of the heart. The right branch of the pulmonary artery free from the pressure. The left is so narrowed that the little finger cannot be passed into it. The aorta is not markedly pressed on until the great vessels are given off; the forefinger can then with some trouble be passed into it, but it is tightly grasped. Both the carotids pass through the tumour, and their bore is narrowed.

The trachea is scarcely pressed upon, but both the bronchi and the lower part of the trachea show that the tumour has infiltrated their walls, and causes a white look on the mucous membrane. The gullet is much pressed upon at the level of the bifurcation of the trachea. The lower lobes of the thyroid body are involved in the new growth. The tumour is tightly adherent to the vertebræ, and the glands about the gullet are large and hard. The tonsils not enlarged, but marked with scars. The thickness of the tumour between the trachea and the sternum is 80 millimetres. It is very hard, white and yellow, marked with white lines into polygonal figures the size of hemp-seeds. Parts of it can be shelled out, leaving smooth walls. Sections of the tumour were kindly prepared by Mr. Groves, and showed small round cells embedded in highly fibrous connective tissue.

The liver is large, smooth surface, with everted edges, and friable as to consistence. On section, it shows the centres of the acini of a natural liver colour, the circumference of a pale opaque yellow. There are no white lines dividing the section, as is common in the liver of leucæmia.

The spleen is of natural size, very soft, of a pale red colour; no white nodules.

Stomach shows three enlarged, hard, white, lymphatic glands at cardia. The mucous membrane natural. Large and small intestines natural.

Kidneys large, quite natural.

Aorta singularly free from atheroma. No enlargement of mesenteric or prævertebral glands.

The two following cases may be studied side by side. In one of them new growths hung by long pedicles into the portal vein, chiefly in the direction against the stream of blood. In the other, the growth spread into the ducts, forming projections like warts on the mucous membrane.

Cancer of the Pancreas and Liver—Cancerous Polypi of the Portal Vein—Polypus in the Pancreatic Duct.

Susannah Knell, aged 78, was admitted into Elizabeth's Ward, under the care of Dr. Brunton, on August 10, 1875.

She said that she had enjoyed very good health until about a year ago. She had never been jaundiced. She had suffered from vomiting in the morning for nearly a year; for the last three months the vomiting had been constant. For the last few days the legs and ankles have swollen.

The woman is very thin. The belly is much distended. The liver is greatly enlarged, reaching to the right iliac region, to two

inches below umbilicus, and to within two inches of left anterior superior iliac spine. Large nodules may be felt all over the liver surface. The heart appears natural.

She continued to vomit, and no relief to this symptom could be secured. On October 5 it is noted that the urine is acid, and contains a trace of albumen. She died on October 28, at nine in the morning.

Examination twenty-seven hours after death.—Body greatly wasted. The liver and nodules on it can be very easily felt. The liver itself seems smaller than during life.

The peritonæum holds a very considerable excess of a clear fluid.

There is some excess of fluid in both pleuræ. The pleura of the apex of the right lung is much thickened, and below it are many highly-pigmented nodules. The same on the left side. The upper lobes of both lungs œdematous, less so the lower lobes. No new growths in the lungs.

Pericardium.—The vessels at the base of the heart bound together by many old and œdematous adhesions; over the cardiac layer of pericardium are many white spots. The heart after being opened weighs 250 grms. The coronary arteries are very tortuous; the heart itself wasted. Both the tricuspid and mitral valves show a very great thickening and opacity, not merely of the edges, but of the whole of the valves. The small flap of the mitral is likewise very much larger than is usual. The sigmoid valves are natural, beyond some slight thickening.

The large and small intestines and mesentery appear quite natural. The spleen weighs 80 grms. It is very small; it has numerous thickenings of its capsule, very like articular cartilage in physical characters.

The porta of the liver shows several enlarged lymphatic glands. On cutting them across, the section shows a white soft surface, with many small hæmorrhages. The common duct is greatly dilated; on dividing it, the flat surface is three times as broad as it should be. This dilatation persists until half an inch from the opening into the duodenum, where it rapidly becomes of a natural size. The duct is everywhere stained yellow. The hepatic duct is likewise much dilated, more on the left than the right lobe. The cystic duct has lost its windings, and opens at once into the gall-bladder. The gall-bladder is very small, not more than an inch long and a quarter of an inch across. It is still stained yellow. There are no gall-stones in it.

The portal vein outside the liver, the splenic and superior mesenteric veins are natural, and free from clots. The hepatic artery natural.

The liver itself weighs 2500 grms. On the surface are many

nodules, about twenty, of varying size, from a mustard-seed to a small apple. Their outline is rounded, colour whitish, and some are distinctly umbilicated. On cutting into the liver, however, the nodules are far more numerous within. They are round, white, rather firm, and many show small hæmorrhages. The liver substance between the nodules is natural, save that the centre of the lobule seems darker than natural. The hepatic veins are natural. On dissecting the portal vein within the liver, one of the first divisions of the right branch is found filled by several polypous-looking bodies. They are smooth and club-shaped at the end where they project into the free cavity of the vein; above they become slightly adherent to the vein wall, which appears perfectly natural. They send prolongations up branches of the portal vein above them, where, after travelling up some short distance, they again end in a bulbous projection. Their origin, however, appears to be in each case in one of the large nodules of the right lobe, to which they are attached by long and narrow pedicles. None of these polypoid bodies can be found in the left lobe. They are of about the same consistence as the nodules themselves.

A large lymphatic gland, similar in appearance to those in the porta, is attached to the under surface of the pancreas, but does not enter its substance. The pancreatic duct, where it makes the turn round the head of the pancreas, is for about an inch filled by a long polypoid body, very soft, which is adherent only at the end near the head of the pancreas. The body of the pancreas seems natural. The head is enlarged, and the cut surface shows many yellow cheesy spots from a mustard to hemp seed in size. The head is rather softer than the body.

The stomach and duodenum natural; no thickening of pylorus.

Many prævertebral glands are enlarged, and like those in the porta of the liver; one especially between the vena cava and aorta, as low down as the fork.

The kidneys weigh 180 grms. The capsule tears off with some trouble, leaving a red granular surface; the cortex much narrowed and indistinct. In the pelvis of the kidney is abundance of reddish-yellow gravel.

The bladder and rectum natural. The ovaries show small cysts, none bigger than small marbles. Around the right is a small extravasation of blood. The uterus small, puriform fluid in cavity.

The aorta is dilated and atheromatous; so are all the large vessels. The vena cava free from thrombus.

The head not opened.

The polypoid bodies from the portal vein were examined by

means of Hartnack's microscope the same day (Oc. 3, Obj. 9 à *im.*) The field was flooded with oval nuclei; many large cells, containing abundance of fat, and with these nuclei in them, were likewise seen. The nuclei often contained two or three nucleoli. Acetic acid brought out the nuclei well, leaving the fat drops undisturbed.

A scraping from the head of the pancreas, taken from a place close to the root of the polypus, examined; an immense number of compound granulation corpuscles were seen, and a large number of cells closely resembling those from the polypoid bodies in the portal vein. A creamy fluid exuding from the root of the polypus showed the same appearances.

The tumours being hardened in chromic acid, were examined with the microscope in the month of December. Thin sections stained with carmine were mounted in glycerine, and looked at with Hartnack's microscope, Oc. 3, Obj. 9 à *im.*

The polypi in the portal vein showed a delicate network supporting cells. The walls of the alveoli were very thin, while the meshes themselves were wide and rounded. The walls were formed of an almost homogeneous material, scarcely fibrous, and showing very few nuclei. The cells were all contained in the meshes of the network: they were somewhat tightly packed, inclining to be polyhedral in shape, containing a large nucleus about the size of a white corpuscle; sometimes two nuclei were present in one cell. The contents of the cells highly fatty and granular. Precisely the same appearances were found in the tumours in the liver.

The polypus in the pancreatic duct shows appearances of the same kind; only the walls of the alveoli were thicker and more fibrous, and the spaces between them narrower. The cells, too, seem more tightly packed and more abundant; for they only slightly exceeded in size the nuclei, which appeared in many places, when the section was thick, to touch each other. The nuclei were large, and stained deeply with carmine. In the head of the pancreas, the meshes were much narrower, and the walls of the alveoli very thick and fibrous. The cells show the same characters as above.

There are very few cases like this on record; chiefly, we believe, because the state of the portal vein within the liver is so seldom looked into. Frerichs speaks of five such cases coming under his own notice,¹ and Carswell gives a beautiful drawing of a polypus hanging from a cancerous nodule.² These, however, are

¹ Frerichs, *Klinik der Leberkrankheiten*, Braunschweig, 1861, Bd. ii. p. 278.

² Carswell, *Illustrations of the Elementary Forms of Disease*. London, 1838. Carcinoma, Plate iv. of first series, fig. 4.

all that we can find in print. Dr. Andrew, indeed, informs us that he has met with similar cases.

These cases must be wholly severed from cases of suppurative thrombosis of the portal vein, with which they were formerly confounded.

In the second case there were cancerous growths projecting into the bile-ducts. There was a growth (presumably the primary growth) in the rectum; and to these the clinical symptoms appear to have been chiefly referable. Her state on admission is thus described in the ward-book:—

Jane Stephens, 50, charwoman, admitted to Faith Ward, under Dr. Southey, May 8, 1876.

Has been married twenty years. Her youngest child is 18 years old. Has had no illness before the present. Family history good. Is now much emaciated. Flushes occasionally. There is well-marked cachexia. Has a slight cough. Appetite bad; much thirst. Temperature, 99.4° . Pulse, 126. Respirations, 22. Complains of no pain, only of increasing weakness. There is a continual watery diarrhœa, without tenesmus. Abdomen full and tympanitic. Skin harsh. Inguinal glands enlarged.

Rectal examination—A finger's length from the anus there is an irregular nodular painful growth, encircling the passage, and much occluding it; not easily broken down, not bleeding readily, not implicating the uterine organs.

She died on May 31st.

Examination thirty-nine hours after death.—Body universally jaundiced, not very deeply. No fat.

Pleuræ adherent at apices; slight excess of jaundiced fluid. At the apex of the right lung is a solid mass highly pigmented, fibrous, tough, with small yellow nodules scattered through it. Similar solid masses are scattered through this and the left lung, but they are not tough or pigmented to the same degree, and the yellow nodules form a more prominent part; some of them have broken down into cavities, very irregular in shape, and sinuous.

The pericardium contains about two ounces of a yellow fluid. Heart wasted, but otherwise natural.

Spleen natural size, soft; Malpighian bodies very well marked.

About two inches from anus the rectum shows on mucous surface a ring of warty growths, soft, discoloured, grey. No other new growths in alimentary tract. Stomach and pancreas natural.

Liver weighs 3200 grms. Gall-bladder collapsed, containing a little yellow mucus. The common duct slightly stained yellow. Before the branching of the hepatic duct, a tumour, the size of a split pea, springing from a large new growth in the liver, appears in the duct, having perforated its walls. A similar tumour, but larger, from the same source, is in a large branch of the left hepatic

duct. Behind these obstructions the ducts are greatly dilated. The portal vein and artery natural. The right lobe of the liver is filled by a cancerous growth, nodulated, arising apparently by confluence of small tumours; central part yellow, opaque; circumference white, nearly transparent. Some few nodules in left lobe. The liver-tissue in right lobe is somewhat jaundiced; that in left coarsely mottled, no jaundice.

Kidneys slightly granular on surface, with adherent capsule, otherwise natural.

Three Cases of Displaced Kidneys.

In the first case the displaced kidney was the right, and was found in the body of a little boy, about three years of age, who died on November 5, and was examined on November 8, 1875. The cause of his death was tubercular meningitis, and tubercle was likewise found in the lungs and liver.

On taking out the liver and intestines, the right kidney was at once seen to be displaced. It lay at least an inch lower than it should, there being a space of an inch between the kidney and the supra-renal body. Both supra-renal bodies were in their right places. The left kidney was in its usual place, and the vessels and ureter quite natural, and arranged as is common, save that the ureter was somewhat dilated. The right kidney, of natural size, lay on the psoas muscle, its middle part being on a level with the crest of the ilium. The hilus, by which the vessels enter and escape, is on the forepart of the kidney, about its centre. There are two right renal arteries, both arising near the same place, about half an inch from the origin of the inferior mesenteric. The smaller passes horizontally into the upper part of the kidney without entering the hilus; the other, passing over the inner part of the kidney, enters the hilus. This artery is accompanied by a renal vein which enters the inferior cava at the level of the artery. Another vein, much larger than this, arises on the outer side of the hilus, passes obliquely over the surface of the kidney to enter the inferior cava at the level of the left renal vein. This large vein is accompanied by a smaller vein which arises from the upper part of the kidney, and which enters the vena cava a little below the larger vein, nearly at the same place.

It is the rule in cases of displaced kidney that the supra-renal capsule should not descend with the kidney, but stay in its natural place; and it is also the rule for the vessels to arise at a part of the aorta on a level with the displaced kidney. It would also seem to be the custom for the hilus in these cases to be on the forepart of the kidney, and not on the inside. It is not, however, the rule for the right kidney to be displaced. It is far more

commonly the left. According to Klebs, this is as it should be, and that cases of displaced kidney on the right side should be viewed with suspicion, being most probably cases of movable kidney only.¹ This case, at all events, was not one of acquired movableness. The kidney was perfectly fixed when first seen to be displaced, and I look upon the origin of the renal artery below the inferior mesenteric as proof of the congenital origin of the displacement.

The specimen is preserved in the Museum.

The next case of displaced kidney was examined about three years ago, and we owe permission to publish its details to the courtesy of Dr. Gee. It was taken from the body of a man aged 24, who died with adherent pleuræ, dilated bronchi, and solid calcareous masses at the apex. The following appearances about the kidneys were found: The supra-renal capsules were in their natural place. The left kidney lay on the spinal column, between the common iliac arteries; not easily movable, that is, not a floating kidney. It was somewhat smaller and rounder in shape than natural. The pelvis lay between the kidney and spine. The arteries were three in number, and supplied from the right (!) common iliac; there was no trace of a left renal artery. The renal vein was about natural, passing upwards to the vena cava inferior. The ureter natural. The pelvis was somewhat distended. The right kidney was natural.

The last case of displaced kidney was taken from the body of a woman aged 41, who died in Hope Ward, under the care of Dr. Black. A large aneurysm of the aorta was found.

The account of the patient's illness is taken from the notes of the house-physicians (Dr. Bridges, and subsequently Dr. Shuter).

Susanna Feathers, aged 41, was admitted to Hope Ward, under Dr. Black, on September 27, 1875. There was a history of rheumatic fever fifteen years ago, with subsequent attacks of rheumatism; of dropsy four or five years ago, and of a cough for the last few months. On admission the face was puffy and lips anæmic; but there was no ascites nor anasarca of the legs. No abnormality of the liver was discoverable. Owing to some deformity of the chest, the physical examination was rendered difficult. The cardiac dulness extended to the right edge of the sternum, and to the left of the left nipple. There was a double murmur heard at the right base and down the sternum; it was loudest at the ensiform cartilage, and decreased towards the apex. There was, however, a systolic murmur heard at the apex, and to the outside. No albumen was discovered in the urine till a short time after admission, and then only a trace. The pulse had not

¹ Klebs, *Handb. der path. Anat.*, Berlin, 1870, p. 610.

the character of aortic disease, but it is noted that it could only be felt in the left wrist, owing to an abnormality of the right radial.

She was discharged on October 8th, and before that time the double basic murmur could only be heard in the erect position.

On June 14, 1876, she was readmitted. She had on that morning been suddenly seized with swelling of the face, lips, and eyelids. They were now hard and swollen; the head and neck also swollen. There was no œdema of the trunk or extremities. There was great dyspnœa, which increased in the evening to an alarming extent. A double aortic murmur was heard all over the front of the chest. There was impaired resonance at the left apex in front, and rather below the right apex behind. No air could be heard to enter the left side of the chest. On the 15th she complained of pain in swallowing, and the voice became indistinct. In the evening she passed urine for the first time during forty-eight hours, but only $\bar{5}$ ij., containing some albumen. On the 16th, at 5.20 P.M., she died suddenly.

The post-mortem examination made eighteen hours after death is as follows:—

Head and neck highly œdematous. Tongue, larynx, trachea, bronchi, and gullet quite natural. Neither food nor air passages seem pressed upon. Pericardium natural. On opening it, a projection from the aorta, which afterwards proved to be an aneurysm, is seen. The vena cava superior is found on dissection to be still pervious, but at its entrance into the auricle it is lined (at a part pressed upon by the aneurysm) by a thin clot of blood. The right side of the heart is quite natural. Both mitral and aortic valves are highly atheromatous; the latter are very stiff, calcified in some parts, and grown together. Immediately above the aortic valves the aorta dilates into an aneurysm the size of a large apple; from the right side of this a bulging, the size of a walnut, presses on the superior cava, and has nearly perforated. On the other side a bulging has formed, and presses apparently on the pulmonary artery. The left ventricle is much hypertrophied, and a little dilated. Aorta is atheromatous throughout. Lungs highly œdematous. Liver and spleen natural. Stomach and intestines natural. Right kidney large; capsule somewhat adherent; cortex very confused in structure. Both supra-renal bodies in natural place; a vein from the left passes into the inferior cava. The left kidney is found displaced into the triangle formed by the promontory of the sacrum and the psoas; almost, therefore, in the pelvis. It is much flattened, and about half the natural size. The hilus is turned forwards; an artery from the very point of bifurcation of the aorta runs into the hilus, and a vein passes into the inferior cava. The kidney

itself is small; not more than 70 millimetres long and about 20 thick. The left ureter is much dilated; it is pervious from the pelvis to the bladder, but a probe cannot be passed from the ureter into the bladder. Looked at from the bladder, the point of the probe can be seen under a thin membrane, but it cannot be passed into the bladder. The right ureter is quite natural and pervious.

Hydatid of the Left Kidney; the left kidney being contracted and pelvis dilated, the right hypertrophied.

Joseph Ferrett, aged 46, was admitted into Luke's Ward, under the care of Dr. Southey, on April 1, 1876.

Note on admission.—Had been well till eight months previously; he then fell ill with cough, dyspnœa, pains in back and shoulders, and palpitations, and for the last three months had laid up. Rheumatic fever twenty-four years ago. Had had bad colic. Is now fairly well nourished. Sits up in bed. No jaundice. Slight cyanosis during paroxysms of cough. Ordinarily has a slight cough. Sputa frothy, sometimes bloody; but there is paroxysmal dyspnœa, with orthopnœa. Legs have been œdematous for last three months. Urine, 1030; acid, albumen, and urates. Pulse irregular in volume; left smaller than right. No other symptom pointing to aneurysm.

Physical examination.—Area of cardiac impulse increased upwards, impulse most distinct in fourth interspace. Præsystolic thrill at apex. Dulness increased downwards and outwards, not upwards. Action irregular. At apex a double murmur (the diastolic portion most distinct), heard also outside the nipple, and faintly behind; proceeding upwards, it is reinforced at the right base by a double murmur, the diastolic portion of which is heard along the sternum, and best heard at the ensiform cartilage; the systolic at the ensiform cartilage. The left base is free.

By April 23d he had improved sufficiently to be up for two days, without cough or dyspnœa; but on May 1st it is noted that he is losing ground. On May 15th there was increased œdema of the legs and œdema of the lungs. Before his death (which took place on July 27th) there was thrombosis of one femoral vein, and sloughing of the tissues of the right thigh.

Examination fifteen hours after death.—Right leg œdematous; foot everted; a large sloughing sore on thigh and ankle. The sore on the thigh leads into a large gangrenous abscess, but does not communicate with the hip-joint.

Brain somewhat wasted; no softening. Arteries free from emboli.

Left pleura shows recent exudation; lung œdematous, with three or four patches of pulmonary infarction at base. Right

pleura obliterated; adhesions at base very tough and cartilage-like; lung œdematous. Pericardium adherent over forepart of heart. Heart very large; dilated, chiefly on left side. No clots in auricular appendices; a large clot, hollowed in its centre, at the apex of the right ventricle. Mitral orifice much dilated; the valves thickened and opaque. No granulations. The aortic valves let water slowly through; the valves themselves do not appear much diseased, though they show some thickening and opacity. No aneurysm of aorta. Liver large and nutmeggy; a large nævus (?) at the back. Spleen large and tough. Stomach and intestines natural. Supra-renal bodies natural. Right kidney large, weighs 320 grammes; surface smooth and red; cortex natural in striation. The left kidney very small. On tearing off the capsule, many small cysts are opened, the ordinary cysts of contracted kidney; one is the size of a hazel-nut. Surface very granular and red. On section, the kidney substance is very much narrowed, the pelvis being greatly dilated, and filling up the greater part of the inside of the kidney. The kidney substance measures little more than 7 to 8 millimetres. Its structure is lost, and it cannot be said which is cortex and which is medulla. Pelvis of kidney holds three small stones, the largest not bigger than a millet-seed; the ureter is dilated for two inches below the pelvis, but then becomes natural in size, and remains so till it enters the bladder. The bladder natural. Above the pelvis of the kidney, but not communicating with it in any way, is a cyst the size of a walnut. The walls are calcified, and it holds semi-transparent membranes folded up on each other, mixed together with a putty-like material. These membranes had a great resemblance, when seen with the naked eye, to hydatid membranes. Under the microscope they were found exceedingly fatty, but still showing a distinctly laminated structure. No echinococci or hooklets could be discovered.

The reason for the extremely contracted state of the left kidney is not at all plain. When hydatids are present in the kidney, they do not, as a rule, do much harm to the surrounding structures, except from their size. In this case the hydatid was small, and pressed on no important organ such as the ureter. Had there been any hindrance from the position of the hydatid to the escape of the urine, the dilatation of the pelvis and the atrophy of the kidney would be easy to explain; but the cyst was placed in the upper part of the kidney, and could apparently exert no pressure on the pelvis or ureter.

There were no hydatids in any other part of the body. Hydatids of the kidney are by no means common. In six years this is the first met with at St. Bartholomew's.

Addison's Disease in a Boy of 15.

James Donovan, aged 15, printer's boy, admitted June 29, 1876, to Mark Ward, under Dr. Andrew. The notes were taken by Dr. Wharry.

June 29.—Small and thin. Dark smoky-brown hair. Dark brown eyes; sclerotics slightly pearly; increased pigmentation of the choroid. General bronzing of skin, which is especially marked about exposed parts of body; also in axillæ, groins, round the umbilicus and nipples.

The mucous membranes of lips and cheeks have scattered patches and lines of bluish-black pigmentation.

There are a few black spots scattered over the body and limbs.

Tongue rather pale and moist. Breath has a slightly unpleasant odour.

Temperature, 98.4° . Pulse, 92; small and weak.

Thorax constricted at lower part. Crepitation at the end of inspiration at right base anteriorly and posteriorly.

Percussion impaired at left apex. Respiration coarse, and expiration prolonged. Moist sounds in left supra-spinous fossa.

Heart's apex-beat in fifth interspace, blowing systolic murmur at apex.

Abdomen.—Pain in left hypochondrium. No enlargement of spleen. No tumours.

Appetite poor. Bowels generally relaxed. Urine acid; specific gravity, 1020; no albumen. About 11 grammes of urea passed in twenty-four hours. Blood shows no appreciable increase in the number of leucocytes.

Sleeps well, but occasionally wakes up with headache.

He complains chiefly of increasing weakness, with loss of appetite, and usually vomiting soon after breakfast. Has been troubled lately by cough.

Has had three attacks of rheumatism in three years. The third attack came on three months ago, and the bronzing had set in since. Last September he began to suffer from vomiting, epistaxis, and giddiness, which has continued up to the present date.

One brother has enlarged cervical glands. One aunt had phthisis. Family history is otherwise good.

He died on July 22.

Examination thirty-nine hours after death.—Body wasted; rigor mortis still present. The face, chest, belly, thighs, and anus highly pigmented; small spots of intense pigmentation, size of pins' heads, over anus. These spots are almost black.

Nothing unnatural in brain. No pigmentation of tongue, pharynx, gullet, or windpipe. The thymus is persistent, and of

large size. The left pleura shows abundant small ecchymoses at back, both on parietal and visceral layers; one of these over the lower ribs is as large as a shilling. There are numerous old adhesions over right lower lobe. No fluid in either pleura. The pericardium shows old adhesions about vessels at base, and a large white spot on forepart of heart. The fluid is turbid, and of a brown colour. There exist numerous hæmorrhages, size of pins' heads, and smaller, over both layers of the membrane. The right side of heart natural. The left shows large hæmorrhages under the endocardium, covering the papillary muscles; the mitral valve thickened and opaque, with numerous granulations along free border. The aortic valves stiff, thickened, and opaque. Left lung natural. Right shows in lower lobe great dilatation of the bronchi, and some small patches of solid lung, tough, around. Rest of lung free from tubercle.

The liver much decomposed, but apparently natural. Spleen almost pulpy. Malpighian bodies very large.

Stomach natural. The intestines show the solitary glands and Peyer's patches much enlarged and very prominent. The mesenteric glands very large and somewhat hard.

On drawing down the upper part of the inferior cava, the semilunar ganglion and nerves entering it are found quite natural to the naked eye. There are several large lymphatic glands in the neighbourhood, embedded in the connective tissue around the plexus of nerves. Some of them are slightly pigmented. Both supra-renal capsules are enlarged, the right more than the left, and are adherent to surrounding organs—the liver and kidney, for example—but do not enter the substance of either gland. On section, the right capsule shows several small abscesses out of which a curdy pus flows, and the tissue generally softer and more translucent than is common in Addison's disease; the tissue also seems more elastic. On the left side the tissue is firmer and more opaque; it shows also small abscesses, but fewer in number than on the right side, and also some calcified nodules. The pus from the abscesses on both sides, when seen under the microscope, is very granular.

The kidneys quite natural.

Sections of the supra-renal capsule, when examined with the microscope, were seen to consist of broad and interlacing bands of white fibrous tissue; the intervals between which were filled up with fat cells.

Purpura Hæmorrhagica—Surgical (?) Kidney.

The clinical history of the following case is taken from notes lent by Mr. Verco, the house-physician.

John Macnamara, 17, a glassblower by trade, was admitted to John Ward, under Dr. Church, on April 13, 1876.

He had never been a strong boy, and for the last two or three weeks had suffered from 'sick headache' and occasional shivering. On the 9th was attacked with severe pain in the back; had then brought up some blood, and had continued to do so ever since. Next day had passed blood at stool, and this symptom also continued; blood appeared in urine on the same day. There had been no epistaxis.

None of his family ever suffered in the same way.

On admission there were hæmorrhagic spots on the face and trunk, but chiefly on the legs. Both conjunctivæ were injected, and the pupils widely dilated. There were black sordes on the lips and teeth, and hæmorrhages into the tongue and mucous membrane of mouth. The urine was thick and claret-coloured. Pulse, 100. Temperature, 99°. Heart-sounds clear. Some rhonchus in both lungs. No ecchymoses from pressure.

The sickness and the hæmorrhage from the bowels and the bladder continued till April 19th. About that time he began to pass large quantities of water, and the symptoms just mentioned began to disappear. On the 21st the right parotid region began to swell and become red and tender. On the 26th slight epistaxis began; next there appeared retention of urine, and symptoms of peritonitis; a 'coffee-ground' vomit was now brought up. On the 29th, at 1 A.M., the temperature, which had been rising during the last week, reached 106.3°; and at 8 P.M. that evening he died. Urine, when free from blood, was free from albumen.

The treatment adopted was as follows: On admission, Hst. terebinthinæ, ʒss. 6tis horis. On April 14, sulphuric acid and sulphate of magnesia. On April 17, pil. camphoræ c. opio ter die.

Examination thirty-nine hours after death.—Body blanched; numerous ecchymoses over legs, fewer over thighs, and still fewer over rest of body; a swelling the size of a small apple over right parotid region.

The brain and membranes quite natural. No hæmorrhages in substance; no excess of fluid in ventricles.

The swelling in the parotid region is due to an abscess which passes behind the ramus of the jaw under the pterygoid muscle, and below towards the branching of the carotid artery. It does not appear to be connected with diseased bone. Some of the glandulæ concatenatæ are cheesy.

The left pleura natural. The right universally adherent by old and recent adhesions. Both lungs highly œdematous. No tubercle. One of the bronchial glands on the right side is cheesy. The parietal pleura of the left side shows numerous small ecchymoses.

Clear fluid in pericardium not in excess. The visceral and

parietal layers show many small ecchymoses. The blood in all chambers of the heart well clotted; but the clots are rather pale. They do not look, however, like those seen in leucæmia. By accident they were thrown away before being examined with the microscope. A few hæmorrhages under endocardium; no disease of valves, or atheroma of mitral or aortic valves. The aorta above the valves is atheromatous in small patches. The walls of the heart flabby and pale. Under the microscope they show well-marked transverse striation; the granules seen in the fibres are soluble in acetic acid.

Many of the coils of the peritonæum are smeared with blood; there are no clots, but simply a uniform layer. There is no peritonitis. There is one large continuous extravasation under the peritonæum covering the pubic surface of the recti, the bladder, the rectum and pelvis. There are old adhesions joining the surface of the liver to the diaphragm and to the stomach. There is no ulceration of the stomach or small intestines. The large intestines are natural until the sigmoid flexure is passed; then the mucous membrane of the rectum becomes highly pigmented in patches of the size of a sixpence, and in two of these pigmented patches there is a loss of the mucous coat of the intestine.

The liver is pale, friable; acini very distinct, their central portions deeply coloured.

Spleen small, natural.

The right kidney somewhat larger than natural; surface smooth, ivory-like, here and there marked with stellate vessels. On section, the whole surface is very pale; scarcely any distinction as regards colour between cortex and medulla. The cortex without trace of striation, pale, but not increased in breadth. The pelvis of this kidney dilated, marked with many ecchymoses; ureter likewise larger than natural.

The left kidney considerably larger than natural. It presents the same characters as the left, but with these added: along the lines of the pyramids are numerous small bodies, usually distinct, at other times moniliform, none so big as a millet-seed. These appear also, but with less distinct arrangement, in the cortex. They appear in clusters on the surface, giving a roughened granular look and feel. Under the microscope these nodules appear to be collections of lymphatic corpuscles in the connective tissue of the kidney; but nowhere do they seem to have softened so as to form collections of pus.

The bladder somewhat thickened. The orifices of the ureters very plain. The triangle formed by these and the urethra, and the back part of the bladder, show a large uniform ecchymosis.

Poisoning by Sulphuric Acid—Parenchymatous Degeneration of Liver and Kidneys.

Alfred Stephen Dowling, aged 21 years, was admitted on Oct. 29, 1875, into Pitcairn Ward, after having swallowed an unknown quantity of sulphuric acid. He died on November 3d.

Examination thirty-six hours after death.—Weather, for time of year, warm.

Rigor mortis well marked. Body wasted. Eyes sunken. No jaundice.

No disease in brain or membranes. The left lateral sinus is absent. The tongue shows the papillæ circumvallatæ somewhat prominent, and a few round spots before these, which look as if due to desquamation of epithelium. There is no appearance of shrivelled epithelium on the velum palati or pharynx. The rima glottidis shows no œdema, but the trachea shows a lining of thick puriform matter, and the bronchial tubes exude a like substance. The lungs themselves natural. The heart natural. The walls of a good colour. Under the microscope the fibres show perfect striation. No ecchymoses on pericardium or pleura. Pulmonary arteries free from clot.

There is a small abscess between the gullet and the seventh cervical vertebra. The mucous membrane of the gullet is blackened, corroded at the lower end, and easily torn off from the tissue beneath it. The stomach slightly smaller than natural, the peritoneal coat of a blackish green along the great and small curvatures; large vessels run over the forepart. Within, the surface is of a black brown. Islets of mucous membranes stand out; between them the muscular coat is laid bare. Towards the pylorus the mucous membrane is more continuous, and may be stripped off in places. The duodenum shows the same appearance as the stomach—that is, the mucous membrane is left in islets for about three inches. Contents of stomach liquid of a dark brown black; so are contents of small intestine. In large intestine solid slate-coloured fœces. The small and large intestine look natural to naked eye.

Liver natural in size, of a red-brown colour. Under surface of green slate colour, which penetrates within for a few lines. A greenish colour of the tissue surrounding the great vessels. The liver-tissue itself is soft, readily taking the print of the finger. Section shows everted edge, dryness, and indistinctness of acini. Under the microscope the cells are all filled with fine granules. In about one-half the nucleus is visible, if the focus be properly adjusted. In the remaining half it cannot be found. On adding acetic acid, the fine granules disappear, and the contents of the cell

which remain are some coarse granules and the nucleus, which becomes visible in all the cells looked at.

The spleen rather small. Malpighian bodies and trabeculæ easily seen.

Supra-renal bodies natural. The kidneys of natural size, perfectly smooth surface. On section, marked contrast in colour between the pyramids and the cortex. The cortex dull yellow; the striation gone, or to be seen only by close inspection; mottled here and there with large vessels, and the Malpighian bodies. The pyramids of a pale purple. Consistence of kidney somewhat less than natural. The epithelium finely granular, but the nucleus in all cases visible.

Aorta almost natural.

Hydrophobia, with Fever and Furor. Death on the third day from first sign of constitutional disturbance, the fifth or sixth from the first sign of recrudescence.

Henry Sewell, aged 13. No sign of puberty. Admitted September 5, 1875, to Radcliffe Ward, under the care of Dr. Black.

For the clinical notes we are indebted to Dr. Bridges.

History (as subsequently obtained).—Was bitten by a strange cat about two months before. This cat could not be traced. There was no reason to suppose it was mad. It had kitted in the lodging of a woman who had offered a penny to any one who would get rid of it for her. In earning this penny the patient received a bite on the ball of the left thumb. He came on February 14th to the Hospital Surgery for treatment, where his name appears among the entries—‘William Sewell; scratched wrist; dressed.’ The wound was not cauterised. The poison remained latent till Thursday, September 2d, when his left hand swelled. On Friday he complained of pain in the elbow, the pain gradually passing up the arm. On Saturday, September 4th, the pain was in the shoulder, and towards evening he showed signs of constitutional disturbance. He was noisy through the night, and refused drink, tossing the mug away from him. On Sunday, September 5th, he was brought to the Surgery in the afternoon, and I was called to see him.

His skin was hot and moist, his pulse high, the expression of his face anxious, as if he was in great pain and apprehension; and judging from his appearance and the pain in his shoulder that he must have rheumatic fever, I admitted him at the request of his mother.

About two hours afterwards I paid him a visit in the ward. When I went up to the bed the boy was lying straight and supine.

On my asking him some question, he seemed unable to answer me, but threw his body into a curved position towards the left side, his legs remaining straight in the bed, and seemed to struggle with something in his throat. Recognising the genuine nervous character of the convulsion, I examined his heart, which beat very violently, regularly, powerfully, and frequently, so that it was difficult to believe that it was not hypertrophied; but there was no physical sign of either enlargement or inflammation. While I examined him, he said that he suffered very much at the stomach and heart, and laid his hand on the parts. The Sister then told me that he had had a convulsion when he drank, so some drink was offered him again. He sat up with determination, as if about to attempt something difficult, and after swallowing three mouthfuls, sank back in a convulsion. The upper part of his body shuddered, and he then beat his stomach violently and rapidly with his left hand, and cried out, 'Oh, it's too much; I have taken too much.' All this time the action of the heart remained unchanged, so that angina pectoris and pericarditis being both excluded, there remained no disease to account for the symptoms but hydrophobia. Another spasm was shortly afterwards set up by the coughing of a patient in the next bed, and during this he used his right hand as he had before used his left. I injected him with morphia, and left him for the night, making arrangements for his removal to Casualty Ward. The body heat at this time was 101° ; the respirations 24; the pulse 100 per minute.

Monday, 6th.—He slept or remained quiet till 4 A.M., when I was called to see him. I found him on all fours in bed, clutching at the ticking with his hands. He had bitten the Sister in the hand. He was again injected with morphia, and removed to Casualty.

10 A.M.—Pain in the shoulder and heart. Has had no more spasms or convulsions. Seems to swallow pretty well. Ordered minced meat.

11 A.M.—Dr. Brunton visited him with me. He drank before us. He had a difficulty in swallowing, and a succeeding spasm.

3 P.M.—Dr. Gee saw him, and in the absence of Dr. Black took charge of the case. He saw him swallow once or twice without spasm, and considered the cause of the phrenitis uncertain, and ordered pot. brom. gr. xv., liqor morph. hydroch. m. v. quartis horis.

4 P.M.—Seen by Dr. Legg with me. He was then conscious, but raving. He swallowed two spoonfuls with determination and difficulty; no spasm.

5 P.M.—He was now in a padded bed, and became very violent, especially when food was offered to him. He saw visions and had hallucinations. He thought the ceiling was falling on

him, and that there were animals about. Mention of drink excited him. In the forepart of the day, when asked to drink, he behaved in this way: 'Are you thirsty?'—'Yes.' 'Will you have some drink?'—'No.' 'Try and take a little.'—'Very well.' Then he would sit up in the bed, seize the cup with both hands, swallow a mouthful, and desist, saying he could do no more. But now he absolutely refuses to drink; and if a cup or spoon is presented to him, he dashes it across the room, darting at it with a certainty that it is impossible to avoid. Of the medicine ordered at 3 P.M. he never took any.

The pulse has been irregular all day, and wavers more every hour.

7 P.M.—I saw him offered drink. He made great faces, puffed out his cheeks, and grabbed at the vessel; foam collected at his mouth, and he vomited. He was raving on all subjects, using a great deal of language and metaphorical expressions, which were sometimes acute, sometimes nonsense. He spits now at anybody near him, and is excited by a bright light.

8 P.M.—About this time there was a remarkable lightening of the symptoms: his fury seemed to leave him for about half an hour, during which time he spoke of his family, and of how they would regard his death. He mentioned his schoolfellows with affection. The excitement returned gradually, and he relapsed into a more furious condition than before.

10 P.M.—He was again injected with morphia, gr. $\frac{1}{4}$. The body heat was 101°.

11.30 P.M.—101.2°. Pulse, 168. Between the exacerbations of his rage he appears much exhausted. He died shortly after midnight.

In this case, which seemed to begin with occasional spasms and to pass on to a continuous fury, which, as it became more severe, was interrupted by intervals of apparent exhaustion, and once by a complete remission, the following facts were also observed. The sound of water did not affect him. There was not at any time any proof of inability to swallow. A draught of air did not seem to provoke a convulsion. There was no priapism. The convulsions set up by drinking and other causes decreased (or ceased entirely, as far as I saw) as the rage came on. He died of exhaustion between the exacerbations of the rage (and not of spasm, as Boerhaave has it). The respiration was irregular. He raved a good deal about cats. He went on all fours, and made noises which might very well have been mistaken for a dog's howl (thus illustrating the popular beliefs). He bit one of the Sisters on the back of the hand, and tore a piece of skin out of one of the house-surgeons' wrists with his finger-nails. He took

a violent dislike to me, attributing much of his disease to the morphia injections.

The wound in the Sister's hand, which had penetrated the skin, was cauterised about a fortnight afterwards. There were no symptoms. The wound in the patient's wrist was not discoverable during life; after death it was distinguished by a slight blush from the general pallor of the body.

The verdict of the coroner's jury was given before the post-mortem examination of the body.

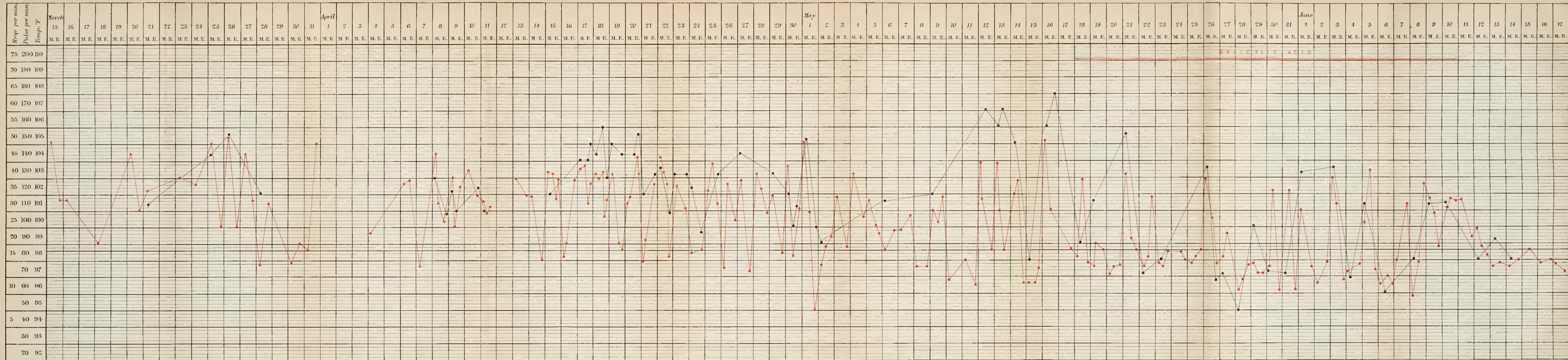
Examination thirteen hours after death.—Two scars, such as might have been caused by the canine teeth of a cat, on the left wrist.

Body very thin; no fat under skin or in omentum.

No naked-eye appearances of disease in spinal cord, brain, or membranes, nor in pharynx, tonsils, œsophagus, or alimentary tract. The pleuræ were slightly adherent by old adhesions. Lungs, heart, liver, supra-renal capsules, and kidneys quite natural.

The spinal cord was delivered to Dr. Gowers for examination with the microscope, and the following is his report:—

‘The cord of the case of hydrophobia I have found a good deal damaged by the post-mortem disintegration which occurred before it reached me. The examination shows, however, that there is little abnormal in the cord. The chief change is that the fibres in the lateral columns near the anterior horns are in places separated by clear areas, which sometimes present an irregular vacuolation. There is, however, no degeneration of the nerve elements. In the lower part of the medulla there are marked vascular changes, the vessels being surrounded by small cells resembling leucocytes, which can also be traced into the adjacent tissue. This is especially marked in the neighbourhood of the hypoglossal and glosso-pharyngeal nuclei. The large cells of these nuclei have, many of them, a more granular appearance than the similar cells elsewhere. The minute post-mortem division of the upper part of the medulla and pons (post-mortem disintegration) made it impossible to ascertain the state of the fifth and other nuclei.’



The red line indicates the temperature, the black line the pulse-frequency.

TWO MEDICAL CASES.

BY

R. WHARRY, M.B.

1. *Meningitis.*

In the following case, the illness beginning as urticaria, soon developed symptoms of meningitis, and the appearance of morbid changes at the apices of the lungs suggested a probability of the meningitis being tubercular. The course of the disease was protracted, the patient only beginning to rally when brought to a state of extreme emaciation. Treatment was chiefly directed towards controlling the temperature. Digitalis seemed to have some effect at first, but did not maintain it beyond a few days. Bathing the patient was resorted to for some time, and the temperature was observed to fall more rapidly when brandy was administered during the bath. The effect of salicylic acid upon the temperature was most decided, but this after a time seemed to lose its influence to a certain extent.

F. T., a girl aged 17, was admitted to Mary Ward, March 18, 1876. About one month previously she had fainted, and had been ill ever since with loss of appetite and general languor. About a fortnight after, a red rash appeared on the arms and legs, which spread in patches.

Condition on admission.—A thin anæmic girl, with a pale complexion and a dark rim round the eyes. Thoracic viscera healthy. Slight retraction of the abdomen. There is an indistinct mottling on the arms and legs, varying in intensity. The surfaces of tibiæ are slightly irregular, and they are tender at night.

Milk diet and arrowroot were given for two or three days, and then it was exchanged for half meat diet. Haust. calumb. alk. c. ammoniæ carbonatis, gr. iv. ter die was ordered.

March 15.—Temperature, 105·1°. Pulse, 130. No splenic

enlargement. No physical conditions can be found to account for the high temperature. 10 P.M., temperature, 101°6'.

The next morning the temperature was 101°6', and on the day after 99°.

March 21.—Eyes suffused, especially the left. Tongue moist and furred. Temperature, 102°2'. Pulse, 114. A few scattered red patches, slightly elevated, have appeared on the arms.

Six ounces of wine were ordered, and tinct. digitalis m. viiss. added to her medicine. The following day, haust. quiniæ gr. v. ter die was given instead of the previous medicine.

March 25.—There is an increase of the rash. Temperature, 105°. Pulse, 144. No enlargement of spleen. 10 P.M., temperature, 100°.

March 26.—Complains of pains in the fingers. The joints appear slightly swollen. Temperature, 105°4'. Pulse, 156. 10 P.M., temperature, 99°.

March 27.—Has some cough. Resonance impaired at the apex of each lung anteriorly. Temperature, 104°4'. 10 P.M., temperature, 100°6'. She moans during her sleep.

March 30.—Complains of much pain in the knees. Tongue clean and red. Slight swelling of the knee-joints, with obscure fluctuation and considerable hyperæsthesia.

The quinine draught was to be given every four hours instead of three times a day.

April 4.—Tongue pale and œdematous. Bowels constipated. The knees are much better. There is pain in the knuckles of first and second fingers of the left hand.

April 7.—Complains of headache. Pain in the chest and over the sacrum.

April 8.—Slept badly. Bowels relaxed. Pain in the left knee. Great hyperæsthesia of whole of the surface of the left tibia, which is aggravated at night. There is nothing unnatural in the outward appearance of the left leg. There is no sweating.

The quinine draught to be exchanged for H. A. A. c. Camphora.

April 10.—Slept poorly. Bowels constipated. The pain and tenderness of left tibia have gone. Both legs are desquamating. Great pain and tenderness in the sub-occipital region.

April 11.—Pain and excessive tenderness throughout the whole length of the spine and in the loins. Abdomen is retracted. There is nothing unnatural about the appearance of the back. Ordered liq. arsenicalis m. iv., infus. gentian. co. ʒj., 6tis horis.

April 15.—Pain and hyperæsthesia along the spine have continued with short irregular intervals. Temperature, 103°3'. Pulse, 120; small and weak. Pain in the spine and knees. The

patient was put into a bath for eighteen minutes, the water being cooled down from 98° to 78° . After the bath the patient was very pale, and shivering severely. Temperature, 101.7° . Pulse, 120.

April 16.—Great pain and tenderness in the knees. Severe frontal headache.

April 17.—Temperature, 103.7° . Pulse, 140. Put into a bath for thirty minutes; water cooled down from 95° to 90° . Temperature, 101.4° . Pulse, 140.

April 18.—Slept fairly. Takes but little food. Tongue moist. Complains of frontal headache, and pain in the neck. Urine neutral; specific gravity, 1006; no albumen. There are signs of limited consolidation at the apices of both lungs, being more marked on the left side (impaired percussion; vocal vibrations increased; prolonged expiration). The eyes examined by Mr. Power. Slight optic neuritis. Vessels small and tortuous. Hazy around margin of disc; the vessels make a perceptible curve on entering the disc. Disc slightly œdematous. Media clear. No deposit.

4 P.M.—Temperature, 103.3° . Pulse, 160. Put into a bath for twenty minutes; water at 95° , cooled down to 75° . Temperature, 100.6° .

April 20.—Has been much better since the 18th. Two eggs were added to the diet. The medicine was discontinued.

10 P.M.—Temperature, 104.2° . Pulse, 144. Severe frontal headache. Put into a bath for ten minutes; water cooled down from 85° to 76° . Great tremulousness; sighing respiration. Temperature, 103.2° . Pulse, 156; smaller.

April 28.—During the past week she has complained much of headache and pain in the suboccipital region. The temperature and pulse have varied much. Face slightly flushed. Considerable headache. The pain in the neck has gone, but the knees are very tender and painful. The left knee is swollen, and there is a small highly-sensitive patch of redness on the outer side.

April 29.—Both legs are drawn up. There is pain in, and effusion into, both knee-joints.

April 30.—There is no pain in the knees, but still some slight accumulation of fluid. Temperature, 103.7° . Pulse, 120. She was put into a bath for fifteen minutes, the water being cooled down from 95° to 85° F. After the bath, temperature, 98.2° ; pulse, 100. During the bathing two ounces of brandy were given.

May 2.—Very great emaciation, slight headache, and slight pains in the knees. Tongue red, with aphthous patches. Appetite bad. Bowels open yesterday. Temperature, 97.7° . Pulse,

90; small and weak. *Subsultus tendinum*. The physical signs in the lungs remain stationary.

May 3.—Ten grains of chloral hydrate were given to allay the pain, and essence of beef oss. was added to the diet.

May 9.—General condition during the past week has improved. The average height of the temperature has been materially lower. She complains of pain in the neck, abdomen, and knees. There is great hyperæsthesia along the shaft of the left tibia. Chloral was given in ten-grain doses for the relief of the pain, with a certain amount of success.

May 12.—Temperature, 103.9° . Put into a bath for fifteen minutes, the water being cooled down from 95° to 75° . Two ounces of brandy were given while the patient was in the bath. Temperature, 101.7° . Pulse, 170.

May 13.—Temperature, 103.9° . Pulse, 160. Vomited yesterday and slightly this morning. Put into a bath for twenty minutes; cooled down from 95° to 75° . Two ounces of brandy were given. Temperature, 101° . Pulse, 170.

May 16.—Temperature, 105.2° . Pulse, 160. Crying out with pain in the knees. Put into a bath for twenty minutes, the water being cooled down from 95° to 75° . Two ounces of brandy were given. Temperature, 101° . Pulse, 160. At 2.30 P.M. the pain had disappeared.

May 18.—Temperature, 98.2° . Pulse, 90. Feels much better, and is free from pain. 3 P.M.—Temperature, 102.9° . Great pain in the head.

Salicylic acid, \mathfrak{Dj} .; glycerini, $\mathfrak{5j}$. ex aquâ, nocte manequ, was given.

May 26.—The patient's condition has considerably improved during the past eight days. The average temperature has been much lower, and there has been very much less pain. To-day there is pain all over the right hand. There is a small erythematous hyperæsthetic patch at the left elbow. The physical signs at the apices of the lungs are stationary.

June 1.—Temperature, 101° . Pulse, 132. Hyperæsthesia over the lumbar spines and splenic region.

June 8.—Temperature, 95.8° . Pulse, 80; irregular in rhythm. Passed a bad night owing to pain. No pain at present.

June 9.—Temperature, 101.7° . The patient has been crying with pain all night. The pain began at 8 P.M. in the head, neck, spine, and legs, and it became aggravated towards 2 A.M. The face is flushed; the right pupil slightly larger than the left.

June 10.—Temperature, 101.2° . Pulse, 112. Passed a bad night with hyperæsthesia and pain in the thighs and neck, had two ten-grain doses of chloral, but vomited slightly after

the second dose. The salicylic acid to be discontinued, and hst. ammon. acet. c. camphora given instead.

From this date the symptoms rapidly subsided, but the patient was left in a very weak state, extremely emaciated. The temperature varied still, but for more than one month rarely rose to the normal standard. The pulse varied between 60 and 70. She gradually gained strength, the physical signs at the apices of the lungs became less marked, and eventually the patient was sent to the Convalescent Home at Walton-on-Thames, August 2.

A chart of the temperature is subjoined.

2. *Leukæmia Lieno-lymphatica.*

The following case of leukæmia lieno-lymphatica presents many features of interest, and not least among them is the fact that at the time of the patient's death, his son, a child of two and a half years, was under observation affected with purpura, having an enlarged spleen. The large doses of quinine with which the treatment was commenced in no degree reduced the size of the spleen, and but slightly affected the temperature and pulse—reducing the former, accelerating the latter. The effect of the phosphorus was more satisfactory; the spleen became markedly diminished in size under its influence. The patient, in giving his history, distinctly referred its commencement to a blow on the left shin four months previous to his admission.

Wm. C., aged 39 years, a printer, admitted into Mark Ward June 1, 1876. Face pale. No marked cachexia. Eyes slightly prominent; sight good; slight atrophy of the choroid. No peculiar intraocular changes; slight sallowness of the conjunctiva. Temperature, 99.6°. Pulse, 100; small and soft. Slight enlargement of the glands of the neck on both sides, and also in both axillæ. The lungs are fairly natural. The heart had a systolic murmur at the base, with an accentuated second sound at the left base (pulmonary).

Abdomen.—A large tumour occupies the left side of the abdomen, from Poupart's ligament to the seventh rib; its anterior margin extends to the middle line, and presents three notches; its posterior margin is felt about $2\frac{1}{2}$ inches from the vertebral column. The tumour is solid, and does not move with respiration. On the right side the liver dulness is extended upwards to the upper border of fifth rib, and also extended slightly downwards. The inguinal glands are slightly enlarged.

The left tibia seems somewhat thickened, so also do the bones about the ankles. There is an old irregular scar on the left tibia, and another below the right ankle.

The blood contains a very largely increased proportion of leucocytes; they are large, well formed, with clearly-defined outline.

The urine.—Three and one-half pints passed in twenty-four hours; pale, turbid, slightly albuminous; no tyrosine could be detected; specific gravity, 1018. Bowels are rather constipated.

History.—Four months ago he received a blow on the left shin which caused him to be laid up for a month. Erysipelas set in; it spread up the left leg and down the right. Soon after returning to his work he felt a tumour in his left iliac region about the size of an egg. The abdomen gradually enlarged, and he began to have difficulty in stooping, with attacks of giddiness. About a fortnight before admission the giddiness became almost constant while walking about. Upon one occasion he fainted. He complains of a sense of tightness, but of no pain, in the abdomen. There is no history of any hæmorrhages. Acute rheumatism four years ago. Syphilis fourteen years ago. Family history is good.

Ordered milk diet; half a pint of essence of beef; four ounces of wine. Olei ricini, ʒj.; tincture opii, m. v. statim. Haustus ferri et quiniæ citratis 6tis horis.

June 3.—Temperature, 101.1°. Ordered full diet; milk, one pint; wine, six ounces. Haust. quiniæ (gr. xv.) bis die.

June 4.—Temperature, 98.2°. He bears the quinine well; there are no signs of cinchonism.

June 5.—Temperature, 98.8°. Pulse, 90. Ordered confect. sennæ ʒj., to prevent any straining at stool. Haust. quin. (gr. xxx.) bis die.

June 6.—Temperature, 98.2°. Pulse, 92. Deafness and slight singing in right ear. No headache. Slight gastric catarrh.

June 7.—In addition to the other symptoms there is frontal headache, flushes of heat, and sweating.

June 8.—Temperature, 98.4°. Pulse, 96. Vomited after taking his medicine. Severe gastric catarrh. The spleen seems to have increased in size and slightly altered its position. The superficial veins are more marked. Frequent sighing. Ordered brandy, ʒij., instead of wine. Haust. quin. (gr. v.) ter die.

June 9.—Temperature, 98.8°. Pulse, 88. The symptoms of cinchonism have very much diminished.

June 10.—Ordered hst. quin. (gr. xxx.) bis die. Temperature, 99.2°. Pulse, 86.

June 11.—Temperature, 98.8°. Pulse, 96. Cinchonism has returned. There is constant 'sawing' pain at the upper and back part of the tumour. The quinine to be reduced to gr. v. ter die.

June 12.—Temperature, 99.5° . Pulse, 96. Abdominal measurements: girth at umbilicus, $34\frac{3}{4}$ inches; at $2\frac{1}{2}$ inches above umbilicus, $36\frac{5}{8}$ inches; at $2\frac{1}{2}$ inches below umbilicus, $33\frac{5}{8}$ inches.

June 13.—Temperature, 99.6° . Pulse, 92. Less pain in the tumour.

June 14.—Better this morning. Slept well. Bowels open twice. The pain is much less, and he can turn on his side. Temperature, 98.2° . Pulse, 96. Slight cough. Yawning and sighing at short intervals.

3.40 P.M.—Lying on right side talking to fellow-patients, when, apparently without cause, he began to look at his left hand, turning it backwards and forwards; he then laughed convulsively; a mug dropped from his right hand, and he tried to speak, but could not.

10.45 P.M.—Temperature, 97.3° . Pulse, 92 (quiet, and its character not changed). Complete loss of power in right arm and leg, which are relaxed, and free from any trace of rigidity. Mental faculties confused; frequent yawning, and passing left hand over forehead. He lies on his right side. The skin is bathed in sweat. Has passed motions and urine in bed. When questioned, can only answer 'No' or 'Yes' irrelevantly. The food dribbles out of right corner of the mouth. The cardiac murmur at the base is changed, being less distinct.

June 15.—Temperature, 98.7° . Pulse, 96. Intellect confused. When asked to put out his tongue, he opens his mouth and shuts it again without protruding it, but occasionally he does protrude it. The wrinkles on right side of forehead are gone, and he frowns only with the left side. The mouth is drawn to the left, and he smiles on the left side of his face only. Right eye does not close completely in sleep. The spleen is markedly increased in size. The limbs on right side are relaxed and powerless; reflex excitability is exaggerated. To all questions he answers 'Yes.'

Ordered milk diet; essence, a half-pint; brandy, \bar{z} ij. One of Tisy's phosphorus perles twice a day.

His general condition remained about the same during the next week. The spleen began perceptibly to decrease in size. The blood was again examined under the microscope, and apparently the proportion of leucocytes had diminished.

June 22.—Phosphorus perle j. ter die.

June 24.—The following measurements were taken of the girth of the abdomen: $32\frac{1}{8}$ inches at the level of the umbilicus, $31\frac{1}{2}$ inches at a level $2\frac{1}{2}$ inches below the umbilicus; showing a marked diminution.

The patient was told to write his own name, using the left hand, and the results of the attempt, with the exception of the first letter, "W," were completely illegible.

June 25.—His intellect is much clearer. He wants to say something, and says, 'To-to, ta-ta-ta.' The abdomen is much smaller. The spleen is much smaller. There is no increase in the proportion of leucocytes. There is general emaciation. Temperature, 96.6°. Pulse, 88.

June 29.—Pulse, 88. Temperature (in both axillæ), 96.7°. Apathetic; countenance dusky; face pinched. The spleen is smaller.

June 30.—Pulse, 100. Temperature—right axilla, 97.4°; left axilla, 96.5°. Measurement of abdomen at the level of the umbilicus, 31 inches; at a level $2\frac{1}{2}$ inches below umbilicus, $30\frac{1}{4}$ inches; at a level $2\frac{1}{2}$ inches above umbilicus, $32\frac{1}{2}$ inches. Again a considerable diminution.

July 3.—More cheerful. Can utter a few more words. Bowels are relaxed. Temperature, 98.5°. Pulse, 108. Tenderness on pressure over the spleen.

July 4.—Progressive and rapid emaciation. Subsultus tendinum. Temperature, 98.8°. Pulse, 132.

The patient died July 6, 1876.

Post-mortem examination six hours after death.—Surface pallid. Great emaciation.

Lungs.—Emphysematous, with a small amount of hypostatic congestion and œdema.

Heart.—Pale and rather flabby. The mitral valves are thickened, with vegetations on the auricular surface of the margins. The valves were partly adherent. Just above the tricuspid valves, and projecting from the wall of the right auricle, is a small tumour, slightly larger than a pea.

Abdomen.—The liver was somewhat enlarged, pale, and of natural consistence.

Spleen.—Greatly enlarged. Firmly adherent to the diaphragm, omentum, and abdominal wall. The capsule is thickened. There are three notches in the anterior margin. It extended from the fifth interspace to one inch below the umbilicus. Its texture is unusually firm. Its substance is thickened. The increase in bulk is general. There are two large triangular masses in a semifluid state (apparently disintegrated infarcts) occupying portions of the spleen, and several smaller ones (pea-sized) scattered through the substance.

The kidneys contained much phosphatic deposit in the pelvis, especially the right, which also contained a small mulberry calculus. Between the striations of the pyramids were rows of

gritty matter, probably phosphates; and scattered throughout the cortex were minute infarcts, varying in size from a pin's point to a pea.

The supra-renal capsules are natural.

The mesenteric and retro-peritoneal glands are considerably enlarged, some being as large as grapes, and resilient to the touch.

The pancreas is natural.

The intestines natural in texture, but contain large quantities of dark red bile-tinted fluid.

The stomach also contained some similar fluid.

The skull-cap had a small rounded exosteal growth of compact tissue, and on the inner surface of the post-parietal regions were two small irregular bony projections.

There were several ossific spiculæ on the inner surface of the dura mater in connection with the Pacchionian bodies.

Brain.—Generally œdematous and soddened. The whole of the tract supplied by the left middle cerebral artery was softened, yellow, œdematous, in places cheesy and even gritty, presenting a granular appearance. A firm white embolus completely plugged the left middle cerebral artery close to its origin.

The vessels of the brain contained an almost colourless fluid full of minute white beads, apparently agglomerations of leucocytes.

Microscopical examination. (Sections prepared by Mr. W. Griffith.)—The tumour in the wall of the right auricle was composed of obscurely-fibrillated tissue, with collections of hæmatoïdin scattered through it, and a few blood-vessels. There was slight granular degeneration of the muscular fibres of the heart. The kidneys showed minute capillaries ruptured, with accumulations of leucocytes, and generally throughout the course of the capillaries were numerous exuded leucocytes. The liver appeared fairly healthy, with a few leucocytes in the course of the vessels. The mesenteric glands and supra-renal capsules presented no unusual appearances. There seemed to be increase in the matrix of the spleen.

F. C., aged $2\frac{1}{2}$ years, son of Wm. C., was admitted into Mary Ward, July 6, 1876, shortly after his father's death. His skin was pale and waxy. Complexion pale. Conjunctivæ and lips very bloodless. Pupils equal. There is slight excoriation of the upper lip, and a little coryza. There are several small ecchymoses scattered about the face. The extremities are pale and puffy. The lungs are healthy. The heart has a slight blowing systolic murmur at the base. The spleen is enlarged, and can be

felt beneath the ribs, descending with inspiration. There are one or two small ecchymoses scattered about the lower part of the abdomen and the thighs. There was no glandular enlargement. The child has always been delicate, and lately has been subject to bleeding from the nose.

An examination of the blood showed no increase in the colourless blood-corpuscles.

Oil of turpentine was given for two or three weeks, and there being no recurrence of the hæmorrhage, tincture of perchloride of iron was substituted.

The patient left the Hospital, August 11th, improved in general health, and having had no hæmorrhage for more than a month. The spleen, however, was still large and palpable.

THE
DENTAL DEPARTMENT AT THE HOSPITAL.

BY
ALFRED COLEMAN.

Following the example of some of my colleagues who have recorded in the Transactions the amount and character of work accomplished in their special departments at the Hospital, I herewith give, as a specimen of that carried out in the Dental Department, the results of the past year:—

Extractions	564
Extractions under nitrous oxide and ether	142
Stoppings	86
Regulation and advice	47
Miscellaneous	38

Of the extractions, which form so large a proportion in the treatment at the department, it may be remarked that they were all cases in which any more conservative treatment was inapplicable; and in the large proportion of cases, the roots only of teeth, loose teeth, or in a few cases sound ones, were removed because they kept up irritation in the case of epithelioma in the tongue.

The permission by the authorities of the Hospital to employ nitrous oxide in the department has proved a great boon to the patients, and the skilful manner in which it is administered in combination with ether by Clover's apparatus by the anaesthetists, often permits of ten, twelve, or even more roots or teeth being removed at one administration.

In regard to the teeth saved by filling, the number of which in proportion to those sacrificed appears so small, it must be borne in mind that each of such operations will average about three-quarters of an hour, and that without a very large staff of officers, supplemented by a still larger number of efficient pupils, it is impossible to undertake more than a very limited number of cases. Still, as a large proportion of the above have

been carried out, and on the whole very successfully, by students of the Hospital, it is to be hoped more may in future be effected in this most desirable direction. In many cases, however, where such aid could not be rendered in the department, arrangements were made for the patients being treated elsewhere.

Amongst the cases of special interest may be mentioned—

1. Two cases of trismus, due to the irritation set up by impacted wisdom teeth.

2. A large number of cases of fistulæ in the cheek, the result of dental abscess.

3. Six cases of stomatitis, all of which soon recovered under chlorate-of-potash treatment.

4. Three cases of dentigerous cyst, treated as described in a former number of the Transactions.

5. Two cases of transplantation—viz., when sound front teeth removed to relieve overcrowding, were transferred to sockets of other patients from which roots of teeth were extracted. Both cases successful.

6. Two cases referred to the surgeons of the Hospital—one of obscure growth in the antrum; the other, malignant disease of lower jaw.

7. Besides the construction of several plates for the treatment of irregularity, mechanical appliances were supplied for the following cases: Two for closing up orifice into nares, and supplying teeth and substance lost after removal of superior maxillæ; two obturators for closing cleft palate; and three inter-dental splints for treatment of fractures of the maxillæ, two of which proved very intractable cases, owing to the complication of external wounds. All these appliances were constructed by my friend Mr. Lyons at his own cost, and in describing the work done in the Dental Department during the past year, I should be doing a great injustice were I to omit stating how greatly any success we may have had is due to his energy and skill.

PROCEEDINGS
OF
THE ABERNETHIAN SOCIETY

FOR WINTER SESSION 1875-76.

October 14, 1875.

Mr. Willett delivered the introductory address.

Dr. Moore showed the following pathological specimens:—

1. The heart from a child (who was cyanotic during life) with adherent pulmonary valves, the orifice being the size of a goose-quill. The septum ventriculorum was incomplete.
2. A piece of the temporal bone with disease of the mastoid cells. The patient had a cyst in the cerebellum.
3. Part of the hind leg of a dog showing osteo-arthritis.

October 21.

Dr. Shuter showed the following microscopical specimens:—

1. A longitudinal section of the supra-renal capsule and kidney of a rat (injected).
2. A longitudinal section of a rat's tail.

Mr. Bruce Clarke showed a large hydatid cyst.

Dr. Shuter read a paper on 'The Causes of Progressive Muscular Atrophy.'

October 28.

Mr. Bruce Clarke showed some specimens of echinococcus.

Dr. Wharry showed a specimen of a kidney with fatty and fibroid change and capillary embolism.

Mr. Schofield showed some microscopical specimens.

Mr. Butlin read a paper on 'Cancer of the Breast following Eczema about the Nipples.'

Of course you are all aware that many attempts have been made to discover the causes of cancer; that numberless theories have been put forward by different observers at various times to account for the occurrence of cancer; that, nevertheless, the question is still almost if not quite as undecided as it was in the infancy of pathology. Perhaps the most probable of all the theories which have been advanced is that so warmly supported by the greatest of German pathologists, Virchow,¹ which ascribes the formation of cancer to some actual injury or irritation. The subject is ably argued by this observer, and particular care is taken to point out how the most exposed parts, whether internal or external, are always those most liable to be effected by cancer. Of external parts, the lower lip, the various organs of generation; of internal parts, the tongue, the œsophagus, stomach, and rectum. We place so much reliance upon these theories that we are always most careful to remove as far as possible every source of long-continued irritation, and to heal ulcers about the mouth, anus, &c., lest these perchance should give rise to the much and justly dreaded disease; for we are obliged to admit that when once a cancer has formed it is absolutely incurable. We may remove it by any means in our power: it will inevitably recur at some nearer or more distant point, will in time affect the lymphatic glands, and will finally, if time be given it, prove fatal. What we want to know is some sign or signs which shall foretell that cancer will certainly occur in some particular part of the body at no very distant period. Had we such signs, we should not hesitate to remove if possible the whole of the part expected to be attacked.

In the Hospital Reports last year (1874) Sir James Paget pointed out such a precursory sign. He says, 'I believe it has not yet been published that certain chronic affections of the skin of the nipple and areola are very often succeeded by the formation of scirrhus cancer in the mammary gland. I have seen about fifteen cases in which this has happened' (p. 87). A statement of this nature coming from so distinguished a surgeon merits every consideration. Opinions vary very much as to the precise value of the communication; but some surgeons are so confident of its truth, and have observed the same connection so often between chronic eczema of the nipple and areola, and carcinoma of the breast, that I have twice had an opportunity of examining breasts which have been completely removed on account of the eczema, although there was no evidence of cancer

¹ Krankhaften Geschwulste, vol. i.

in either. The operation in each case was performed by one of our own surgeons. The first was a private case of Mr. Savory's. The breast, together with an elliptical portion of integument, including the areola, was removed from a lady 57 years of age. The nipple was indistinguishable; the whole of the areola was thickened and crusted over with dry yellow and grey scabs; there was no disease apparent in any part of the breast save about the areola. This eczematous condition had existed for about two years, not yielding to any treatment, causing continual annoyance and distress to the patient, and at length proving so troublesome that the lady readily submitted to the serious operation performed by Mr. Savory. It may be said, Would it not have been sufficient to have removed the diseased areola? Well, Sir James Paget says that he has twice done this, but that it was 'too late,' by which I suppose he means that the breast became cancerous after the removal of the part.

The second case was an Hospital patient, 52 years old, under the care of Mr. Smith, in Lucas Ward. She had been troubled with eczema about the areola for nearly three years, and the condition of the nipple and areola was very similar to that described in the last case. In addition, however, there was an ill-defined mass of induration at the upper and inner part of the breast, apparently not cancerous, but merely of a chronic inflammatory character. This was stated to have been noticed some two or three months. A consultation was held on this patient in the Theatre, and the opinion of those present was unanimously in favour of complete removal of the breast. When the indurated part was cut into after the operation, it bore a very suspicious resemblance to certain forms of cancerous infiltration, and I should not have been surprised had I found the microscopic characters of carcinoma.

Portions of each breast were hardened for some days in chromic acid, were then placed for a week in spirit, and were finally cut with a razor, coloured and mounted in glycerine or Canada balsam. Sections were made in each case through the areola completely down through the breast, so that perpendicular sections of an inch or more in depth were cut. Sections were also made of the indurated portions of the second breast.

The changes discovered were so exactly similar in the two breasts that one description will suffice for both, a slight addition being only necessary for the indurated portions of breast.

The mucous layer of the epidermis of the areola had undergone proliferation; its cells were increased in number, many of them contained two nuclei; whilst at intervals there were fibres or fibre-cells passing upwards between the cells towards the surface.

The corium and subcutaneous tissue were infiltrated with large numbers of small round cells like leucocytes. These are the ordinary changes which have been discovered and described as produced by chronic eczema, therefore there was nothing surprising in the fact that they should occur here.

But in addition to these changes, the galactophorous tubes or milk-ducts were all found widely dilated, whilst their epithelium, instead of being the ordinary form of cylindrical cell, arranged so as to line the ducts evenly and regularly, was now composed of squamous or spheroidal cells, irregularly disposed upon the walls, often jutting out in uneven masses into the interior of the ducts, and even in some cases completely filling them, so that no lumen was visible.

And in the fibrous tissue, lying generally not far distant from the ducts, was the same sort of small-cell infiltration as that occurring in the corium and subcutaneous tissue.

These conditions existed through the whole depth of the sections—for an inch or more, therefore, below the surface of the eczematous skin. That they were continuous from the most superficial to the deepest parts there can be little doubt, for although they could not be traced continuously in any one section (as might be expected from the nature of such sections), they existed in every section, and in all parts of such sections.

The indurated portions of the second breast showed a similar dilatation of the milk-ducts, a similar alteration in their epithelium, and a similar infiltration of small cells in the surrounding tissues.

There was also increase in size of the acini of the mammary gland and proliferation of the glandular epithelium.

There was not any tissue deserving of the name of carcinoma. All the induration appeared to be due to the enlargement of the ducts and acini, the increase in their epithelial elements, and the small-cell infiltration of the surrounding tissues.

Such were the changes in these two breasts which could be traced from the diseased areola, and which may therefore be fairly supposed to have been caused by the irritation of the long-standing eczema, perhaps also by the remedies which had been employed for the cure of the latter. They do not, you see, in either case amount to carcinoma; nor can it be said that carcinoma would inevitably have supervened. I think, however, this would probably have been the case in the indurated part of the second breast; for the changes which were observed in this part were exactly the same in character as those often seen in the outskirts of a growing carcinoma of the mammary gland, exactly the same, therefore, as those changes which may be sup-

posed immediately to precede the development of carcinoma in the gland.

What practical lessons are to be learned from a consideration of the facts just stated? What are we to do in cases of long-standing eczema or psoriasis of the areola—in cases which have lasted, for instance, for two or three years in spite of treatment? Are we to persevere in treatment? Are we to remove the diseased portions of integument, or are we justified in removing what appears to be an entirely healthy breast in order to prevent it becoming cancerous at a later period?

Sir James Paget tells us that clinical observation has led him to believe that in all cases carcinoma forms in breasts in which the areola has been the seat of chronic eczema, and that the time of the supervention of cancer varies from one to two years.

Other surgeons of position—Mr. Savory, for instance—have noticed the same thing, and believe most thoroughly in eczema as a precursor of cancer.

Yet it must be confessed that we very rarely obtain the history of pre-existing eczema of the areola in the cases of carcinoma of the breast which come under our notice in the Hospital. Mr. Arnott of St. Thomas's Hospital, who has been working long and carefully at the subject of cancer, tells me that since the publication of Sir James Paget's paper he has made careful inquiry in every case of cancer of the breast, but has failed to elicit in even a single instance any connection between eczema and cancer.

Still such negative evidence as this cannot of course be compared with the positive evidence to the contrary; it only proves that carcinoma of the breast is by no means frequently due to the irritation of eczema. And this is what might be expected; for whereas carcinoma of the breast is very common, chronic eczema of the areola is, I believe, comparatively a rare disease.

The pathology of the disease, as we have learned it thus far, would decidedly incline us to adopt the radical measure of removal of the whole breast, since nothing short of this would certify us that all the altered textures were removed.

Nevertheless, I think we require a larger experience, both clinically and pathologically, than we have at present, before we shall be justified in removing breasts for eczema about the nipple. The mutilation is so extreme, the operation so considerable, even occasionally hazardous to life, that I think we shall be rather justified in watching for some time to come all such cases with the greatest care, even at the risk of losing a certain number of patients from cancer.

November 4.

Mr. Hastings showed the stomach and intestines of a patient who died from malignant disease of pylorus with secondary deposits.

Mr. Schofield read the notes of two cases of loco-motor ataxia.

Dr. Godson read a paper on 'Subinvolution of the Uterus.'

Subinvolution of the uterus is perhaps the most common of all the conditions which we find among those who apply for relief in the department of the Hospital devoted to the diseases of women.

It is only of late years that this affection has been recognised. It does not even find a place in the 'Nomenclature of Diseases.' Many of the older practitioners, at all events those who do not find time or inclination to keep themselves posted in the medical literature of the day, know not even the meaning of the term, and the consequence is that the question is frequently put, 'What is subinvolution?' You all know that when an ovum has found its way into the uterus, this organ at once sets to work to adapt itself to the requirements of its tenant. It has not only to increase the size of its chamber, but it has to make itself the medium of communicating the nutrition which shall contribute to the support and development of its occupier, and so the little uterus must undergo great and important changes. In fact, its mucous membrane becomes hypertrophied, the tubular glands enlarge, the lymphatics become extremely big and numerous, the arteries become larger and more convoluted, the veins attain a greater capacity and expand into sinuses, and the fibre-cells grow into strong muscular fibres.

All this is, of course, not effected at once; the uterus gradually adapts itself to the increasing wants of the foetus. And so, as soon as it has lost its tenant, robbed of the importance which was hitherto attached to it, it sets to work at once to return to its pristine state, to be ready for any fresh duty which may be imposed upon it. This retrograde change is called 'involution.'

And if anything takes place to interfere with this process, leaving the uterus just in the condition it has then reached, this state of arrest is recognised by the title of 'incomplete involution,' or, as we better know it, 'subinvolution.'

Our next inquiry is, By what means is an organ, measuring fourteen inches in length and weighing twenty-five ounces, reduced so greatly that, in a very few weeks, it measures but three inches in length and weighs only two ounces? In other words, How is the process of involution carried out? Delivery having taken place, the cavity of the uterus is at once diminished in size

by the muscular contraction of its walls, the arteries and veins on their part contract, and about four days subsequently the muscular fibres commence to undergo a process of fatty degeneration, so that in from six to eight weeks' time not a trace of their existence should remain other than fibre-cells. In this process the lymphatics are said to assist greatly. The fatty degeneration commences in the inner layers of the walls, and passes from them to the outer layers. Under the microscope, a few days after delivery, a series of glistening particles are seen deposited in the course of each individual fibre. Now it must be borne in mind that this change occurs at whatever period parturition take place, so that subinvolution may arise after abortion as well as after labour. And inasmuch as the exciting causes are greater after abortion than after delivery at full term, I believe we shall find that the disease under discussion occurs even more frequently after the former than after the latter.

We must now pass on to the consideration of the causes which are likely to check the progress of involution.

First, and foremost, any febrile condition or constitutional disease seriously affecting the general health is likely to produce uterine atony, and so give rise to it.

Secondly, absorption of septic matter, or whatever may cause inflammation of the pelvic viscera, is sure to arrest the process. How frequently we hear of suppression of the lochia! and is not this a manifestation of it? Later on, and this is perhaps the most frequent cause of all, uterine congestion is likely to occur from many circumstances: too early, and too frequent sexual indulgence, assuming the upright posture, and moving about without restraint while the uterine walls are still thickened, and the weight of the organ much increased; and this is naturally liable to give rise to displacements, so frequently found in connection with subinvolution. The rectum, too, is in such intimate relationship to the uterus, that constipation, hæmorrhoids, and the like have a tendency to interfere with the physiological process. What, then, are the symptoms which lead us to suspect this condition?

The patient resumes her ordinary occupations, or endeavours to do so, and finds that this is attended by great discomfort; there is a sense of weight, or bearing down of the womb, sometimes amounting to actual pain: and this is frequently referred to the lower back, to the sacral region; she feels as if she required a support, and suggests the application of a strengthening plaster to the part. In addition to this there is uneasiness in the bladder, she has a frequent desire to empty it, and finds it inconvenient to be so situated that she cannot thus relieve herself.

There is also a feeling of pressure on the lower bowel, and frequently difficulty in evacuating it. Another, and a most prominent symptom, is in connection with menstruation, should it have recurred. As a rule, whereas before pregnancy it continued for five or six days, moderate in amount, with an interval of three weeks or more, it lasts now from seven to nine or ten days, and is very profuse, and, after the lapse perhaps of only a fortnight, it again makes its appearance. Most commonly, too, the patient suffers from a troublesome leucorrhœa, of a watery character; she has to constantly change her linen. She attributes it to weakness, and uses astringent lotions injected into the vagina; but she gets no better, and at length applies for medical advice. And at this stage she comes under our observation. On making an examination, what do we find? and how should this examination be conducted?

First, never omit to investigate the abdomen. On placing the hand on the hypogastrium, and making firm pressure, we perhaps feel a globular body occupying it, which we at once suspect to be the enlarged uterus. There may or may not be tenderness present. With the left hand still on this swelling, the forefinger of the right is passed into the vagina; its walls are probably found lax, and bedewed with moisture; it is found diminished in length, because the weighty uterus is low. Next, the condition of the cervix is investigated, and it is found short, very likely correspondingly increased in diameter, and the mouth of the womb allows the finger to pass readily within it—the patulous os, so well known. If the condition be one of simple subinvolution, the texture is found soft and flabby, just as if parturition had but recently occurred; but if inflammatory action have been set up, we find it, on the other hand, firmer in texture than it should be—*indurated*, as we describe it—and the lips probably everted.

And this brings me to the two forms of subinvolution which you have so frequently heard me speak of in the Out-Patient Room—simple subinvolution and inflammatory subinvolution—two very different conditions, and equally common.

The inflammatory is that which we generally find at a later stage, the simple form having previously existed, and the areolar hyperplasia, as Thomas terms it, being a consequence of inflammatory action subsequently occurring in the uterine tissues.

Having ascertained as far as possible by the impulse conveyed from the hand on the hypogastrium to the finger on the cervix the extent of enlargement, the condition of the cervical tissue is carefully noted, whether or not it feel abraded, or whether fungous growths appear to exist.

The next step is to sweep the finger round the cervix to ascer-

tain the state of the original roof, whether or not any abnormal swelling be present. It should be elastic to the touch, but if there be a displacement of the uterus, which, as I have before stated, is a frequent complication, the absence of the fundus uteri in the hypogastrium having perhaps suggested it, a resistance in the form of a swelling may be discovered in some relation to the cervix, most commonly posteriorly, or anteriorly; or the hypertrophic walls of the uterus may be felt bulging in every direction.

Having carefully ascertained that there is no reason to suspect pregnancy, and no other cause to suggest delay in employing the uterine sound, it may be passed within the os. By means of this instrument we shall be able to ascertain whether the cavity of the womb be increased in length, or in general capacity: instead of measuring $2\frac{1}{2}$ inches in length, it will probably be found to register 3 or 4 inches; and any swelling in the roof of the vagina which may have been previously noted, should it be the body of the uterus, will have disappeared in case of flexion; or if retroversion exist, the concavity of the sound will have entered in an opposite direction to that in which it should, and on being twisted round gently with the finger and thumb, the uterus should move with it, and be capable of being replaced. If, however, the cavity be much enlarged, as is frequently the case, the sound may be turned round within it, producing but little impression upon the uterine walls. Having ascertained so much by the sense of touch and the use of the sound, the speculum should be passed within the vagina, in order to get a view of the cervix. Frequently in its passage it will have collected a quantity of *discharge* within it, and this must be wiped away before we proceed further, and the character of it noted. In all probability the epithelium will be found more or less denuded from the surface around the os, and sometimes fungous-looking growths are observed, extending as far as we can see within the cervical canal; these may be partially obscured by a very tenacious discharge, on wiping away which, with difficulty, by means of a piece of cotton wool held within the speculum forceps, the surface is at once excited to bleed. This is the condition so frequently found associated with simple subinvolution. In the other variety the cervix will probably fill the entire summit of a good-sized speculum, the mucous membrane appears blue and distended, and a highly-tenacious plug is observed blocking up the os uteri. There is generally a certain amount of epithelial abrasion.

We pass on to consider the plan of treatment which should be adopted for the relief of these conditions.

Let us take the simple variety first.

This is generally associated with anæmia and broken-down health, and our efforts must be directed to improve such as much as possible by means of tonics, cod-liver oil, change of air, and good diet. There is one drug specially indicated, and this is iron. Another is the ergot of rye. The uterine fibres are wanting in contraction, to remedy this we look to ergot. It should be administered in doses ranging from twenty to forty minims three times a day. It seems to act more powerfully when combined with strychnia. I generally prescribe a mixture of the ammonio-citrate of iron, with the liquid extract of ergot, and liquor strychnia, the latter in doses of four minims. It is a great thing to avoid constipation, and this preparation of steel seems less likely to give rise to it than any other. The rectum is situated so close to the uterus that a great effort must be made to keep off pressure from it upon the womb, and in all uterine affections the regular action of the bowels is the first thing to be attended to. If, therefore, aperients is necessary, they must be prescribed. The vaginal walls being equally relaxed, astringent lotions or suppositories should be employed. The lotion must be injected by means of a proper vaginal syringe, perhaps that known as Higginson's is the best. The glass syringe so commonly used by the poorer classes is worse than useless, it is dangerous, and should never be employed. The support of a pessary may be needed, but unless well adapted, and borne with comfort, it only increases the mischief. If with all this improvement does not take place, it will be necessary to adopt some more active plan of treatment. I have found great benefit from passing into the uterus, as often as the opportunity occurs—say on alternate days—a strong solution of perchloride of iron, by means of a piece of cotton wool saturated with it attached to a Playfair's probe. This serves two purposes: first, and foremost, it excites the uterus to contract. When inserted, it finds its way into the uterus, perhaps only barely touching the walls of the cervical canal; but on attempting to withdraw it, it will be found to be tightly grasped by them. The other purpose which it serves is that of a styptic; thus applied, it tends to overcome the menorrhagia to which I have referred.

Thomas says that fungous degeneration of the villi is frequently associated with this state, and for such the perchloride of iron would admirably apply.

The inflammatory variety is that, however, which demands the most active treatment. It is with this that displacements are so frequently found to occur. If such exist, they must be treated according to their requirements. If a pessary can be so

applied as to keep the uterus in position, and can be borne without discomfort, it should be employed. But we must also set to work to excite the uterus to take on action, and allow absorption of its hypertrophic walls to ensue.

As I have stated, the cervix looks discoloured and distended, and this condition at once suggests relief by local depletion. Such treatment should not only relieve congestion of the vessels, but have the effect of setting up absorption of the organised products of inflammation. The late Sir James Simpson was the first to describe this disease. He did so, in 1852, in a paper entitled 'Morbid Deficiency and Morbid Excess in the Involution of the Uterus after Delivery.' He strongly recommends the application of eight or twelve leeches to the vaginal portion of the uterus, or to the anus or perineum. Of late years a much more convenient method is employed, that of puncturing the cervix by means of a lancet. Thus the amount of blood allowed to escape is under the control of the operator, and the process is far less fatiguing to the patient. In this way, from time to time, one or two ounces of blood may be extracted from the cervix. It will be well, too, in some cases, to apply counter-irritation to the lower abdomen, over the enlarged uterus; the hypogastrium may be painted once or twice a day with a strong solution of iodine till the skin be vesicated, or a series of small blisters may be employed. I am accustomed to deplete the cervix two or three times during the month, and, as often as opportunity offers, I pass into the uterus a strong solution of iodine soaked in cotton wool, by means of a Playfair's probe. I always follow this up by applying against the cervix a good-sized tampon of cotton wool saturated with glycerine. Glycerine is most useful as a depletory agent. Its great affinity for water is well known; it seems to extract from the congested vessels a large quantity of serum. Patients complain that their linen is kept soaked for a day or two after the application, the characteristic of the discharge being that it will not dry. I have proved lately in several cases in Martha Ward the rapidity with which the abraded cervix heals after the frequent application of these glycerine pledgets.

Sir James Y. Simpson recommends suppositories of the iodide of lead, also of bromide of potassium, and mercury. I have never seen any good effects from their employment.

Sponge-tents, inserted at short intervals, have been used, and Thomas says that they of themselves exert an alterative influence upon nutrition. I do not recommend their employment for such a purpose; they are always attended with risk, and I think their efficacy extremely doubtful.

I must not neglect to mention the heroic plan of treatment proposed and extensively practised by Dr. Atthill of Dublin. He advises one of two methods; either passing, by means of Simpson's porte-caustique, ten grains of solid nitrate of silver into the uterus, and there leaving it, or swabbing out the uterine cavity with fuming nitric acid. The way in which these plans are carried out is fully described in Dr. Atthill's valuable little work on diseases of women. It appears that after the application of the acid, it takes five or six weeks for the slough caused by it to separate.

Dr. Atthill says with regard to the treatment by nitrate of silver, that he has practised it freely for many years, and believes it to be both simple and safe. He admits that it may give rise to pelvic cellulitis, and that he should not be at any time surprised at its occurrence; but says that the fear of it would never deter him from carrying out the treatment, for he considers an attack of cellulitis of much less importance than the continuance of profuse menorrhagia. It will be well for the practitioner to weigh thoroughly these points in his mind before deciding upon adopting this method. The same risk applies to the application of fuming nitric acid to the cavity of the uterus; I have seen three cases of pelvic cellulitis following it, and one of the patients nearly died. I think, therefore, it requires an amount of courage for a general practitioner to undertake these heroic methods in his private practice. Death resulting from his treatment of a chronic uterine disorder might seriously damage his local reputation.

I do not wish to declaim against this plan of treatment, for I have seen admirable results from it. After one thorough application, the continued losses of blood have ceased, and menstruation has occurred with regularity, and without excess.

I have reserved till the last the medicinal treatment which appears most useful in these cases. The iodide and bromide of potassium given together seem to be of great service; the latter drug, however, is the one chiefly to be depended upon. It was first introduced into medicine by the late Dr. R. Williams for the purpose of causing absorption of the products of inflammation. Sir James Y. Simpson has written that it may be depended upon as an active stimulant to absorption, besides possessing, beyond all other remedies, the property of acting as a special sedative on the reproductive organs.

I frequently combine this drug with citrate of iron, hoping to obtain also the benefit to be derived from the tonic.

Rest in the recumbent posture and freedom from sexual intercourse are important points in the treatment of subinvolu-

tion. Such must be enforced as much as possible. I believe the success with which the cases are treated in Hospital is in a great measure due to the thorough way in which these points are carried out.

November 11.

Dr. V. Harris showed three pathological specimens.

1. Liver, spleen, and kidneys. The liver was red and slightly granular. The right lobe contained a large cheesy mass. The spleen, kidney, and intestines were amyloid.

2. A very dilated heart, containing many large ante-mortem clots in the right auricle and left ventricle. The aortic valves were much diseased.

3. An aneurysm of the descending part of the arch of the aorta, eroding the upper dorsal vertebræ, pressing upon the left bronchus, left vagus, and the œsophagus.

Mr. Lyons read a paper on 'Diseases of the Jaw connected with the Teeth.'

In this paper I have to treat of those affections of the jaws of which the teeth are the immediate cause, and firstly of alveolar abscess. It is the result of an acute inflammation of the peri-odontal membrane, which may arise from a blow on the jaw or teeth, from cold or other source of irritation, but it is most commonly the sequence of an acute inflammation of the pulp of a tooth. A patient complains of a dull heavy pain arising from a certain tooth which is tender on pressure, after a time the pain becomes more acute and throbbing, pus is formed, and finds an exit at the labial or buccal surface of the jaw, or at any point internal to the dental arch.

The fang of the tooth being contained in a bony cavity, inflammation arising either in the pulp cavity or peri-odontal membrane, inflammatory exudation takes place around the fang, within the cavity, going on to the formation of pus; the walls of the cavity become absorbed, and in one place or another perforated; the pus then escapes either through an opening in the cheek, or below the lower jaw, but most commonly into the mouth.

Alveolar abscess is a trifling matter as a rule, but it has been known to end fatally, by causing extensive disease of surrounding structures, as in the case related by Mr. Salter. The common error with regard to alveolar abscess is that it may be mistaken for diseased bone in those cases in which the orifice occurs on the surface of the face; the tooth, so far as the vitality of the pulp is concerned, is dead, and is the source of the irrita-

tion which keeps up the discharge, being therefore analogous to the sequestrum in necrosis.

Attention to the following points will lead to an accurate diagnosis.

In alveolar abscess there is less general swelling, except at first and in acute cases; there is an absence of that diffused induration of surrounding tissues which is present in necrosis; the canal leading to the abscess is single, usually short and direct; the characteristic fœtor of the discharge from dead bone is also wanting. The locality, in a doubtful case, being in the neighbourhood of the jaw, is in favour of the idea of a tooth being the cause, and an examination of the state of the teeth seldom fails to settle the question.

With regard to treatment there is a point of importance to be noticed. Poultices and fomentations should not be applied to the face, though at the commencement of the peri-odontitis and prior to the formation of the abscess they are grateful to the patient; but when pus has formed, they may have the effect of causing the abscess to point externally. For example, an abscess occurring at the base of an upper bicuspid tooth, the swelling is on the buccal surface; on a poultice being applied externally, pus would travel above the fold of mucous membrane, connecting the superior maxilla with the cheek, and appear among the buccal muscles, ultimately passing through the skin, thereby causing those scars so often seen on the faces of patients who have undergone this treatment. I would therefore recommend that all poultices be applied within the mouth, so that the abscess may point therein. The immediate extraction of the offending tooth or teeth effects the most rapid cure, as an exit is made for the pus and the source of irritation removed simultaneously; but it is not always desirable to extract the teeth. I have sometimes done so, and swabbed the sockets well with antiseptics, such as a strong solution of carbolic acid or of chloride of zinc (40 grains to 1 ounce), and then returned the teeth to their sockets with a fair amount of success.

Of the many cysts that occur in connection with the jaws, I have simply to deal with the so-called dentigerous cysts.

A cyst is a cavity containing fluid or pultaceous material which is separated from surrounding structures by a more or less distinct capsule; it may be a new formation or a pre-existing structure which has become distended by its own secretion, or by extravasation into it. Generally a dentigerous cyst forms in a pre-existing structure, the commonest exciting cause being an impacted tooth; for we find that when one of these cysts is formed, there is a tooth short of the normal number, gene-

rally it is the canine or first or second bicuspid, though it may be any other, which tooth will be found in the cyst to which it has given rise.

Why a tooth embedded in the jaw should cause these serous collections is not clearly understood. I have seen a number of skulls wherein teeth were lying horizontally across the jaw, encased in a bony chamber, and which simply through their position were unable to assume their places in the dental arch. I also know patients in whom the same thing is going on, without the slightest symptoms of the formation of a cyst.

The symptoms of a dentigerous cyst are an enlargement of the jawbone in that part where the cyst is seated. An examination of this enlargement shows it to be an expansion of the bone itself; and on pressure the characteristic eggshell or parchment like crackling is elicited, the absence of one or more teeth that have not been extracted confirming the diagnosis.

In my practice I have seen but three, and they were all in the upper jaw. In one the canine was missing, and in the others the first bicuspid, which, when they were taken from their respective cysts, were found imperfectly developed, the fangs being wanting.

We must bear in mind that even though all the permanent teeth be present, there may yet be a temporary one impacted.

Alveolar abscess not uncommonly becomes converted into a cyst, even after the extraction of the offending tooth.

I had a case of a little girl who had suffered repeatedly for a long time from a recurrent alveolar abscess arising from the fang of a lower bicuspid, in connection with which was a cyst extending two inches on the left side of the jaw.

The term epulis is applied to a variety of growths springing from the alveolar process and gums. I confine the term to a growth that is fibrous in structure springing from the alveolar process, usually from the little slip between two teeth, growing slowly, involving the bone, periosteum, and more or less of the peri-odontal membrane. They are more common on the buccal than on the lingual surface of the jaw.

An epulis tumour seems to have a certain relation to the teeth in whose neighbourhood it forms.

It always makes its appearance where there are teeth or fangs. It usually invades one in a very marked degree more than any other near to which it may be situated, by dislocating and pushing it out of place. It has nothing to do with caries of the teeth, and the removal of a certain tooth together with the excision of the tumour is almost always accompanied by the immediate and complete cure of the disease. Occasionally these tumours appear

where teeth have been removed, and the gums seem edentulous ; it will, however, generally be found in these instances that a fang of one of the teeth has been left behind, and is associated with the irritation that has caused the growth.

Complete extirpation and the extraction of the neighbouring teeth is the rule for the treatment of epulis.

The following case, however, shows that such a proceeding may be unnecessarily severe. A surgeon excised a large portion of the outer plate of the socket of a central incisor tooth, on which an epulis had grown, and though two years since, the tumour has not yet returned. But we ought to supplement the excision by painting the gums once a week for two months with undiluted carbolic acid.

With regard to that terrible form of necrosis arising from phosphorus poisoning, it is a recognised fact that it cannot occur in patients who have perfectly sound teeth, but only in those whose teeth are carious.

The subject of nervous affections arising from diseased teeth is important and interesting—important because of the remote parts that are sometimes affected by that which is generally an obscure cause, and interesting through the variety of maladies that not infrequently ensue. Mr. Salter divides these affections into those which are reflex, secondary, and remote ; and those which are direct, immediate, and from contiguity. In the former category would rank epilepsy, neuralgia, and paralysis ; in the latter, local pain, facial palsy, and some forms of amaurosis. The situation of the teeth, their abundant supply of nerves, and the great and diffused swelling which their diseases produce in contiguous structures, inevitably evolve much nervous disturbance and complication. The superior molars are but little removed from the tonsils and Eustachian tube, and their fangs are so close to the orbit and its all-important contents, that it will not be difficult to account for the nervous complications which are directly entailed by the spread of inflammation from the periosteum of diseased teeth. Of course the most common reflex nervous disturbance to which dental irritation gives rise is neuralgic pains in the head, and this especially where the upper teeth are implicated. In the supra and infra orbital nerves, the globe of the eye, the temple—in these regions dental neuralgia is very common. The several branches of the fifth pair appear to be the most susceptible of reflex action, caused by the dental irritation of one of them ; and next, the branches of the cervical and brachial plexuses: hence pains of the neck and shoulders, bend of the elbow, and with them occasionally loss of motor powers, are common. Reflex nervous irritation dependent on nervous

disease in teeth is uncertain in its manifestation. As regards the teeth which excite nervous irritability, nearly all their diseases appear capable of causing this condition—caries with or without exposure of the pulp, exostosis, nodular development of dentine in the pulp cavity, peri-odontitis, impaction of permanent teeth in the maxillary bone, and crowding of teeth from insufficient space.

By way of illustration, I relate a few cases taken from published accounts and my own practice.

In 1874 a woman applied to Mr. Salter for relief on account of severe neuralgic pains connected with one of the central incisors of the upper jaw. At times the pain was simply confined to the region of the tooth, but on sudden pressure or change of temperature it flew all over the face. The tooth was to all external appearances perfectly healthy. On its extraction Mr. Salter made a vertical section, and found an oval pearl-like excrescence of dentine growing from the side of the pulp cavity, so as to encroach much upon it, and occupying for a short space more than half its diameter, which by pressing on the pulp gave rise to the neuralgia.

A patient of Mr. Bell's had suffered from neuralgia for many years. The attacks appeared associated with certain teeth from which the pain seemed to emanate. At first the neuralgia was confined to the branches of the fifth pair, but as it increased in intensity, it spread to the arms, legs, and indeed to the whole body. The teeth appeared quite sound, but always gave pain on being slightly struck. Mr. Bell extracted those teeth which appeared most involved, and this was followed by complete cessation of pain, but only of temporary duration. The fangs of the teeth were each discovered to be encrusted with small nodules of exostosis. It was found necessary from time to time to extract some teeth, and this was done until all were gone, and then only was the patient entirely free from neuralgia. The narrator of this case seems to think that nodular exostosis is rarely the cause of neuralgia. I believe it is not uncommon. In 1869 a gentleman came to me and complained of severe neuralgia on the right side of his face. On examining the jaw and percussing the teeth, I found the right upper bicuspid sensitive, and after trying various constitutional remedies for the relief of the pain without success, I extracted the tooth, and on examination I found a nodule of exostosis at the extremity of the fang, which I pared, and returned the tooth to its socket, and since then this patient has never been the subject of neuralgia.

A nodule of exostosis is hypertrophy of the cementum, and may range from the size of a pin's head to a small pea.

Chronic trismus is an affection which arises from impaction of wisdom teeth. There is hardly a month passes without a case being seen in the Dental Department of this Hospital. It occurs through the difficult eruption of the wisdom teeth owing to want of space. It is not an uncommon operation to extract the second lower molar to make space, and the trismus invariably ceases.

Mr. Hancock published a case where wry neck had been caused by carious teeth. A young woman was brought to Charing Cross Hospital with her head drawn down nearly to the left shoulder, medical treatment having been tried and failed. No disease could be detected in the spine, but on looking into the mouth, a stump and a partially-decayed tooth were seen in the lower jaw ; the teeth were extracted, and she was well in a few days.

Dr. Ramskill records a case of epilepsy arising from a carious tooth. It was published in the 'Medical Times and Gazette' in 1862. A boy, aged 13, had frequent attacks of epilepsy for eighteen months. Latterly it was noticed that the boy for some days would rub his left cheek and complain of face-ache, and then a fit would follow. Upon examination of the mouth, a carious molar was discovered on that side, and extracted. During the following four months no recurrence of fits took place.

Mr. Hancock relates a case of amaurosis arising from crowded teeth. It occurred in a little boy aged 11 years. A month before he saw him, the boy awoke one morning and found himself perfectly blind. Before that he had nothing the matter with his eyes, and when he went to bed on the preceding night he could see distinctly. Deriving no benefit from the treatment of the country practitioner, he was sent to the Hospital. His pupils were dilated, fixed, and uninfluenced by light, which he could not distinguish from darkness. The suddenness of the attack, combined with other symptoms, led Mr. Hancock to conclude that the mischief was functional rather than structural, and he examined the teeth, and found that they were much crowded and wedged together. Accordingly four teeth were extracted. The same evening the boy could distinguish light from darkness, and the following morning could distinguish objects ; and within a fortnight he was dismissed entirely cured.

I had a patient who, according to his own account, suffered from neuralgia for three years. During the last seven months the pain had been so excruciating that existence was almost unbearable. On examining the mouth, there was seen to be an opening in the upper jaw, opposite to the spot where the left central incisor formerly was placed, and out of this pus was oozing. On passing a pair of ordinary dressing forceps, something was

felt, and withdrawing them, the dead fangs of a bicuspid tooth fell out. Within two days after the extraction, the neuralgia had entirely disappeared. This case had been mistaken for necrosis, and so treated for two years.

I could cite cases of paralysis, wry neck, deafness, ulceration of the neck, and facial palsy, arising from affections of the teeth, but believe the above cases will be sufficient to illustrate the value of the study of the nervous diseases of the teeth, and thus may tend to throw some light on the sometimes obscure origin of diseases about the region of the neck and face.

November 18.

Dr. Moore showed two specimens.

1. An aorta showing an abnormal origin of one of the coronary arteries.

2. An amyloid kidney with a gummy tumour in it.

Dr. Moore read a paper on 'Bronchitis.'

The term bronchitis is scarcely met with in the authors of the last century. Sydenham has described one variety under the title of peripneumonia notha; Laennec describes all varieties under the heading of pulmonary catarrh. While adhering to the term bronchitis, I wish to lay down as a first principle that it is a general, a constitutional affection. It has many varieties, and in most of these a cough bringing up sputa is the prominent symptom.

Cough is only absent where the patient is very young. The respiratory muscles are not sufficiently under their control to enable them to eject the morbid secretion of the mucous membrane, and they are apt to die, not of the disease as a whole, but of the mechanical results of this particular part of it.

Most of the cases of bronchitis I have seen may be grouped under one of the following heads: (1) Peripneumonia notha; (2) Cough of rickets; (3) Acute bronchitis of young people; (4) Winter cough; (5) Bronchitis with hæmorrhage; (6) Bronchitis with fibrinous sputa.

Bronchitis with fibrinous sputa is a rare disorder. The cough is always chronic, and perhaps for this reason the characteristic sputum is rarely seen in our wards. They sometimes occur as small fragments, but more often as branched masses in shape like a leafless tree. Their shape led Tulpius to describe them as coughed-up pieces of branches of the pulmonary vessels. As he is probably the first author who mentions this sputum, it is worth while to give one of Tulpius' cases.

'Whole veins cast up from the lung.—A captain of Amsterdam,

who had long lived at sea, when well on in life contracted after repeated colds a cough which was worse on land than at sea. This cough wearied his lungs for more than two years. He did not cough up blood, but two large branches of veins, which, when spread out, covered as great a length as an extended hand. They were altogether free from any of the parenchyma of the lung, and showed distinctly the features of their ends, just as if they had been dissected out by a careful anatomist—a clear proof this that the whole lung was destroyed. No wonder, therefore, that he died shortly after. His physicians were much astonished that there could be so great a breaking down of the parenchyma without any previous appearance of pus.

This variety is rare in our wards, and is not common among the out-patients. In two cases I have seen the cough had lasted for more than a year, and in both there were hard fits of violent coughing. The patients presented no symptoms characteristic of this form of disease.

Bronchitis with hæmorrhage is often met with among the butchers of Smithfield. When young, the butcher suffers from the most violent forms of pneumonia, of pleurisy, and of bronchitis; when old, or rather when in middle life, from chronic Bright's disease and its accidents, and from the lowest and most hopeless forms of pneumonia and bronchitis. The bronchitis which this kind of man has when young is seldom seen in other people. Traces of blood occur now and then in the sputa of chronic bronchitis, but this is not the hæmoptysis to which I allude. It resembles that which precedes phthisis. It is a copious sudden discharge of blood. It has been preceded by a violent cough, and this cough, usually somewhat diminished in severity, remains after the hæmoptysis. The treatment consists in keeping the patient quiet in bed, in a low diet, and in free purging. In a few cases where nature has not relieved the circulation sufficiently, it hastens recovery to bleed from the arm. The most important point, however, about this disease is its differential diagnosis. Such hæmoptysis in a young man is in most cases justly regarded as a certain indication of the commencement of phthisis. The full-blooded condition of the butcher, the absence of wasting, of night-sweats, of any signs of debility, of the physical signs,—these are weighty arguments against phthisis. I believe that the shape of the chest has hardly been sufficiently insisted on as a point to be observed. If a patient has a chest the outline of which shows the normal proportion of antero-posterior and transverse diameter—that is, the proportion $1 : 1\frac{1}{2}$ —and if the outline has no marked irregularity at any point, it is likely that the true diagnosis of his case is bronchitis with

hæmorrhage. On the other hand, if the patient's chest have the proportion 1 to $1\frac{1}{2}$, if his scapulæ stand out from it like wings, if it is unduly flattened or unduly rounded here or there, I believe that his disease is most likely to be phthisis.

The acute bronchitis of young adults is characterised by a violent cough, but if there be any trace of blood in the sputa, it can usually be found to come from the fauces. This is a febrile disorder, of which the first local manifestation is usually laryngitis, causing hoarseness. The laryngeal affection passes off, but the cough continues. The sputa are often scanty, and consist of tough viscid mucus. Young people with mitral disease, however slight, are especially liable to this form of bronchitis. If there be considerable mitral obstruction, the disease assumes a grave aspect, and this is the only case in which this bronchitis gives rise to dyspnœa. The sputa are then very abundant and watery.

The treatment of this bronchitis, when uncomplicated with heart disease, should be expectorant. It is in this kind that squill, senega, and serpentary are most useful. A purgative should always be given. The diet of the patient need not be restricted, but all stimulant, except perhaps claret-and-water, should be cut off for the time. The rule of not going out after twilight must be strictly enforced.

A winter cough begins to affect many people about the age of forty. This is the form of bronchitis which leads to emphysema. The cough comes on in violent fits, which are sometimes to a slight degree under the control of the patient. If children in the house happen to have whooping-cough, the adult's cough sometimes takes the whooping character; but in the adult there is rarely vomiting. The coughing is often worst at night. This is no doubt sometimes due to the fact that many bedrooms grow intensely cold as the night goes on, which very often results in a sudden awaking about 2 A.M. with cold ears and a suffocating sensation in the chest. The patient has one fit of coughing, and then another, and only dozes off to sleep when the morning sun has given a little warmth to his room.

Blood is now and then seen in the sputa. It comes from the bronchi or the larynx. Small ecchymoses are sometimes to be seen there post-mortem.

Every subject of winter cough must have his special regulations, but the main points in all are to stay in doors in bad weather, to wear non-conducting materials, to eat and drink moderately.

Children in arms are often brought to you with a severe cough. Examine their ribs and their wrists, and you will often

find signs of rickets. On questioning the mother, you find the cause of the disease over-suckling or too early feeding. There are too few teeth, and the fontanelles are too widely open. The child has perhaps had repeated coughs. When you have satisfied yourself that the case is one of rickets, give no cough mixture. Regulate the diet and treat the rickets. Give cod-liver oil, or where this is unsuitable, bark or iron, and the cough rapidly gets well. Infants not rickety may have coughs, and these are usually due to chills received in washing.

When an infant, rickety or otherwise, is in a state of collapse owing to impending suffocation from sputa which are not coughed up, it must at once be made to vomit, and mechanical means are often the quickest. The vomiting will relieve it a little. It may then be given half a grain of Am. Carb. dissolved in water, the dose to be repeated in a few minutes. Sometimes this will not be at hand, then brandy may be given, but three drops should be the limit.

Peripneumonia notha is a subacute form of bronchitis. It has two symptoms besides the cough—frontal headache and constipation. Sydenham has described it with his usual truth, and whoever follows his directions as to the treatment will succeed. He gave low diet and purgatives, and cut off all stimulant. I think I have observed that middle-aged people whose constipation is due to a partial hernia often begin with dyspeptic symptoms, and go on to a cough, with slight rise of temperature and headache—in short, to peripneumonia notha. Pain in the side is common in this as in all kinds of bronchitis. It is probably external and muscular, and not pleuritic. I have seen a case in which it was so severe as to draw off attention from the pulmonary symptoms, and to lead the physician to talk of diseases of the liver.

In these rough notes on bronchitis I have raised objections to the use of stimulants. I wish this to apply to all the forms I have mentioned, for I am of opinion that the avoidance of alcoholic stimulant is the main point which distinguishes the right way from the wrong in the treatment of bronchitis. A short time ago I saw a lady apparently dying of bronchitis. Her face was flushed and approaching duskiness, her skin hot, her pulse 124; she was about 60 years of age, and of a feeble constitution. On inquiry I found that she had port, champagne, and brandy every day in large quantities. These stimulants had fanned a slight flame into a fire. They were reduced in quantity and her fever declined, and she recovered her usual health.

Bronchitis is so well-worn a subject that some apology is per-

haps needed for a paper on it. The author of the 'Lily of Medicine' says, 'Quia igitur memoria hominis labilis est, quod communiter de practica dicitur, repetere humilibus non erubescio quoniam secundum Gal. vii. de ingenio, nullus potest Deo melius approximare quam studendo in veritate et pro veritate.'

November 25.

Mr. Bradshaw read the notes of a case of varicose veins in the leg treated by means of eschars produced by Vienna paste. The case terminated favourably.

Mr. Macready read the notes of two cases of 'nervous disorders following concussion by a railway accident.'

In the first case the patient, who was seen soon after the accident, had continuous trembling, which lasted for several days, without any other very definite symptoms.

The second case was that of a man, aged 22 years, who had received a blow across his loins, and was admitted in a state of concussion. The patient also had general hyperæsthesia, and in addition several fits daily of an hysterical nature, which could be produced by touching his head or his spinal column.

Mr. Hames read the notes of a case of death following an injury to the abdomen in a boy aged 8 years. The patient had his abdomen struck and compressed by the handle of a barrow: this was followed by constant sickness and the general symptoms of shock. The temperature and pulse rose gradually in a definite ratio (the temperature from 97° to 103°) within twenty-four hours. The patient then suddenly died. The only change found post mortem was a slight sub-peritoneal ecchymosis near the head of the pancreas.

December 2.

Dr. Herbert Taylor read the notes of a case of dislocation of the wrist.

Boy, aged 14 years.—On November 15, 1875, in the afternoon, while practising on a vertical pole in a gymnasium, he suddenly felt faint, loosed his hold, and fell to the ground; when he was picked up, it was found that he had sustained some injury to his left wrist. I saw him within ten minutes of the accident.

The appearance of the left forearm and hand may be described as follows:—

At the level of what appeared to be the lower end of the radius and ulna on the dorsal aspect was a rounded swelling with which the carpus, metacarpus, and phalanges were directly continuous; this swelling extended across from radius to ulna, and did not

appear to be confined to either bone. On the palmar aspect, on a plane lower than that of the dorsal prominence, was another elevation directly continuous above with the shafts of the radius and ulna; and owing to the occurrence being so recent, there was but slight effusion, and I have no doubt that the inferior termination of the palmar swelling was formed by the articular surface of the radius and the triangular fibro-cartilage on the ulna. There was but very slight pain on manipulating these surfaces, and the sensation to the fingers through the skin was the same as that associated with the touch of the upper articulating surfaces of the human wrist-joint.

Thus the carpus appeared to be on a higher plane than the radius and ulna.

The hand was not deflected towards the radius nor ulna, but lay in one axis with those bones. There was no lesion of the skin. The length from the point of the olecranon to the tip of the styloid process of the ulna was the same in each arm, namely, $8\frac{2}{3}$ inches. The length from the point of the olecranon to the proximal end of proximal phalanx of little finger was 11 inches in the right arm, $10\frac{1}{2}$ in the left, so that there was shortening to the extent of $\frac{1}{2}$ inch between the styloid process and the phalanx. There was but very slight impairment of pronation and supination; no crepitus obtained in ulna or radius either before or after reduction. Very little pain in manipulation either at the seat of injury or higher up the forearm.

The continuity of the styloid process of the ulna, and the articular surface of the radius, with their respective bones, seemed complete.

Treatment consisted in reduction by bending the carpus backwards and then extending. When reduced, the lengths of the two arms were identical, and the deformity did not recur on removing the extending force.

One long external splint; shorter palmar pads over the positions of swelling; bandage and sling.

November 19.—Removed and readjusted splints, allowing the fingers free movement; very little pain or swelling.

November 26.—Readjusted splints; free movement of fingers and thumb.

December 2.

Taking into consideration all the above points, I came to the conclusion that this was not a case of fracture of the forearm, but of dislocation of the carpus backwards and upwards on the bones of the forearm.

This injury has always been considered of very rare occurrence, and some authorities, among them Dupuytren, maintain that such an accident as simple dislocation of the wrist without

fracture of one of the bones cannot occur, and that all those cases that have been thus designated, have been found on closer examination or on dissection to be complicated with fracture, or even to be fracture without dislocation.

Many surgeons, however, in their written works, though admitting the rarity, allow the possibility of the accident.

Its rarity, as we may suppose, is not due to the freedom of that part of the human frame from external violence; for it is one of those most exposed to danger, the lower end of the radius being, next to the clavicle, the most frequent seat of fracture, and, as we know, injuries to the forearm and hand form a large item of the entries in the surgery-book.

In the month of April 1875 there were entered—

30 sprained wrists.

15 Colles' fracture.

2 fractures of both bones at wrist.

1 greenstick fracture of ulna.

The cause of the rarity of dislocation is due to the comparative weakness of the structure of the radius at the point where the shaft expands into the cancellous tissue of the epiphysis; hence in falls on the hand, if anything more violent than to produce a sprain, the force is transmitted from the carpus through the wrist to this weak spot, and the more frequent result, as we have seen, is a Colles' fracture. The injury my patient sustained was rupture of the posterior ligament, and probably anterior fibres of the internal and external lateral ligaments, with stretching of all the parts: whilst in sprains there is uniform swelling not coming on at once or in so short time; absence of any characteristic deformity; no shortening.

The injuries with which this form of dislocation may be confounded are—

1. Fracture of both bones above the wrist.

2. Separation of the epiphyses.

3. Colles' fracture.

4. Sprains.

1. From the first it is to be distinguished by—

(1.) The continuity of the articular surfaces and styloid processes with the shafts of their respective bones.

(2.) A comparison of the length of the bones of the forearm on the sound and on the injured limb.

(3.) The absence of crepitus on moving together the supposed fractured ends.

(4.) The less pain in dislocation.

(5.) Comparison of length of carpus on sound hand with that of the displaced carpus. In dislocation, they will be identical; in

fracture, on the injured side the length will be greater by about $\frac{1}{2}$ to 1 inch or more.

2. The above reasons will also distinguish it from separation of the epiphyses with displacement backwards.

3. In this injury (Colles')—

(1.) The dorsal prominence is more confined to the radial side of the forearm, and immediately beneath this on the palmar aspect is a hollow indicating the absence of the lower end of the radius, while the palmar prominence formed by the pronation of the upper fragment of the radius is higher up the arm than in dislocation.

(2.) The hand lies at an angle with the forearm, in consequence of its following the displacement of the lower piece of the radius upwards and outwards.

(3.) Again, if reduction be effected, whether easily or with difficulty, crepitus will be obtained, and on remission of the extending force the deformity will recur; whereas in recent dislocation the reduction is easy, there is no crepitus, and the deformity does not recur.

Dr. Wharry read a paper on 'The Part taken by the Alimentary Canal in the Conservation of Health.'

I propose to call the attention of the Society to the consideration of one of the functions of the alimentary canal—viz., to its 'compensative action in maintaining and restoring the balance of health.' As illustrating the performance of this function, I may cite the following case:—

'A patient ate mushrooms for supper, headache soon supervened, with vomiting, a restless night, a sharp attack of diarrhoea, and in forty-eight hours the symptoms had subsided.'

Here we have the stomach ejecting its injurious contents; the intestines, by increased vigour of the peristaltic action, hastening these injurious contents onward; the intestinal flux washing the poison out from any nooks in which it may have lodged. But though we may reasonably infer it, we cannot prove that the poisonous matter absorbed into the blood has been eliminated by the intestines.

If we turn to the experiment of Feltz and Ritter, we shall see that poisonous matter in the blood may be and is eliminated by both the stomach and intestines.

M.M. Feltz and Ritter injected fresh ox-bile into the veins of dogs. When large quantities were injected, bilious diarrhoea set in, with vomiting first of food, then of bile and blood; other serious symptoms were developed, but the animal usually recovered.

The vomiting that is so troublesome after the inhalation of chloroform is a familiar example of elimination from the blood and ejection by the stomach of an injurious substance.

After parturition, when the mother loses a large quantity of blood, after operations during which there has been much hæmorrhage, and during convalescence from acute disease, the bowels are almost always confined for several days. Here we have the alimentary canal husbanding the resources of the system, allowing nature to make up the lost ground under the most advantageous circumstances. This is another example of compensative action on the part of the intestines.

In cirrhosis of the liver, and under all circumstances giving rise to obstruction of the portal circulation, we have the compensative function of the intestines again at work. The engorged veins and the distended spleen are relieved by fluxes and hæmorrhages into the bowel; and in some cases an exact relation exists between the hæmorrhages and the enlargement of the spleen, so that the physician can prognosticate a hæmorrhage from the size of that organ. When, under these conditions, a hæmorrhage takes place into the stomach, the blood is ejected by vomiting.

There is one more variety in the form of this compensative action exhibited in those cases where women have menstruated vicariously by means of hæmorrhages from the stomach. Such cases I regard as typically compensative in maintaining the balance of health. Here we have a natural function suspended, which in the usual course of events would seriously impair the health, but instead of this we have developed an anomalous periodical hæmorrhage from the stomach, and the healthy standard is maintained.

To return to the consideration of vomiting as a conservative effort of nature.

In our text-books on toxicology, we have hardly a poison there tabulated which does not number 'vomiting' amongst its symptoms, and the more speedy the vomiting the greater is the chance of the patient recovering from the effects of the poison.

These poisons not only produce vomiting when introduced into the stomach, but when introduced directly into the blood, even if we divide the pneumogastric nerves. D'Ornellas, who has been experimenting in this direction recently, gives us the following results.

Emetine, when introduced into the blood of a dog, produces vomiting, and the rejected matters produce vomiting in pigeons. After division of the pneumogastric nerves these results likewise occur, the vomiting being retarded and less efficient. If the dog

is killed directly vomiting sets in, emetine may be detected in the stomach. These experiments prove beyond a doubt that the stomach is capable of eliminating poisons from the blood, and they also demonstrate the truth of Claude Bernard's statement, that the sympathetic nerve supplying the stomach is capable of taking on the action of the pneumogastric when that is deficient, thus furnishing an example of another kind of compensative action.

The vomiting early in smallpox, measles, and scarlatina is an ineffectual attempt to eliminate the poison.

The natural assimilative function of the alimentary canal is imperfectly adjusted to the wants of the organism. Some believe that it habitually assimilates more than is necessary for the sustenance of the body, and that the surplus is got rid of by the kidneys as urea. Others consider that in order to secure the assimilation of sufficient nutritive material, a large excess of food must be taken. Now, with regard to the supposed excretion of surplus food as urea, the following facts, which I have myself observed, incline me to look upon it as unproved, and that urea is derived solely from the retrograde metamorphosis of the tissues: 1. A man fed solely on a reduced quantity of non-nitrogenous food excretes almost, if not quite, as much urea as when upon ordinary diet. 2. A man fed upon a diet of ten ounces of solids and ten ounces of fluid, excretes a very large quantity of urea in proportion to the nitrogen in his food.

Assuming the digestive process to be imperfect, this imperfection is a result of the existence of its power of elimination, and the more the eliminative power is exercised, the less perfect is the performance of digestion. This proposition is borne out by the fact, which I have noticed in the recent epidemic of typhoid fever, that those patients that have suffered most from diarrhœa have suffered most from imperfect digestion.

The diarrhœa of typhoid fever is nature's attempt to throw off the poison by the alimentary canal. It is tolerably certain that typhoid fever is caused by the passage into the intestines of the miasm suspended in fluid; it is also probable that the glandular affection of the intestines and mesentery is the direct effect of the action of the germs absorbed by them. This does not account for the typhoid stools,—the fluid of the stool is the flux that occurs when the intestine is excreting a poison; the solid particles in the stool are the food which the bowels (fully occupied in eliminating) refuse to digest.

In the surgical wards we sometimes see granulating wounds, which, after progressing favourably for some time, will one day have changed their aspect, having an 'angry' look about them;

and upon inquiry the patient is found to have some diarrhœa. This condition arises from slight septic absorption, and the system throws off the poison by means of the diarrhœa.

The importance of recognising the compensative function of the alimentary canal is manifest when we come to deal with disease clinically. If a poison is taken into the stomach and vomiting ensues, it is our obvious duty to ensure the efficiency of the vomiting. If an irritating substance gets into the alimentary canal, fluid is poured out, and the peristaltic action becomes vigorous; we must give medicine calculated to increase both the flux and muscular action. If there is constipation after any severe hæmorrhage, we must leave it to itself, and avoid purgatives. In acute Bright's disease the kidneys do not perform their work properly, and there is an accumulation of nitrogenous matter in the blood, the patient begins to show signs of uræmia, vomiting and diarrhœa set in, and the uræmic symptoms disappear. To have arrested the diarrhœa and vomiting would have been to paralyse nature's compensative efforts. This extraordinary function of the alimentary canal must be educated, and yet not to excess, for in proportion as this function develops the assimilative function will fail. Compensative vomiting occurs in many cases of cardiac disease, after a meal. The heart's action being seriously disturbed by the pressure of the food in the stomach, reflex vomiting occurs, the food is ejected, and the heart is relieved.

In advanced fatty degeneration of the heart, we find vomiting and marked dyspepsia. After food is taken into the stomach it is absorbed directly and indirectly into the blood; this raises the intra-vascular pressure, more resistance is offered to the contraction of the fatty ventricle, and the heart becomes distressed. By reflex action the absorption in the alimentary canal is arrested, and vomiting of the unabsorbed food takes place. In those forms of heart disease in which there is stagnation of the blood in the large veins of the abdomen, diarrhœa sets in, and the circulation is relieved to a certain extent.

In patients that suffer from habitual constipation, diarrhœa is apt to occur; an attempt is thus made to relieve the loaded bowels by a flux. Not till thorough evacuation of the bowels has occurred must the flux be controlled.

December 9.

Dr. Vincent Harris read a paper on the 'Etiology of Anæmia.' Using the term anæmia in the same sense as it was first used by Andral, it is defined as a special morbid state, in which there is

a relative diminution of the mass of blood, the general composition differing from the normal standard; or in other words, a state in which the mass of blood is diminished, the liquor sanguinis is watery, poor in albumen, and contains an excess of salt, and the red blood corpuscles are deficient. As regards classification, anæmia may be roughly divided into three sets of cases:—

1. Those in which the blood is diminished in quantity and deteriorated in quality by direct losses, such as hæmorrhages; or by indirect losses, such as fluxes.

2. Those cases in which the quality of the blood is deteriorated from mal-nutrition, and depending upon discoverable organic lesions.

3. Those cases in which the same change is brought about from apparent mal-nutrition, with no coarse lesion to account for it.

Under Class 1 are included *acute anæmia* from traumatic causes, as surgical operations, venæsection, detachment of the placenta, from blows, injuries, and the like, and more chronic cases from congestions, inflammations, ulcerations, and diseases of the blood. The most frequent cases of anæmia are from gastric hæmorrhage, due very often to gastric ulcer or cancer; hæmorrhage from the uterus, a normal function carried too far; and hæmorrhage from piles—the last being not infrequently an important constituent in the cachexia observed in cirrhosis of the liver. Next with regard to fluxes. One of the chief is the *drain of albumen* from the blood in chronic renal disease, producing, at all events in part, the well known ‘pasty and waxy’ look which the patients with this disease invariably have. Another important flux is *excessive lactation in the strong and ordinary lactation in the weak*, which acts by abstracting from the blood the allied albuminous substance, casein. Dr. Barnes says that this is most likely to occur in the working classes, where deficient food, bad hygiene, and hard work combine to produce a very thin, worn and pallid condition, and to cause, in addition to uterine troubles, such as sub-involution, vertigo, syncope, and dimness of sight.

Prolonged suppuration and purulent discharge, as in abscess, empyema, purulent peritonitis, chronic eczema, leucorrhœa, and gonorrhœa, cause anæmia in this way. Sometimes anæmia from gonorrhœa is extreme. Dr. Regal reported a case of excessive anæmia in a girl aged 23, who had suffered from gonorrhœa for five years. It may be well to mention in passing that some observers trace the anæmia in these last cases not to the purulent

discharge, but to the strong balsamic remedies used in their treatment. Bronchorrhœa is said to cause anæmia.

Diarrhœa is the most important flux, and soon produces an anæmic state, whether it is the diarrhœa caused by simple irritation or organic disease.

In reference to the second division of the subject, affections of the spleen are perhaps most liable to be complicated with anæmia. In many of the enlargements of this organ there is an increase of white blood-corpuscles, but in 'hypertrophy of the spleen' a condition is produced, called by Niemeyer and others 'cachexia splenica,' which is a true anæmia. Malarial fevers, remittent and intermittent, thus indirectly produce the condition. Mr. Dickenson, of the Bengal Medical Service, describes such an anæmia as a sequela of one of the most malignant malarial fevers of India, called 'Bhootan.' He adds to an account of the affection, 'the emaciation and peculiar sand-like colour of the skin were so remarkable, that among a crowd of invalids you could at once, without hesitation, pick out with perfect certainty the "Bhootan-wallahs," as they were named by the other soldiers.' That some anæmias occurring in malarial districts are not always due to enlargement of the spleen, several French physicians record a series of cases of tropical anæmia occurring in Cayenne in 1867-68. In these they state, that the morbid state was due to the presence of a parasite found everywhere in the mucous membranes, except in that of the large intestines.

Diseases of the stomach and intestines appear to cause anæmia when from any cause dyspepsia arises, but simple gastralgia may produce the same effect. Affections of the lungs (especially phthisis), liver, kidneys, uterus, peritoneum, and other parts may give rise to anæmia, if severe or long continued.

The third division of the subject includes the cases of so-called idiopathic anæmia. It may be subdivided into—

First, what are called diseased states of the blood; and second, disease of the vascular system and nerves. In the first are anæmias due to impure air, such as of coal-mines, bad hygiene, starvation, and acute fevers; the syphilitic, carcinomatous, scorbutic, purpuric, and scrofulous states; chronic poisoning by lead, mercury, antimony, bicarbonate of potash, and other substances. These are not all true cases of anæmia, because, although they are accompanied by pallor, &c., the blood also contains, we are bound to believe, some foreign substances.

The second subdivision consists of the cases of idiopathic anæmia, that is to say, cases of anæmia in which all the above-

mentioned causes have been excluded, and where, after death, no coarse lesion has been found in any organ. Some cases called idiopathic during life have been proved after death to have been due to some lesion undiagnosed. Peukert reported a case of this kind, which was explained, on autopsy, by the discovery of a cancerous ulcer of pylorus; and others, at first obscure, later on show symptoms and signs of well-marked disease.

Dr. Addison prefaced his work on the supra-renal capsules with some remarks on 'a very remarkable form of general anæmia occurring without any discoverable cause whatever,' in which, after death, nothing was found but some fatty degeneration of the heart, semilunar ganglion, and solar plexus. This was the first instance, we believe, of such a disease having been noticed. Since Dr. Addison's time, many cases of what he called 'idiopathic anæmia' have been recorded.

Lately Corazza (in 1868) reported a case in a girl, aged 24, in whom nothing was found after death to explain her extreme anæmia.

Dr. Gairdner, too, relates a good case of like kind in his clinical medicine, and adds, 'that with regard to this most mysterious form of disease, it has no real relation to chlorosis of young women. It occurs in men, and often during middle age. The colour of the skin is essentially different from that of chlorosis, having, in fact, a tendency to sallowness. The general appearance is much more that of malignant disease than of any other condition with which I am familiar.'

Gasserow published five cases in 1871 of the same kind.

Biezmer stated that he had met with fifteen cases in five years in which anæmia occurred without any discoverable cause but fatty change in the papillary muscles, and of the small vessels of different organs, and less frequently extreme fatty degeneration of the heart.

We have ourselves seen three cases during a period of about two years. The result was sufficient to justify the diagnosis of pernicious or idiopathic anæmia.

There are a few, but very few, cases of fatal anæmia in which no changes, not even fatty degeneration, have been found on autopsy. These may be put down in some way to nervous causes. They are usually somewhat sudden, and occur from, as it were, continued shock.

The fatty changes which have been found in these obscure cases are said by some to be the effect, and not the cause, of the abnormal circulation. Fatty change resulting from an imperfect nutrition of the organs.

It is not unlikely, therefore, that the first pathological lesion is one in the sympathetic system, especially as Dr. Addison's case showed fatty change in the solar plexus and semilunar ganglia.

The anæmia which appears in Graves' or Basedow's disease, an affection which is now almost universally acknowledged to be nervous in its origin, shows that anæmia can be a nervous symptom, and the symptoms usually noticed early in cases of fatal anæmia, such as palpitation, restlessness, and languor, point also to the nervous system as the first part of the body affected.

In support of this theory may be also adduced, that the starting-point is sometimes to all appearance mental; a great grief, as in Dr. Gairdner's case, or a sudden shock. And again, that in sudden fatal cases, those of so-called nervous phthisis, no fatty changes have been discovered.

Mr. Bruce Clarke read the notes of a case of 'Intra-thoracic Tumour.'

December 16.

Mr. Bruce Clarke read a paper on 'The Spread of Cholera.'

Though it was not until 1831 that cholera appeared in Europe, yet it seems to be certain that it had been known in India some twenty years before that date; whilst if we accept the statements of some, it existed there in a modified form for a much longer period.

It is only since 1831, however, that we have begun to know anything of its natural history. Various views have been held as to its nature.

The first fact observed about its spread was that it attacked the great lines of traffic; hence it was assumed to be contagious, a conclusion since shown to be erroneous. Since the attack of 1848 it has been shown by Pettenkofer and others that three factors—place, time, and individual—are necessary for its propagation; hence it is usual to speak of the local, temporal, and individual disposition.

Shortly after this attack, it became evident, when the march of cholera was accurately mapped out, that whilst it was undoubtedly true that the places attacked were ranged along the great traffic lines, still other factors came into play, for whilst Paris and Marseilles were almost decimated, Lyons remained free. But further than this, one part of a town suffered more than another. Munich, Flaidhausen, and Berg were good examples of this. In these places it was almost universally

found that the houses attacked were situated on the limestone gravel, whilst the overlying brick-clay afforded an almost perfect immunity from the disease.

The rationale of this fact is not far to seek. Moist soils which are not saturated with water contain a considerable amount of air, which is hourly drawn into our houses whenever they are warmer than the external air. In a clay soil, however, there is but little of this ground air, and hence the comparative immunity which it affords.

A soil which has been recently wet and then suddenly deprived of its interstitial water is in a far more favourable condition for the spread of cholera, owing, it is believed to the passage of the air over the moist earth.

The time of year most favourable for an epidemic appears to be the end of summer and beginning of autumn: of this fact there seems no doubt. Besides this, it must be obvious that some individuals are more prone to catch the disease than others; it may pass all through a house, and yet exercise apparently the most capricious selection.

In England, though these conclusions have in the main been accepted, yet the local variations appear to be hardly as clearly recognisable as in Germany.

But if the conditions accessory to the spread of cholera are well made out, it is far otherwise with the active agency by which this disease is circulated; that it is very readily conveyed by the *fœces*, however, is very certain.

Various ideas have been put forward on this subject. The cholera germ has been supposed to be fungoid; it has been alleged to be a bacterium; but so far all to no purpose. Lately, however, the experiments of Drs. Lewis and Cunningham in India have shown that a temperature of 100° C., even if kept up for some considerable period, does not destroy the vitality of the poison, thus negating the idea that it was due to a low form of animal or vegetable life. On the contrary, it would appear that it should be placed rather in the same category with snake-poisons, and that it is more nearly allied to the alkaloids.

The only practical conclusions that can be drawn from such a study of this disease are (1) the necessity for cutting off from our houses the supply of ground air, and (2) the liberal use of disinfectants.

January 13.

Dr. Moore showed some pathological specimens.

1. A small urinary calculus passed *per urethram* after the use of diuretics.

2. Part of the left middle cerebral lobe, showing an abscess with a well-defined wall, associated with general basal meningitis, and with necrosis of the petrous portion of the temporal bone.

3. A drawing representing the face of a child with congenital syphilis, showing a peculiar form of disease of the nose.

Dr. V. Harris showed a specimen of an intra-thoracic tumour.

Dr. Venn read a paper on 'Post-Partum Hæmorrhage.'

January 20.

Dr. Hall showed a specimen of the sputum of a case of plastic bronchitis.

Mr Hastings read a paper on 'Addison's Disease.'

After introducing the subject, the author gave a brief historical sketch of our knowledge of the malady.

The symptoms are those of increasing debility, languor, restlessness, depression, nausea and vomiting, with loss of appetite, pains about the loins, feeble heart's action, with bronzing of the skin, beginning in the most exposed parts. There is little emaciation, the temperature is rather low, and the mental faculties unimpaired.

Undefined in its outset, it usually ends fatally within two years. The progress is often intermittent.

Some cases are recorded which have remained latent and proceeded to a fatal termination in a few days.

The author related in detail the notes of a case in which he had verified the diagnosis by an autopsy. The state of the supra-renal capsules was abnormal, they were enlarged and adherent to the surrounding parts, with spots of pigmentation scattered over the surface. The substance was firm, yellow, and cut with difficulty. The left capsule contained three small cysts, full of pus and cretaceous matter, no distinction existing between the medulla and cortex. The right capsule was a putty-like mass, with a thin shell of fibrous tissue.

Mr. Hastings described the structure of the capsules, with special reference to their nerve supply, and considered the minute structure of the morbid changes to be identical with that of tubercle, the nerves and other adjoining structures becoming involved.

The general symptoms are similar to those produced by severe hæmorrhage, and are due either to the heart failing to supply blood in sufficient quantity to the brain, or to the blood being drawn away by the abdominal veins.

The pigmentation is seated in the deeper layers of the

epidermis, and is more intense around the nipples, umbilicus, flexures of limbs, &c.

Mr. Hastings described the cutaneous pigmentation as being due to the dilatation of the capillaries from paralysis of the vaso-motor nerves, and refers to the pigmentation resulting from blisters as analogous. The dark lines and patches of pigmentation in the mouth are less easy to explain, though they partly depend upon irritation from the teeth.

Dr. Greenhow considers that the most frequent causes of this disease are abscess connected with the vertebræ, blows, strains, &c.

The disease is three times more common in men than in women, and occurs chiefly in adult life.

The vomiting is best treated by bismuth, hydrocyanic acid, &c.

The application of the continuous current to the hypochondria and sides of the neck has proved serviceable during exacerbations.

Mr. Hastings showed to the Society a patient with well-marked symptoms of Addison's disease.

Mr. Hames showed three patients illustrating the good effects of transplantation of the skin after extensive losses.

January 27.

Dr. Hall showed a patient (suffering with subacute rheumatism) having transposed viscera.

Dr. V. Harris showed a heart with vegetations on the mitral valve and purpura of the endo-cardium, spleen and kidney, with numerous infarcts.

The cerebellum had a large blood-clot on the right side.

Dr. Hall read 'Remarks on Diseases of the Heart.'

Attention was first drawn to the importance of a careful study of the subjective symptoms in heart disease, as Dr. Hall, though far from underrating the immense assistance to be derived from the use of the stethoscope, could not help believing that too much reliance was placed on the physical signs alone. The paper, which was of a practical nature, was chiefly founded on notes of fifty-two cases which had been under the author's care, those of disease of the mitral and aortic orifices being selected as by far the most numerous, and consequently the most important. In considering the etiology of mitral disease, in more than half the cases referred to it was due to rheumatism; pleurisy and chorea being other causes; in one-fourth of the cases the origin was not specified. As rheumatism, which is the most frequent cause of

endocarditis, occurs chiefly in young people, so mitral disease is often met with at an early period of life, whereas aortic disease, being generally due to atheroma, is seldom found except in those advanced in years.

The symptoms of the various forms of heart disease were then treated of in detail. Palpitation was first considered, and Bellingham's points of distinction between palpitation due to organic disease of the heart, and that independent of organic disease of the heart, were quoted with approval; but it was suggested that the list might have been augmented by stating how frequently palpitation is due to excess in tea and smoking, and to the consequent digestive derangements.

The causes of functional palpitation were enumerated as follows:—

1. As occurring in young persons of both sexes, but especially females, whose digestion is impaired, and who lead a sedentary life; frequently associated with pain in the splenic region or in the intercostal muscles.

2. In old people, with degenerate arteries, the cause being probably a like degenerate change occurring in the heart from impaired nutrition.

3. Of a reflex origin, due to the stomach being over distended with food, or the ingestion of irritant articles, such as ice or shell-fish, in some persons.

4. A poisoned or depraved condition of the blood, as in gout, rheumatism, anæmia, leucocythæmia, &c.

Under the heading of palpitation dependent on organic disease of the heart were grouped two distinct classes of cases.

1. Where there is some organic disease of the heart or its valves.

2. Where the heart is acting at a disadvantage in consequence of some displacement, as from pericardial or pleural effusion, &c., distension of the abdomen by flatus or fluid, &c. As regards the cause of palpitation, the old idea that it was due to increased action of the heart was discarded, and it was stated that palpitation implied not augmented power, but laboriousness of action.

Palpitation is much more frequently met with in mitral than in aortic disease, and this fact was well illustrated by the cases on which the paper was founded.

Whenever palpitation occurs, the points to be taken into consideration are its severity, frequency, constancy, circumstances giving rise to it, such as exertion, going upstairs or uphill, mental emotion, &c.; and if these be well weighed, a correct conclusion can generally be arrived at as to its cause, though, of

course, the physical signs will always be of the greatest importance in checking the result which may be arrived at.

Attention was next directed to dyspnoea, as being the symptom most commonly present in valvular disease of the heart, and the one which generally leads to the patient seeking medical advice.

The panting and gasping character of the dyspnoea, with the oppression at the chest, especially on making the slightest exertion, even in the early stages of heart disease, were alluded to. Dr. Ormerod's graphic description in his Gulstonian Lecture was quoted to show how patients with heart disease die.

The chief cardiac causes of dyspnoea were stated to be—

1. Failing power of the heart's action, due either to loss of blood or shocks to the nervous system.
2. Valvular disease of the heart, and especially of the mitral orifice.
3. Any displacement or other affection of the heart which interferes with the proper performance of its functions.
4. Degeneration, fatty or atheromatous, most frequently met with in people getting on in years.

The pulse has in all ages been rightly looked upon as a most valuable aid in diagnosis and prognosis, and in no disease more so than in cardiac affections. The pulse which has attracted the greatest amount of attention is that met with in aortic regurgitation, and called the 'locomotive pulse' by Bellingham and Todd, and by others named the 'water-hammer pulse' or 'the pulse of unfilled arteries.' An easy mode of identifying an indifferently marked pulse of this description is to get the patient to elevate the arm above the head, so as to throw the force of gravity into the scale.

An intermittent pulse need not necessarily give rise to much anxiety, as it is frequently met with in people getting on in years without any other serious symptom, in gouty people, and in those subject to flatulent dyspepsia.

It is, also, however, met with in valvular disease, and in fatty and other forms of degeneration of the heart.

In an irregular pulse some of the beats are very rapid and others slow. An unequal pulse signifies that owing to a want of uniformity in the heart's action, the quantity of blood thrown into the arterial system varies, so that a full pulse may the next moment be followed by a feeble one. An irregular and unequal pulse are both of much more unfavourable augury than one which is simply intermittent.

The frequency of the pulse is an important point; a very slow pulse—say one of forty—would suggest the idea of fatty degeneration if met with in a person past adult life.

As a general rule, it may be stated that in mitral disease the pulse is frequent, quick, and irregular; whereas in aortic disease the pulse is of normal frequency, or it may even be somewhat less frequent than normal, slow and regular.

By frequency is understood the number of beats per minute, and by quickness the duration of the beat under the finger. Cough, which is so frequently met with in heart disease, and especially in affections of the mitral valve, is due to the congestion of the lungs. In aortic disease, so long as the compensative hypertrophy of the left ventricle is able to overcome the impediment to the circulation, there is an entire absence of any pulmonary symptoms.

Hæmoptysis is a very important symptom in cardiac disease, as it shows that the conservative power of nature, which has hitherto sufficed to keep the blood within the vessels, has at last given way, and the part where the block begins is that in which the hæmorrhage commences, and so we get hæmoptysis. It was shown that the blood in cases of heart disease came from the pulmonary capillaries, whereas in asthma it is furnished by the bronchial capillaries. Dr. Hyde Salter was quoted to prove that 'while the occurrence of the hæmorrhage has a very unfavourable influence on the prognosis of the heart disease, the prognosis of the hæmorrhage itself is not as a rule unfavourable.' In only three of the cases on which the present paper was based did hæmorrhage occur. Epistaxis and purpura are other forms of hæmorrhage due to cardiac disease.

Dropsy is another of the very common symptoms in cardiac disease, though generally a late one. It usually commences as a slight puffiness about the ankles, but in the progress of the disease anasarca may invade all the tissues of the body, and ultimately there may be fluid in all the serous cavities, and the patient usually dies from œdema of the lungs increasing the dyspnoea caused by the hydrothorax. The only other symptom to which allusion was made was that of pain or discomfort in the cardiac region.

The author's observations were quite in accordance with what Dr. Walshe says on the subject—namely, 'that pain is more commonly connected with disease of the aortic than of any other valves.'

Patients have various ways of expressing their pain and discomfort. One will say that he has a dull aching pain; another speaks of a gnawing pain; and a very common complaint is a feeling of tightness or constriction across the cardiac region.

As regards prognosis, many points have to be taken into consideration, such as the occupation, habits, and social position of

the patient, as a gentleman of easy means, who lives a quiet and temperate life in a healthy climate, stands a very different chance to a labouring man addicted to drink and living—if such a word is not a misnomer—in one of the reeking dens which are such a disgrace to the civilisation of this great city.

In aortic regurgitation, as is now well known, sudden death not infrequently occurs, and it is a curious fact that the freer the heart is from any other disease, the greater is the liability to this termination.

In aortic stenosis patients may enjoy very good health so long as the compensative hypertrophy of the left ventricle is sufficient to neutralise the harmful effects of the diseased valves, and patients may not even suffer from dyspnoea, a symptom which, as has been already shown, is experienced in almost all cases of mitral disease. Death is usually brought about in aortic disease by œdema of the lungs, or more rarely by embolism. In aortic regurgitation apoplexy is sometimes the cause of the fatal termination. Another mode of death, as has been before stated, is syncope.

In mitral disease general dropsy, œdema of the lungs, hæmoptysis, and hæmorrhagic infarctions in the lungs, and intercurrent maladies, carry off the sufferers. As regards prognosis, one point is of particular importance, and that is, that the loudness of a murmur *per se* has nothing whatever to do with its gravity. A high-pitched loud murmur may simply be due to the vibration of a small vegetation on one of the valves, which interferes very little with the proper performance of its functions, whereas it is well known that even loud murmurs occasionally cease before death, owing to the heart being unable to propel the blood with sufficient force to cause the murmur.

The next division of the paper consisted of an abstract of the notes of typical cases of the various forms of heart disease, which may be omitted here.

The chief points to be observed in the treatment of patients suffering from mitral disease is to maintain their strength by appropriate diet, to give tonics, of which the preparations of iron, and especially the tincture of the perchloride, are the most useful; but of all drugs, *the remedy par excellence* is digitalis. Its cumulative action has been held up as a bugbear, and has prevented full benefit being derived from it. A patient under the author's care suffering from mitral regurgitation took twenty drops of the tincture three times a day for a whole year without intermission, and improved in a most marvellous manner. Palpitation and præcordial pain are best relieved by the application of a belladonna plaster. Should hæmoptysis occur, the indication

is to relieve tension in the vessels by hydragogue purgatives. Ascites and œdema are to be combated in a similar manner.

In the treatment of aortic stenosis the great object is to maintain the nutrition of the left ventricle, and prevent degenerative changes taking place, so that it may be enabled to overcome the obstruction offered by the contracted orifice. For this purpose the albuminous and fatty articles of diet, such as meat, milk, cream, cod-liver oil, &c., are very useful. Iron also acts as a blood tonic, and stimulant may be required.

In aortic regurgitation, on the other hand, the indication for treatment is not quite so clear, as the patients suffering from this form of heart disease appear more full-blooded than in aortic constriction, and may even suffer from apoplectic symptoms; consequently they do not stand quite so stimulating a plan of treatment. Like all sufferers from heart disease, they should be warned to keep quiet, to abstain from eating and drinking to excess, and a daily evacuation of the bowels should be secured. Dr. Hall laid down three rules for the use of digitalis in aortic disease—1. That it is contra-indicated in an uncomplicated case of aortic regurgitation. 2. That when the aortic disease is complicated with secondary mitral affection, it is often extremely beneficial. 3. That when the pulse is comparatively infrequent, say 60 to 72, digitalis is more likely to do harm than good.

Dr. Hall concluded by briefly alluding to the benefit to be derived from venesection to the amount of four to six ounces in cases in which the right side of the heart was engorged; the removal of this amount of blood would allow the ventricle to contract upon its diminished contents, and the impetus thus given would often continue for a long time. Repeated bleedings, by producing a state of anæmia, would be highly injurious, and tend to accelerate the fatal termination.

Mr. Schofield and Mr. Lewis showed some microscopical specimens.

February 3.

Mr. Mills read a paper on "The Administration of Anæsthetics."

Mr. Mills began by remarking that the administration of anæsthetics should not be left entirely to specialists, for any medical man may be called on at any time to give an anæsthetic, and every opportunity should therefore be seized of becoming thoroughly conversant with the best and safest means of administering one or more of them.

He then referred briefly to the history of anæsthetics, remark-

ing that it is interesting to note that anæsthesia was not entirely unknown in A.D. 50.

Decoction of mandragora was recommended by Dioscorides to produce sleep in those about to undergo an operation.

Theodoric in the thirteenth century recommended that spongia somnifera impregnated with spirituous extracts of various narcotic substances should be held to the nostrils till sleep was induced. But anæsthetics did not come into use until 1844, when Dr. Horace Wells of Boston took nitrous oxide gas for extraction of a tooth, its properties having been discovered by Sir H. Davy in 1798. The properties of ether were discovered in 1818 by Faraday, and it was first employed by Dr. Morton in 1846. The properties of chloroform were discovered by Waldie in 1847. It was really first used in the form of chloric ether, which is merely chloroform diluted with alcohol, at St. Bartholomew's Hospital, by Sir William Lawrence and Mr. Holmes Coote. But Sir James Simpson was the first to use pure chloroform.

1. Conditions supposed to contra-indicate the use of Anæsthetics.

Much has been written about the danger of anæsthetics, especially chloroform, in cases of heart disease; but the only affection which contra-indicates them is fatty degeneration, which is extremely difficult to diagnose. And if it were necessary for a patient with fatty disease to undergo an operation, he would be much more likely to die from the shock of the operation without an anæsthetic than he would be to die from the anæsthetic, even were this chloroform, and there would be still less danger if ether were used.

Patients with slight valvular disease, who are otherwise healthy, as a rule take anæsthetics very well.

2. Preparation of Patient.

A patient who is about to undergo an operation under the influence of an anæsthetic should take no solid food for some hours previous, to lessen the chance of vomiting.

He should have a good meal about five hours before the time appointed for the operation, and after that nothing but beef-tea or soup with a glass of wine two hours before the operation.

It is very important that the stomach should be empty at the time an anæsthetic is given, not only on account of the danger of the patient being choked by some undigested portion of food being vomited into the pharynx, and at the next inspiration being drawn down into the larynx or trachea, and so asphyxiated.

ing the patient, but on account of the accompanying faintness which is so commonly seen both before and after the vomiting.

Without doubt the process of digestion is much interfered with by the nervous state in which a patient always is previous to the operation. This is particularly the case in nervous women, who will sometimes vomit large pieces of undigested food during an operation, when it is certain they have taken nothing solid for three or four hours previously.

What is the best course to pursue if retching occur while the patient is going under? Some say, Push the administration, and the retching will pass off without vomiting. But this is by no means certain. It is better, therefore, to allow the patient to come round a little, and encourage the vomiting; and if there be food in the stomach, get the stomach thoroughly empty (by tickling the fauces, if necessary) before proceeding any further with the anæsthetic, and by this means all further trouble about sickness will probably be avoided. If after retching nothing comes up, and the stomach appears to be empty, the anæsthetic should be continued, and as the patient gets under its influence the retching will pass off.

A case was then related in point.

3. Relative Merits of Chloroform and Ether.

Mr. Mills remarked that much has been said of late as to the relative merits of chloroform and ether, and numerous letters have been written in the medical papers. But up to the present time chloroform has been used so many more times than ether that it is not possible to say for certain which is the better anæsthetic; but from experiments on animals, it appears that the heart may be paralysed by chloroform, but not by ether.

Judging from the experience he had had of the action of the two, Mr. Mills remarked that ether is decidedly the more stimulating, chloroform acts rather as a depressant. He had never seen that faintness under ether which is common towards the end of long operations under chloroform.

Persistent after-sickness, which is sometimes so very troublesome after chloroform, is very rare after ether. He had met with but two cases in nearly one thousand administrations.

The reason that the use of ether was given up for that of chloroform was, that it required much more of the former drug to produce the desired effect; it was much more unpleasant to breathe, producing a disagreeable sensation of choking; it took longer in action; there was often much struggling while the

patient was going under ; and it was followed by a delirium resembling that of intoxication.

But by the improved methods of administering it which have now come in use, many of these disadvantages are entirely done away with.

He referred especially to the plan of giving some nitrous oxide gas first.

So far as it is possible to judge at present, there seems to be less danger attending the administration of ether than that of chloroform, and it should therefore be used in all suitable cases.

4. *Cases in which Chloroform is to be preferred.*

In some cases chloroform is preferable ; in operations about the mouth and nose, for instance.

The influence of chloroform lasts longer than that of ether, and it can very conveniently be given on a piece of lint when there is a gag in the mouth, which would admit too much air, and be very much in the way of a face-piece if ether were given. In addition to this, ether causes much more coughing and choking, and excites a flow of viscid saliva, which is very unpleasant in such an operation as cleft palate, where the operator requires the parts to be as still as possible, and it is necessary for the surgeon to get a good view of them without the necessity of perpetually sponging out saliva from the fauces.

In delicate operations about the eye, Mr. Mills said he prefers chloroform, as it causes less congestion and hæmorrhage, and the patient, as a rule, is quieter under its influence.

In cases of fracture which require an anæsthetic whilst the parts are being placed in apposition during the time the muscles are relaxed, chloroform is preferable, because patients recover quietly from its effects ; the inhalation of ether being generally followed by a state of noisy delirium and struggling, which would be likely to displace the fractured ends, and necessitate their readjustment.

For children, too, chloroform is generally used, because they take it so well, and it is very rarely followed by any unpleasant symptoms.

5. *Chloroform.*

Many different kinds of inhalers have been invented for giving chloroform, but Mr. Mills said that he preferred just a doubled piece of lint, on which a little chloroform is poured from time to time from a drop-bottle.

When fresh chloroform is put on, it should be held two or three inches from the patient's face, and gradually brought nearer as the vapour gets weaker. This requires a little practice, but one soon gets into the way of doing it almost mechanically. It is just as well to smell the lint occasionally, to ascertain that there is sufficient chloroform on it without its being too strong.

The best apparatus for giving chloroform, if one be used, is Mr. Clover's.

The apparatus was shown, and the way in which it is used described.

The chief objections to this apparatus are that it is expensive and cumbersome; and as some patients require a much stronger dose than others, it may be necessary to refill the bag in the middle of an operation, which is very awkward unless there is some one at hand to assist. Then, again, unless great care is taken, there may be too large a percentage of chloroform to air. The apparatus may, moreover, get out of order from disuse; one case of this kind has happened, and resulted in death to the patient. It was found that instead of 1100 cubic inches of air having been passed into the bag, only about 840 had.

However, it is a very ingenious apparatus, and in the hands of Mr. Clover works admirably.

The patient should be in the recumbent position, should have none but light garments on, and these should be quite loose. All that is required is a drop-bottle containing the chloroform, a piece of lint, and a pair of dressing forceps.

The patient should be told to breathe quietly, and not to take violent inspirations. It is well to begin by dropping very little chloroform on the lint (from five or ten to twenty drops, according to age), and hold it about two or three inches from the face, just to allow the patient to become accustomed to its smell. This should gradually be brought nearer to the face, and the quantity from time to time increased, but gradually, so as not to provoke coughing or choking; if either of these occur, the lint should be held a little further from the face for a time.

'If we begin with very small proportions of chloroform and gradually increase it, we find that the system gradually accommodates itself to the influence, there is no discomfort or terror at the outset, there is no resistance, and the phenomena assimilate to those of natural sleep.'

Claude Bernard has proved by experiments that animals accustomed to vitiated air will live in an atmosphere that is instant death to a healthy animal. A sparrow will live for three hours in a glass bell breathing the same air over and over again,

but if a fresh sparrow be put in at the close of the second hour it will immediately die.

The pulse should be held with one hand and the lint with the other. The respirations and the face should be carefully watched.

At first the patient should be allowed to lie comfortably without being held. As he begins to struggle, he should be held sufficiently to prevent his raising himself, and to prevent his doing any mischief with his legs and arms, but it is not necessary to lean your whole weight on him and keep him absolutely still. Patients suffering from delirium tremens and drunkards always give the most trouble in this stage. In any case, there is always less struggling if the administration is gradual and not hurried.

It is of the greatest importance to watch the patient carefully during this stage.

Mr. Mills thought it best to go on with the administration when an unusual amount of struggling and straining occur, unless the breathing be impaired, in which case the lint must be removed and the patient allowed to have a few inspirations of pure air, and then if breathing well, the chloroform may be continued. It is best while the patient is going under to turn the head to one or other side.

A patient is generally said to be ready for the operation when touching the inner border of the eyelids or ocular conjunctiva produces no reflex action; but the cornea is more sensitive than the conjunctiva, and it is better to judge by it. The eye is, however, by no means a certain test, as its sensitiveness varies very much in different patients, and it is best to judge also by the relaxation of the muscles.

Some patients are ready for the operation when the cornea is still sensitive; others will start if the operation be commenced though the cornea be insensitive.

For operations about the more sensitive parts, as the genitals or anus, it is necessary to get the patient more deeply under. The operation in these cases should not be commenced until the pupil no longer acts with light. If the chloroform be pushed further than this, there may be loss of reflex action in the involuntary muscles, and in some cases, especially in children, expulsion of feces and urine. The muscles used in respiration may fail, and lastly the heart itself stops.

When the patient is under, he should be kept so with small quantities of chloroform. Time should not be given for him to come round before putting on more. It is well to remove the lint for a few seconds just as he gets under, because of the cumulative property of chloroform. Its effects may become more intense half a minute after leaving off administering it,

which is no doubt due to a portion remaining in the lungs which is afterwards absorbed.

The pulse generally beats quicker at first, then sinks to normal, and in long operations may become slow and weak. It may become irregular or cease suddenly.

The respiration must be carefully watched. It is not sufficient to simply watch the chest-walls and abdomen, we must listen to the breathing, especially when there is the slightest doubt, as the chest-walls will sometimes go on moving though no air is entering the lungs. The respiration may become slow and shallow, and if the chloroform be pushed too far, may cease altogether. If shallow, it may often be improved by drawing the chin from the sternum. If it stop, the administration must of course be immediately stopped, and without loss of time. The sternum should be compressed once or twice, and this will generally induce respiration; but if it fail, the tongue must be drawn forwards with a pair of forceps, and Sylvester's method of artificial respiration used without delay, but with this difference from the ordinary mode, that the arms must be first pressed to the side to empty the lungs, and then drawn over the head. If this does not at once succeed, it should be continued, and if necessary persevered with for an hour or an hour and a half, or as long as there is any hope of recovery.

Galvanism should be applied, one pole to the epigastrium and the other to the neck, to galvanise the diaphragm and phrenic nerve. The current should be broken while the chest is being compressed, and applied while the arms are being raised, to imitate natural respiration, and because strong and continuous currents appear rather to exhaust than to restore muscular activity.

A hot-water bottle should be applied to the legs and friction employed, and the patient kept warm with blankets.

An enema of brandy may be given per rectum, and when the patient has come round sufficiently to swallow, brandy should be given by the mouth.

Sometimes the pulse stops simultaneously with the respiration, and there is then of course less probability of resuscitation; but some cases have by prompt and active treatment, such as that which has been just described, been brought round even when this has occurred. No time should therefore be lost in employing them. Nélaton's method too, of supporting the patient by the legs, head downwards, has proved successful in some cases.

Mr. Mills said he thought, as Dr. Snow had said, that 'such remedies as dashing cold water on the patient and applying ammonia to the nostrils can hardly be expected to have any

effect on a patient who is suffering from an overdose of chloroform, for they would have none whatever on one who had inhaled it in the usual manner, and is merely ready for the operation, but in no danger.'

During the excited stage the conjunctiva becomes congested, particularly if there is much struggling, and the pupil dilates; but as the patient gets under, the pupil contracts, and when fully under does not act with light. Dilatation generally occurs when the patient is about to vomit.

Snoring may be caused in two ways. The first, or palatal stertor, is like the snoring of natural sleep, and is of no consequence; it is merely caused by the flapping of the soft palate, and may occur at any time during the administration. The second, or laryngeal stertor, is of great importance, because it only occurs when there is some obstruction to the entrance of air into the larynx. It generally occurs when the patient is deeply under, and is more laryngeal in character. It is caused by the falling together of the parts at the opening of the larynx. The tongue is apt to fall back unless the face is turned well over to one side.

When this sound is heard the administration should be stopped, and the chin drawn forcibly away from the sternum; by this means the apposition of the epiglottis with the back of the pharynx is prevented, and the air-passage straightened. If this does not stop the stertor, and there is reason to think that sufficient air is not entering the lungs, the tongue must be drawn forwards with a pair of dressing forceps; but this last procedure is very rarely necessary if the precaution has been taken to turn the head over on one side, and drawing the chin from the sternum generally stops the stertor.

The face sometimes becomes somewhat livid during the struggling stage; the lint should then be held further from the face, because, as the patient is slightly asphyxiated, it is very undesirable to give too strong a vapour of chloroform.

Or the face may become livid when laryngeal stertor occurs; or if the breathing be otherwise impeded, as by spasm of the glottis, which is induced by giving too much chloroform at the commencement of the administration; or by a clot of blood in the larynx, which is only likely to happen in operations about the mouth or face. The chloroform must be stopped when lividity occurs.

Mr. Mills then related a case in point. The operation was for the removal of a tumour of the superior maxilla in a woman, and was accompanied by profuse hæmorrhage. The patient took chloroform very well, but towards the end of the operation he noticed that although the chest-walls were moving, no air was

being respired, and the patient was becoming rather livid. Not more than a minute before this she had been turned over on to her side, and her mouth was opened just to ascertain if any blood had found its way into the mouth. As none was found, she was turned on her back again and the operation proceeded with. She was then breathing well, and one minute afterwards it was found that no air was entering the lungs. She was immediately turned on to her side again, her mouth was opened, some blood ran out, and he pulled from the back of the throat and larynx two large blood-clots. She then immediately took some deep inspirations and went on breathing. Her pulse remained good. This case shows the advantage of listening to the respiration carefully, and also watching the face; for though the chest-walls were moving, no air could be heard entering the lungs, and the face was becoming slightly livid.

The face may become white from loss of blood in the operation, or because the patient is about to be sick, or because he is faint from the effects of chloroform. In any case, the administration should be stopped and the head lowered; if it does not soon pass off, it is well to give some hot brandy-and-water.

It sometimes happens during the early stage of the inhalation that the patient has a fit; although this is rare, it is well to consider beforehand what would be the best course to pursue if such a thing happened.

An hysterical fit is not dangerous, and so the chloroform should be continued, and it passes off satisfactorily; but in an epileptic fit there is always danger on account of the asphyxia. The chloroform must therefore be instantly stopped should this occur.

Death may occur suddenly, from sudden cessation of the action of the heart, or from spasm of the larynx, induced by giving too large a proportion of chloroform to air at the commencement. And it is also said to occur from shock, from an insufficient quantity having been given.

Death may occur gradually, by asphyxia, from an overdose, or from obstruction to the air-passages; or by syncope induced by hæmorrhage, vomiting, or an overdose.

6. *Nitrous Oxide Gas.*

Nitrous oxide gas being without smell, is the most pleasant anæsthetic to take. It is suitable, however, for short operations only, such as the extraction of a tooth or opening an abscess, and the chief thing in its administration is to take care that no air is admitted, and to see that the respiration is not impeded.

If used for extraction of a tooth, a small gag or prop should be placed between the teeth at a convenient distance from the tooth to be extracted, to keep the mouth wide open. Having ascertained that the gag is held firmly between the teeth, the administration may be commenced.

The patient may be told to take good deep inspirations. It is a very good plan to allow the patient to hold the operator's hand while going under, and as the muscles become relaxed, the hand drops, and he is then ready.

It may be taken sitting up or lying down. If the breathing is arrested, one compression of the sternum will generally suffice to restore it.

It leaves no unpleasant symptoms, such as headache or sickness.

If faintness occur, the patient's head should be lowered.

One death only is reported. Gas was given for the extraction of a tooth. No post-mortem examination was allowed, but a piece of the gag used was missing, and whether or not it found its way down the larynx has remained a mystery.

Good strong gags should therefore always be used, and care taken that the teeth when extracted are not left loose in the mouth.

Mr. Mills showed a very convenient apparatus for administering the gas, and some gags made by Mr. Lyons.

7. *Ether.*

The ether which should be used for inhalation is the pure anhydrous ether, or the anhydrous methylated ether with a specific gravity of about 720. The latter being made from methylated spirit, is cheaper than the former, but is equally good for inhalation, and has been used by Dr. Keith of Edinburgh for many years.

The same preparations that are necessary before chloroform, are necessary before the inhalation of ether, but the patient should particularly avoid stimulants for some hours before the operation. The ether itself acts as a stimulant.

It may be given on a towel rolled up into a cone with a sponge fixed in the apex, or on a felt cone made for the purpose. It is very unpleasant to breathe, and generally causes a sensation of choking or suffocation, and induces coughing and salivation.

The patient, if an adult, should be warned that after a few moments' inhalation, it will give rise to a very unpleasant sensation of choking, and he should be exhorted when he feels this coming on to take a few good deep inspirations, instead of trying to

put the face-piece off, and the unpleasant feeling will then soon pass off. At the same time, preparation must be made, with the assistance of others, to hold him firmly at this stage, should he struggle and endeavour to pull the face-piece off. It is very foolish to allow him to come round and to argue with him, because he is simply intoxicated with ether, and it will then be necessary to go through the same thing again; and if you hold him, and keep the face-piece on, he will soon get under, and on coming to, will remember nothing about the struggling.

The administration differs from that of chloroform, too, inasmuch as it is not necessary to commence so gradually. An ounce may be poured on to begin with, and another ounce added from time to time to keep the towel or felt saturated with ether until the patient is well under. Very little air is required. If he become livid, the face-piece should be removed for one or two respirations, and then replaced, recovery being very quick in the early part of ether inhalation.

The pulse is generally quick and bounding from beginning to end, and the respirations are forcible and quick.

The patient is ready for the operation when the muscles become relaxed; it is seldom necessary to get the conjunctiva insensitive, but this varies very much, more even than with chloroform.

Some patients can take ether with comparative ease, and others cannot be induced to inhale it. Mr. Mills recommended that in the latter case chloroform should be given until the commencement of the exciting stage, when the air-passages become insensitive, and then ether substituted.

8. *Nitrous Oxide Gas and Ether.*

Mr. Mills then described Clover's apparatus for giving nitrous oxide gas and ether, the apparatus which is now commonly in use for nearly all operations at St. Bartholomew's Hospital. Its object is to give some gas to render the air-passages insensitive before giving the ether. In this way the patient goes quickly and quietly under the influence of the gas without struggling or coughing.

Mr. Mills said that he first tried giving gas with an ordinary gas-apparatus, and having got the patient under its influence, quickly substituted an ether-inhaler for the gas face-piece; but the patients recovered from the effects of the gas, and struggled violently before the ether had time to have more effect than to cause excitement. But by Clover's apparatus the ether is turned on gradually after a few inspirations of gas, then without remov-

ing the face-piece the gas may be turned off and ether only breathed ; so that when the gas is turned off, and ether turned on, the patient has already been breathing ether.

Mr. Mills, in recommending this apparatus, said that it was particularly advantageous in dental cases. Patients generally recover from nitrous oxide gas somewhat suddenly, but if some ether is given too, they remain much longer narcotised, and awake gradually. Then, too, the amount of ether can be so easily regulated, according to the time the effects are required to last.

If ether alone is given, it takes some time for the patient to get under its influence. If ether is preceded by gas, the patient is under in about a minute and a half or two minutes ; and in short operations, having inhaled less ether than if it were used from the commencement, requires a shorter time to recover from its intoxicating effects. Indeed in this way ether may be given in dental cases sufficiently to very materially prolong the effects of the gas without leaving any intoxication whatever.

The administration by this apparatus certainly requires a little care and practice. The most difficult part of it is to know exactly when to turn on some ether.

If it is turned on too soon, it induces coughing and struggling. If it is not turned on soon enough, the long-continued inhalation of the gas produces so much lividity that it is necessary to allow the patient one or two inspirations of fresh air, and then he is almost sure to come round. It is most difficult to lay down any rule as to when the ether may be turned on ; it requires a considerable amount of judgment, but, as a rule, from about the eighth to the twelfth inspiration seems to be the best time.

But patients breathe in such different ways, that one cannot, say for a certain time or for a certain number of respirations, give gas and then turn on ether.

Some being perhaps afraid of the gas, take very small inspirations at the beginning, and so take longer going off.

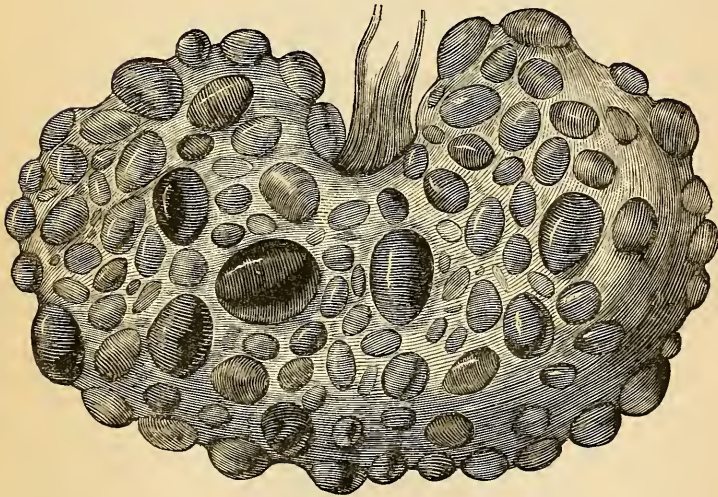
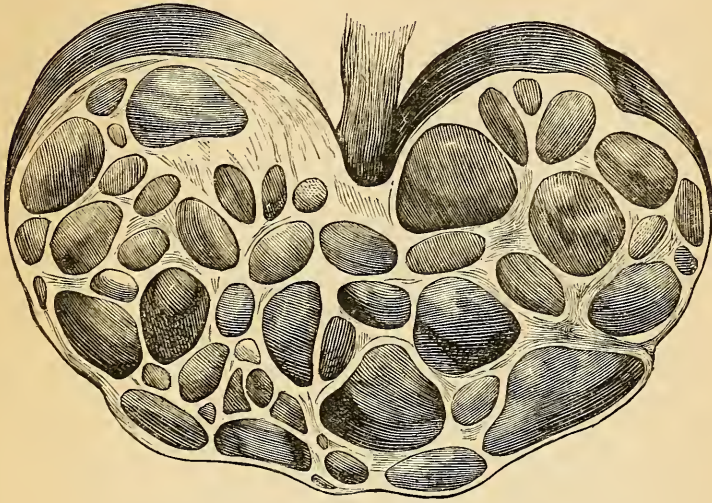
If on turning on the ether there is any coughing, it must be turned off again for one or two inspirations ; but it is desirable to begin the ether as soon as it is possible to do so without causing any unpleasantness, and gradually turn on more and more until only ether is being inhaled. Then the face-piece should be removed once in about six or eight respirations, and more frequently towards the end of long operations, to allow the patient to get a breath of pure air.

February 10.

Dr. Vincent Harris showed two kidneys presenting well-

marked cystic disease, and read short notes of the case from which they were taken.

The kidneys (engravings of which are appended) were enlarged,



measuring six to six and a half inches in length, and four in breadth, and weighing about twenty-two ounces each. The

surface of the kidneys was irregular, being studded with large and small cysts, containing fluid, varying in colour from a pale straw to a dark reddish brown. The largest cysts were situated along the convex borders of the kidneys, and were of the size of a nut; the size of the others varied from a pin's head upwards. Some of the cysts were collapsed, but the majority were tense. The capsules could be removed only in places. On section, the proper cortical substance was almost entirely occupied by cysts and fibrous matrix; very little of the medullary substance also could be seen. The cysts appeared larger on section than before. Several of the largest measured about three-quarters of an inch across. The pelves and ureters of the kidneys were not dilated or otherwise abnormal. On examination of the contents of the cysts, the fluid was found to be very albuminous ($\frac{1}{3}$), and faintly acid. The sediment contained altered blood-cells, fatty and granular débris, and numerous plates of cholesterine. There was no evidence of the presence of uric acid.

Notes of the case.—G. F., æt. 48, a butcher's porter, was talking with some friends in the Smithfield Market on January 4, 1876, when he suddenly fell down, became unconscious, and was found by the bystanders to be paralysed on the right side. When admitted into the Hospital, he was semi-conscious, but soon became quite comatose. His pupils were equal, not contracted, and acted to light. Temperature, 97.6° F. Pulse, 100; full and regular. He continued in very much the same state until the time of his death on the 7th, except that his temperature gradually rose, and reached 102° just before he died. On the only occasion when it was possible to save a little of his urine, it was found on examination to be pale, cloudy, of specific gravity 1016, and to contain lithates and a slight trace of albumen.

Autopsy disclosed a large clot on the left side of the brain, disorganising the corpus striatum and optic thalamus, and extending into the lateral ventricle. The left side of the heart was hypertrophied and dilated, without valvular disease, and the arteries generally were atheromatous. Pleuro-pneumonia in an advanced stage on the right side was discovered.

The kidneys have been already described.

Mr. Samuel Benton read a paper upon 'The Treatment of Talipes.'

The object of this paper was to draw attention to a simple and efficient way of treating patients, especially children, suffering from club-foot, without recourse to operative measures and expensive instruments.

The usual cause of failure in curing cases of talipes is—1stly,

want of perseverance and daily attention ; 2dly, discharging them without a proper-shaped shoe, or some retaining apparatus.

Great stress was laid upon the indisputable advantages of tenotomy, but its practice being easy, and apparently devoid of risk, the operation is now very often abused.

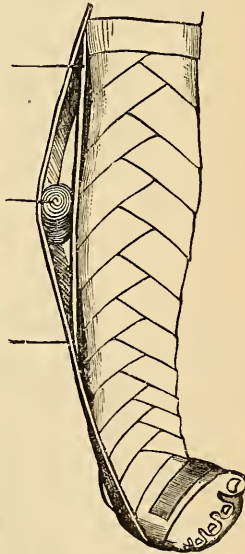
As Dr. Little has it, no surgeon is justified in operating upon a child, unless he personally, or a trustworthy assistant, can conscientiously follow up daily the after-treatment, which is of far more importance than the division of tendons. It is very difficult, indeed, to overcome re-contracted tissues, except by slow and cautious proceedings, and it then requires the greatest vigilance to prevent a slough.

Each case of talipes needs a different, or at least some modification in the treatment, therefore it is impossible to contrive a boot which will cure all cases. In fact, in many cases, by ordering an expensive instrument, the deformity is eventually aggravated.

More depends upon the tact, patience, and perseverance of the surgeon than upon the apparatus employed. The daily application of a few strips of adhesive plaster, a wooden splint, and a cleverly-adjusted bandage, may in ingenious hands supply the place of the most elaborate contrivances (*vide wood-cut*). After the restoration of the foot to its normal position, a retaining apparatus must always be employed ; by this means repetition of operations and treatment is rarely requisite. The paper was illustrated by a number of cases which had been successfully treated by bandaging.

Mr. Benton also showed a variety of appliances used in the treatment of talipes, many of these being made by Messrs. Arnold & Sons under his own direction.

Particular attention was called to a retentive boot worn after treatment, by which means the foot could be turned in or out by the aid of a simple screw at the heel.



February 17.

Mr. Macready read the notes of a case of stricture of the urethra. The patient was admitted to the Hospital, and after

thirty-six hours rest in bed a No. 3 catheter was passed with difficulty. The urine was drawn off, and a few drops of blood followed the withdrawal of the catheter. Within an hour a severe rigor occurred, and delirium set in. The rigor lasted fifteen minutes. Temperature, 108° . Pulse, 132. (A slight rigor after the passage of a catheter was not unusual in his case.) Copious sweating ensued, and within two hours vomiting of dark blood-stained matter. Temperature, 106° . The following day the patient was collapsed, sweating profusely. Temperature, 102° . Death occurred the following day, the patient having had almost complete suppression of urine since the rigor.

Mr. Schofield read the notes of three cases—two of them albuminuria, the third empyema.

The interesting features of the first case were—

1. Considerable prolongation of life, though the patient seemed to be *in extremis* on admission.
2. Large quantities of albumen and casts in the urine contrasted with great diminution before death.
3. Total suppression of urine on two occasions for forty-eight hours without coma or convulsions.
4. Great relief was afforded by wet cupping.

In Case II. the absence of pulmonary and heart complications, the slight œdema and the uræmic convulsions ending fatally. General eczema occurred, and was cured by a sitz bath.

In Case III. was exemplified the difficulty of diagnosis and the sometimes negative results of exploratory puncture with a fine trocar; also the advantage of thoracentesis.

February 24.

Mr. Burgess showed a congenitally malformed heart with an imperfect ventricular septum.

Dr. Brunton read a paper on 'Diuretics.'

The motions of the body are kept up, like those of the steam-engine, by combustion. In both, carbonic acid and water are formed by the process, and an incombustible residue of ash is left behind. The carbonic acid and water pass off by the chimney in the steam-engine, and by the lungs in the body; while the ash falls into the ashpit in the steam-engine, and the products of waste in the body are excreted by the kidneys. The bladder in which these waste products temporarily accumulate before being finally ejected may therefore be compared to the ashpit of the steam-engine, and the kidneys to the grated floor of the furnace, which allows the ash to fall through, while it retains such large cinders as are likely to be of still further use in keeping up

the fire, although very small ones may be allowed to pass. Thus the healthy kidneys will allow not only urea, the ultimate product or ash of the combustion of nitrogenous substances in the body, to pass freely through them, but also uric acid which is not completely oxidised, and might be compared to a small cinder. On the other hand, however, they refuse to let albumen or peptones, which may be compared to fresh coals, pass through them; nor even under ordinary circumstances do we find kreatin, kreatinin, and suchlike substances, which we may compare to large cinders, in the urine.

But while the function of the kidneys resembles that of the furnace bars in affording an exit to waste material while retaining substances of value, the manner in which the function is discharged in the two cases is totally different, the grating acting like a simple sieve, the kidneys like a most complicated filter. The arteries of the kidney divide within the organ into branches, which without anastomosing with each other in the manner in which arterial twigs usually do in other parts of the body, suddenly break up into little bundle-like plexuses, the glomeruli, as if the usual anastomoses had all been collected into one small point in each vessel. The vessels in these anastomosing bundles again unite into a single one called the efferent vessel, and in contradistinction to the one which branched out to form the plexus, which is termed the afferent artery. The arrangement of these two is very peculiar, and deserves particular attention. The whole plexus or glomerulus is enclosed in a bag or capsule, the purpose of which we will consider presently. The afferent artery and the efferent vessel both pass through the same aperture in this bag, but the afferent artery begins to divide into a plexus as soon as it gets inside the aperture, while the efferent vessel comes from the very middle of the plexus. In consequence of this the blood flows easily through so long as the pressure in the interior of the efferent vessels is moderate; but when it becomes too great, they press the branches of the afferent vessel against the sides of the capsule, and so close them. The flow through the afferent vessels is thus stopped until the efferent vessels have had time to empty themselves, so that we have here a regular governor-ball arrangement to guard against any excessive pressure in the glomeruli. This, at any rate, is the explanation given by Ludwig, and I believe that it is quite correct; but at the same time this arrangement reminds me strongly of the operation of wringing a towel, and the intermittent squeezes given by one set of arterioles to the other must, I think, assist in pressing fluid out of them. For this part of the kidney is a filtering apparatus, the glomerulus being the filter through

which water holding in solution urea and salt passes from the blood into the capsule, which is the funnel wherein they are collected. From this capsule a tube passes, which at first is wide and winding, afterwards becomes narrow and straight, and then becomes again wide and winding before it passes finally into a wide straight tube. The fluid from the capsule would easily flow away if the tube were wide along its whole length, but the contraction prevents this, and obliges it to remain for some time in the wide part at the beginning of the tube, and only allows it to flow slowly away.

The efferent artery, after leaving the glomerulus, breaks up into a plexus which is distributed over the surface of these tubes. Thus the blood, which has been deprived of much of its water in the glomerulus, again comes into close contact with the fluid it has lost, but separated from it by the epithelium lining the tubules. Here the blood again takes up some of the water and salt, leaving the urea, so that the solution of urea gets more and more concentrated as it passes along, until it is discharged along the straight tubes into the ureters. Thus, the greater the pressure behind, the more quickly does it flow, and the less time is there for absorption and concentration in the tubules. The less the pressure behind, or the greater the resistance in front, the more slowly does it flow, the more time is there for absorption, and the more scanty and concentrated is the urine. There are thus two factors which cause the amount of urine to vary—1st, The pressure of fluid from behind, *i.e.*, the pressure of the fluid exuding from the glomeruli; and 2d, The resistance in front, which consists chiefly in the pressure of urine in the ureters.

We will now consider these separately. 1st, The pressure from behind. The quantity of fluid exuding from the glomeruli depends in the kidney, as in an ordinary filter, upon the pressure within them. The greater the pressure of blood, the more fluid passes out of the vessels into the capsule. As we have seen that this increases the rapidity of the flow within the tubuli, and does not allow time for absorption, it is evident that increased blood-pressure must increase the flow of urine in a double manner, and at the same time render it more watery.

Now the pressure in the glomeruli may be raised or decreased like the pressure in a gas-jet. Just as the pressure of gas in the burner rises or falls with the pressure in the main, so does that in the glomeruli rise and fall with the general pressure of the blood in the aorta and arterial system. You cannot have a high pressure in your burner if that in the main is low, and you cannot have a high pressure in the glomeruli if that in the aorta is low; but still you can modify the pressure in your

burner within certain limits, by means of the stopcock, letting on the gas fully when the pressure is low, and turning it partly off when too high; and so it is with the kidney. When the arterial pressure is low, the renal arteries can dilate and let in more blood, and when the arterial pressure is too high, they can contract, and prevent the pressure in the glomeruli from being too great; but notwithstanding this modifying power of these vessels, the blood pressure in the kidney, as a general thing, rises and falls with the general blood pressure.

This pressure may be raised by drinking water, and thus increasing the bulk of the blood, and large draughts of water form a most efficient diuretic. The pressure also rises when the vessels of the skin are contracted by the application of cold, and the blood they would otherwise contain is driven in upon the other vessels. In some experiments I made several years ago, I found that if the weather suddenly changed from warm to cold, the excretion of urine rose to nearly double its previous amount.

It is possible also to raise the pressure locally by dilating the renal arteries, and Claude Bernard says that irritation of the vagus has the power to do this, and so to increase the secretion of urine. Blood pressure may be lessened generally by abstinence from fluids, by heat, which dilates the vessels, by general debility, causing loss of tone, and all these lessen the urine.

It may be lessened in the kidneys by the contraction of the renal vessels. But the contraction must be considerable to do this.

If the artery does not contract to less than one-half its usual diameter the flow of urine is not diminished, but if it is contracted to less than one-ninth the flow of urine is entirely stopped; and after the circulation is allowed to go on again the urine secreted becomes albuminous.

The contraction of the renal arteries may be caused either by the vaso-motor centre in the medulla, or by conditions present in the kidney itself, of which a different composition of the blood passing through it is, in all probability, one of the most important.

Having now considered the means by which the pressure of blood in the kidneys and the flow of urine from them may be increased or diminished, we may now proceed to consider the action of diuretics.

The first of these is water. This increases the pressure in the glomeruli by augmenting the bulk of the blood, but it increases the amount of urinary water out of proportion to the increase in blood pressure. This is probably because the blood, after

leaving the glomeruli and passing into the plexus, is not so greedy of water as it would otherwise be, consequently does not abstract so much from the liquid contained in the tubules.

The next diuretics are common salt, and other salts of potash and soda. These act as diuretics by causing people to drink more than usual by the thirst they create ; but this is not all.

They have a specific action on the kidney, and cause it to secrete more urine even when its nerves are cut.

They may do this by causing contraction of the efferent vessels and plexuses, and thus raising the pressure in the glomeruli, but this we cannot be certain about.

The third diuretic in my list is digitalis. This drug in moderate doses causes the vessels to contract and the blood pressure to rise, and it thus acts as a diuretic. But in dropsy it has a further action ; and after its administration the fluid which has been effused into the tissues is often seen rapidly to disappear at the same time that the urine has been increased. Now we might say that this effect was simply due to the blood sucking up the fluid from the tissues, in order to replace what had been drained away from it in the kidneys. But this does not seem to be the whole truth.

Edema, we must remember, depends on more fluid exuding from the blood-vessels than can get back by the lymphatics and veins ; in other words, it is the *excess* of fluid which has filtered out from the vessels. Now the amount of fluid which filters out from the vessels generally bears exactly the opposite relation to the general blood pressure that the secretion of the kidney does. When the vessels generally contract the blood pressure rises, and the kidney secretes copiously, but the very contraction of the vessels which supplies this organ with extra blood is cutting it off from some others, and is lessening the supply of fluid to them.

At the same time the absorbents take up the fluid more rapidly from the tissues, and the œdema is diminished in a two-fold way. There is less fluid poured out into the tissues, while there is more taken from them, and the excess is poured out by the kidneys.

This effect of digitalis is due to its action on the vaso-motor centre in the medulla oblongata, by which it influences the vessels of the body generally.

But Mr. Power and I have found that, in addition to this general action, it has a special action on the vessels of the kidney. When we gave a large dose of digitalis to an animal we found that the blood pressure rose, but instead of the urine being increased, as it ought to have been, the secretion stopped

altogether for a while, and then began to flow with great rapidity, although the blood pressure had now sunk below the normal, and the urine ought, according to rule, to have been scantier than normal.

The explanation we gave of this curious fact was, that the digitalis had at first caused contraction of the arteries generally, and so raised the blood pressure in the aorta, but had at the same time caused the renal arteries to contract so completely that no blood flowed through them, and thus their secretion was arrested. When this action upon the vessels began to pass off the renal arteries dilated more quickly than the others, so that the proportion of blood running into them raised the pressure above the normal.

Now this action of digitalis on the renal arteries leads us to consider another function of the kidneys, viz., that of eliminating poisons, and gives us an explanation of the so-called cumulative action of the poisonous substances which we use as remedies.

A great number of poisons are eliminated by the kidneys, and are thus prevented from exercising any prejudicial effect upon the body.

The arrow poison, woorari, produces paralysis when injected into the blood or into a wound, but is harmless when swallowed. The reason of this is that it is excreted by the kidneys as quickly as it is absorbed from the stomach, and thus there is never a sufficient quantity in the blood to produce any bad symptom. But if the kidneys are extirpated or the renal arteries tied, it cannot get out by the kidneys, and thus accumulates in the blood and causes death. Now most of the drugs we use, when given in larger quantities than can be eliminated, begin to show a gradual increase in their action, but there are two which form a marked exception. These are digitalis and strychnia, which, after being continued for some time, occasionally cause sudden symptoms of poisoning, and on account of this peculiarity they are specially said to have a cumulative action. The reason why digitalis does this, I believe, is, that after it has been accumulating gradually in the blood, it begins to act on the kidneys in the way I have mentioned, and thus stops its own elimination, and suddenly accumulates in the blood, although the quantity taken by the stomach is not increased. In support of this, I may mention that a sudden diminution in the secretion of urine precedes or accompanies the appearance of the symptoms of poisoning in these cases, and in one instance the urine was stopped entirely for about two days. I do not know whether strychnia has a similar action on the renal arteries, but I think it highly probable.

This action on the renal arteries also explains the fact that sometimes digitalis will not act as a diuretic at all, for if it persistently keeps up contraction of these vessels, it is evident that no contraction of those in the other parts of the body will increase the blood pressure and the secretion from the kidneys.

Besides digitalis, we have several other diuretics of great repute—viz., squill, broom tops, spirits of nitrous ether, buchu, pareira, &c. ; but how these act we do not know.

I have now come to consider the effect of resistance in front as a cause of diminished secretion. While we are standing or sitting, the kidneys are high up above the bladder, and the urine runs freely down the ureters, so that there is no pressure to retard secretion. But in the recumbent position this is not the case, and although the pressure the secretion has to overcome is but slight during health, yet still it does exist, and may be one cause why we pass less urine during the night than during the day.

In diseased conditions, however, the pressure may be greatly increased, and may form an important hindrance to the action of diuretics. Thus in dropsy, when a great accumulation of fluid has taken place in the abdominal cavity, it is often found that the diuretics we give do not act well. But if the patient be tapped, and the distension of the abdomen removed by the withdrawal of fluid, the kidneys resume their function and secrete abundantly, in all probability because the pressure on the ureters, which previously hindered their secretion, is now gone.

Hydragogue cathartics also aid the action of diuretics, but what the exact *modus operandi* is I am not prepared to say, but I think it not improbable that they may act by relieving the congestion of the liver. When the liver is congested, it will more or less press on the vena cava, and the slightly increased resistance may partly impede the flow of blood from the renal veins. This will cause increased resistance in the capillary plexus of the kidney, distension of the efferent and compression of the afferent vessels of the glomeruli, and consequently diminished flow of blood and less secretion of urine.

The addition of a little mercury in the shape of blue pill to digitalis and squill is found greatly to add to their efficacy, and the cause of this may be the action of mercury on the liver.

The uses for which diuretics are employed are—

1. To remove fluid from the body when it has accumulated in the cellular tissue causing œdema, or in the serous cavities causing dropsy.

These conditions depend on, 1st, venous congestion, such as is consequent on valvular disease of the heart, allowing regurgita-

tion and dilatation of the right heart, in consequence of emphysema; or disease of the liver, causing pressure on the portal veins.

The second cause is disease of the kidneys themselves. In this case the œdema is more apt to show itself first in the eyelids and scrotum, or wherever there is loose cellular tissue; while in œdema from heart disease it is more likely to be in the feet and legs, while the eyelids and scrotum may be free.

The diuretics we use in heart disease are chiefly digitalis and squill, although much benefit is often derived from associating these with bitartrate of potash and also a little blue pill.

In dropsy depending on the liver we may use mercury more freely along with squill and bitartrate of potash. Digitalis in such cases has not such a beneficial action as in cases depending on heart disease. In dropsy from disease of the kidneys we generally try to relieve these organs as much as possible by diaphoretics and purgatives, but we also use potash, broom, digitalis, squill, and copaiba.

In the second place, diuretics are used to increase the secretion of urine, and thus diminish its irritant qualities when the bladder or urethra are inflamed. For this purpose alkalies and copaiba are used, the latter having both a diuretic action and a healing power over the inflamed mucous membranes themselves.

In the fourth place, they are used to prevent the urine from becoming too concentrated, and depositing calculi in the bladder.

In the fifth place, they are employed to eliminate the products of waste.

For this purpose water itself is an excellent diuretic, and many people suffer from not employing it sufficiently. A very gouty old gentleman once told me that by two methods he had succeeded in warding off an attack of gout for a long time. The first of these was to drink a large quantity of water in the morning. 'For little water,' said he, 'is a great cause of gout;' and whenever you get a gouty patient in the upper ranks of life, always ask him how much water he drinks. You will generally find that they take a small cup of tea in the morning, and a small cup of coffee in the evening. This is all the water they take except what they get in the shape of wine, beer, or brandy and soda—water pure and simple many of them never touch. In such persons gout may often be warded off by washing out their tissues with a large draught of water in the morning, and making them drink more water and less wine at meals. But if this is not enough, and the gout still threatens, give them twenty grains of nitrate and thirty of bicarbonate in a large tumbler of water.

In this way we get rid of the products of nitrogenous waste which are supposed to produce gout.

But this brings us to the question, do the kidneys secrete urea or not, or is the condition which occurs in advanced disease of these organs, and termed uræmia, due to urea formed by them but not excreted.

Several attempts have been made to decide this point by examining the blood from the renal vessels, and comparing the amount of urea in the blood going to, with that returning from the kidney.

The great objection to these experiments is, that it is very difficult indeed to estimate the urea in blood with any degree of certainty.

But there is another method which has been followed by Rosenstein. He destroyed one kidney but left the other. If urea were formed by the kidneys the amount excreted should be diminished, but instead of this he found that it remained the same, and therefore concluded, and I think rightly, that urea is not formed in the kidneys.

For my part, I believe that it is formed partly in the liver and partly in the tissues, and that the chief agents in its formation are the ferments in the body which split up its albuminous constituents into urea, or some substances such as kreatin, &c., which yield it, and into some non-nitrogenous substances, such as glycogen, sugar, or fat. These are burnt up and excreted as carbonic acid and water, while the urea is passed out by the kidneys. In fevers this process goes on more rapidly, and the old treatment of fevers, and one still much used, is to give diuretics to get rid of the waste; but of late years we are gaining such knowledge of the action of such drugs as quinine and salicylic acid, as inclines us rather to attempt to arrest the decomposition of the tissues, and thus stop the fever which their oxidation causes, than simply to remove the products of their waste.

How much room there is for farther investigation into the action of drugs I think this paper will show, for, if I have tried to give you some idea of the mode of action of such a diuretic as digitalis, I have been obliged to tell you frankly that, regarding the mode of action of most of them, I know no more than you do, and come here in the expectation that some of you during the course of the discussion may very possibly light upon a reasonable explanation of what is still a puzzle to me.

March 2.

Mr. Sheehy showed—1. Bony mass within the eyeball. 2. Foreign body within the eyeball. 3. A glaucomatous cup.

Mr. R. Bruce read the notes of a case of "Diaphragmatic Hernia" that terminated fatally.

Mr. Vernon read a paper on the antiseptic treatment.

March 9.

Mr. Macready read the notes of a case of cancerous stricture of the Sigmoid flexure.

Mr. Reid showed an improved form of suppository tube.

Dr. Ormerod read a paper on "Thoracic Aneurysm."

EXAMINATIONS, 1873-74.



Lawrence Scholarship and Gold Medal—

E. CRÉTIN.

Brackenbury Medical Scholarship—

A. J. STURMER.

Brackenbury Surgical Scholarship—

E. CRÉTIN.

Senior Scholarship in Anatomy, Physiology, and Chemistry—

F. S. EVE.

Junior Scholarships—

T. E. HAYWARD.

R. H. A. SCHOFIELD.

H. T. STEELE.

Open Scholarship in Science—

R. H. A. SCHOFIELD.

Preliminary Scientific Exhibition—

T. E. HAYWARD.

Jeaffreson Exhibition—

F. H. CRADDOCK.

Kirkes Gold Medal—

J. MACREADY.

Bentley Prize—

J. K. BARTON.

Hichens Prize—

S. M. SMITH.

Wix Prize—

A. S. ECCLES.

PRACTICAL ANATOMY.

SENIOR.

- Foster Prize—*F. S. EDWARDS.
 2. F. S. EVE.
 3. T. J. VERRALL.
 4. W. A. SHOOLBRED.
 5. A. UPTON.
 6. W. R. PEARLESS.
 7. H. A. GLYN.
 8. A. G. WILLIAMS.
 9. { J. W. ROUGHTON.
 { H. SLOMAN.
 11. L. B. CALCOTT.

JUNIOR.

- Treasurer's Prize—*P. A. STEEDMAN.
 2. { A. R. ANDERSON.
 { T. G. DAVEY.
 4. H. T. STEELE.
 5. G. O. MEAD.
 6. H. J. CAPON.
 7. J. WILMOT.
 8. R. H. A. SCHOFIELD
 9. A. J. BATHE.
 10. R. F. CUMMING.

EXAMINATIONS, 1874-75.

Lawrence Scholarship and Gold Medal—
(Not awarded).

Brackenbury Medical Scholarship—
G. H. HAMES.

Brackenbury Surgical Scholarship—
M. VERNON.

Senior Scholarship in Anatomy, Physiology, and Chemistry.
R. H. A. SCHOFIELD.

Junior Scholarships—
G. COATES,
M. PRICKETT,
R. GILL.

Open Scholarship in Science—
G. COATES, } Equal.
J. C. SAUNDERS, }

Preliminary Scientific Exhibition.
R. GILL.

Jeaffreson Exhibition—
T. W. H. GARSTANG.

Kirkes Gold Medal—
G. H. HAMES.

Bentley Prize—
F. W. EVANS.

Hichens Prize—
A. UPTON.

Wix Prize—
(Not awarded.)

PRACTICAL ANATOMY.

SENIOR.
*Foster Prize—*P. A. STEEDMAN.
2. H. J. STEELES.
3. R. H. A. SCHOFIELD.
4. { W. J. HAMES.
 { G. O. MEAD.
6. A. R. ANDERSON.
7. T. WILMOT.

JUNIOR.
*Treasurer's Prize—*C. J. BAMBER.
2. C. B. LOCKWOOD.
3. M. PRICKETT.
4. D. A. COLES.
5. C. A. D. CLARKE.
6. G. COATES.
7. E. C. BOUSFIELD.
8. R. J. COLENZO.
9. N. W. BOURNS.

EXAMINATIONS, 1875-76.



Lawrence Scholarship and Gold Medal—

R. H. A. SCHOFIELD.

Brackenbury Medical Scholarship—

R. H. A. SCHOFIELD.

Brackenbury Surgical Scholarship—

W. PYE.

Senior Scholarship in Anatomy, Physiology, and Chemistry—

G. COATES.

Open Scholarship in Science—

C. P. LUKIS.

Preliminary Scientific Exhibition—

A. C. DISMORE.

Jeaffreson Exhibition—

T. KIRSOPP.

Kirkes Gold Medal—

A. G. WILLIAMS.

Bentley Prize—

T. J. VERRALL.

Hichens Prize—

F. H. CRADDOCK.

Wix Prize—

F. H. CRADDOCK.

PRACTICAL ANATOMY.

SENIOR.

*Foster Prize—*G. COATES.

2. W. GRAHAM.
3. C. LOCKWOOD.
4. { C. J. BAMBER.
M. PRICKETT.
6. G. P. SYLVESTER.
7. E. C. BOUSFIELD.
8. ALLEN DINGLEY.
9. G. W. P. DENNYS.

JUNIOR.

*Treasurer's Prize—*C. SHEPHERD.

2. A. J. WHARRY.
3. H. C. NANCE.
4. W. OUTHWAITE.
5. G. H. BARLING.
6. C. F. CUTHBERT.
7. A. FRANKLIN.
8. { A. A. BOULBY.
G. L. PARDINGTON.
K. TOWNSEND.

ST. BARTHOLOMEW'S HOSPITAL & COLLEGE.

THE MEDICAL AND SURGICAL STAFF.

Consulting Physicians—Sir G. Burrows, Bart., D.C.L.,
F.R.S., Dr. Farre, Dr. Martin, Dr. Harris.

Consulting Surgeon—Sir J. Paget, Bart., D.C.L., F.R.S.
Physicians—Dr. Black, Dr. Andrew, Dr. Southey, Dr.
Church.

Surgeons—Mr. Holden, Mr. Savory, F.R.S., Mr. Callender,
F.R.S., Mr. Thomas Smith.

Assistant-Physicians—Dr. Gee, Dr. Duckworth, Dr. Hensley,
Dr. Brunton, F.R.S.

Assistant-Surgeons—Mr. Willett, Mr. Langton, Mr. Morratt
Baker, Mr. Marsh.

Physician-Accoucheur—Dr. Greenhalgh.

Assistant-Physician-Accoucheur—Dr. Godson.

Ophthalmic Surgeons—Mr. Power, Mr. Vernon.

Dental Surgeon—Mr. Coleman.

Administrator of Chloroform—Mr. Mills.

Casualty Physicians—Dr. Wickham Legg, Dr. Hall, Dr.
Moore.

Medical Registrar—Mr. Champneys.

Surgical Registrars—Mr. Butlin, Mr. Macready.

LECTURES.

Medicine—Dr. Black, Dr. Andrew.

Clinical Medicine—Dr. Black, Dr. Andrew, Dr. Southey,
Dr. Church.

Surgery—Mr. Savory, F.R.S., Mr. Callender, F.R.S.

Clinical Surgery—Mr. Holden, Mr. Savory, F.R.S., Mr.
Callender, F.R.S., Mr. Thomas Smith.

Descriptive and Surgical Anatomy—Mr. Thomas Smith,
Mr. Langton.

General Anatomy and Physiology—Mr. Marrant Baker.

Histology—Dr. Klein, F.R.S.

Chemistry and Practical Chemistry—Dr. Russell, F.R.S.

Materia Medica—Dr. Brunton, F.R.S.

Forensic Medicine and Hygiene—Dr. Southey.

Midwifery and the Diseases of Women and Children—Dr.
Greenhalgh.

Botany—Rev. George Henslow.

Pathological Anatomy—Dr. Gee.

Comparative Anatomy—Dr. Moore.

Ophthalmic Medicine and Surgery—Mr. Power.

Dental Anatomy and Surgery—Mr. Coleman.

Mental Diseases—Dr. Claye Shaw.

DEMONSTRATIONS.

Morbid Anatomy—Dr. Wickham Legg.

Diseases of the Skin—Mr. Marrant Baker.

Orthopædic Surgery—Mr. Willett.

Diseases of the Ear—Mr. Langton.

Diseases of the Eye—Mr. Vernon.

Practical Surgery—Mr. Marsh.

Practical Anatomy and Operative Surgery—Mr. Cumberbatch,
Mr. Walsham.

Assistant Demonstrators—Mr. Cripps, Mr. Keetley.

Mechanical and Natural Philosophy—Mr. Graham.

Practical Physiology—Dr. Shuter.

Assistant Demonstrator—Mr. Pye.

Medical Tutor—Dr. Norman Moore.

COLLEGIATE ESTABLISHMENT.

Warden—Dr. NORMAN MOORE.

Students can reside within the Hospital walls, subject to the College regulations.

Ten Scholarships, varying in value from £20 to £100, are awarded annually.

Further information respecting Scholarships, Pupils' Appointments, and other details, may be obtained from Mr. Marrant Baker, Dr. Norman Moore, and at the Museum or Library.



STATISTICAL TABLES

OF THE

Patients under Treatment

IN THE WARDS OF

ST. BARTHOLOMEW'S HOSPITAL

DURING 1875.

BY

THE MEDICAL REGISTRAR,
FRANCIS H. CHAMPNEYS, M.B. (Oxon.)—M.R.C.P.;

AND

THE SURGICAL REGISTRAR,
HENRY T. BUTLIN, F.R.C.S.

LONDON:

HARRISON AND SONS, ST. MARTIN'S LANE,
Printers in Ordinary to Her Majesty.

1876.

PREFACE.

No alteration is made this year in the Medical Tables.

Table III is omitted from the Surgical Tables. The cases included therein in the last Report are placed as formerly in the Medical Tables.

A Table of Anæsthetics is added this year to the Report.

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OCCUPATIONS OF MALE PATIENTS.

Acid workers	2	Chimney sweep	1	Foundrymen	2
Acrobat	1	Cigar makers	4	Frame makers.. ..	4
Artists	2	Cigar sorter	1	French cook	1
Asphalte workers ..	3	Clerks	52	French polishers ..	11
Author.. ..	1	Clickers	3	Fruit seller	1
Bagmaker	1	Clogmaker	1	Furnaceman	1
Bakers	12	Coachmakers	3	Furriers	2
Barbers	3	Coachman	1	Galvanizer	1
Barge builder	1	Coalheaver	1	Gardeners	16
Bargemen	2	Coal porters	3	Gasfitters	6
Barmen	19	Coastguard	1	Gatekeeper	1
Basketmaker	1	Coffee-house keeper ..	1	General dealers ..	4
Beer bottlers	6	Collar makers.. ..	2	Gilders.. ..	3
Billiard marker	1	Colour grinder	1	Glassblower	1
Blacksmiths	7	Combmaker	1	Glasscutter	1
Boatman	1	Comic singer	1	Goldminer	1
Bone turner	1	Commercial travellers ..	5	Grocers	6
Bonnet-shape maker ..	1	Commission agents	2	Grooms	4
Bookbinders	17	Compositors	13	Guards.. ..	2
Bootcutter	1	Confectioners	5	Hairdressers	6
Bootmakers	36	Conjuror	1	Harnessmaker	1
Boot-tree maker	1	Cooks	4	Hatters.. ..	7
Box carriers	2	Coopers	2	Hawkers	9
Brass dipper	1	Cork-cutters	4	Hop-picker	1
Brass finishers.. ..	4	Cork-sole maker	1	Horsehair carder ..	1
Brassfounder	1	Cornice-ring turner ..	1	Horse-tender	2
Brass workers	4	Corn samplers.. ..	2	Ice vendors	3
Brewers	14	Costermongers.. ..	3	Indiarubber worker ..	1
Bricklayers	10	Cowman	1	Instrument makers ..	3
Brushmakers	2	Customhouse officers ..	2	Iron-foundry labourer .	1
Builder.. ..	1	Detective	1	Ironmongers	2
Futchers	47	Dispensers	2	Iron moulders.. ..	2
Butler	1	Dock labourer.. ..	1	Ivory turner	1
Cabinetmakers	36	Drapers	43	Japanners	2
Cabmen	35	Draymen	7	Jewellers	6
Canal keeper	1	Drovers	3	Joiners.. ..	25
Cameo maker	1	Dyers	2	Keepers in lunatic asy-	
Carmen	129	Egg merchant.. ..	1	lums.. ..	4
Carpenters	52	Engine drivers	13	Labourers	549
Carpet maker	1	Engineers	22	Lace makers	2
Carriers	3	Engravers	6	Lamplighters	4
Carters.. ..	2	Envelope cutters	2	Laundryman	1
Carvers.. ..	3	Errand boys	18	Leadpipe maker	1
Case makers	8	Farrier.. ..	1	Leather dressers	9
Cashier.. ..	1	Figure maker	1	Leathercase maker ..	1
Cats'-meat-man	1	Firemen	5	Leather stainer	1
Cellarmen	13	Fish curer	1	Lemonade makers ..	5
Chaff-cutters	2	Fishing-rod maker	1	Licensed victualler ..	1
Chairmakers	2	Fishmongers	6	Lightermen	2
Cheesemongers	4	Fish salesmen	3	Lithographers	1
Chemists	5	Flower makers	2	Locksmith	1

OCCUPATIONS OF MALE PATIENTS (continued).

Lucifer maker.. ..	1	Poulterer	1	Sweeps..	5
Machinists	4	Press boy	1	Swimming master ..	1
Machine ruler	1	Press readers	2	Tailors	27
Masons..	14	Priest	1	Tassel maker	1
Match seller	1	Printers	65	Tea warehouseman ..	1
Meat dresser	1	Printers' boys	2	Telegraph boy.. ..	1
Mat maker	1	Publicans	4	Telegraphic marker ..	1
Metal polishers	2	Purse maker	1	Telegraph porter	1
Milkmen	8	Rag sorters	3	Telegraph jointer	1
M.R.C.S.	1	Riveters	2	Ticket collector	1
Musical instrument		Sailors	51	Timber man	1
maker	1	Sawyers	12	Tinbox maker	1
Musician	1	Scalemaker	1	Tinfoil maker	1
Navvies	5	Scavengers	2	Tobacco makers	2
Necktie cutter.. ..	1	Schoolboys	228	Tobacconists	9
Newsboy	1	Schoolmasters	4	Tobacconist's boy ..	1
Office boys	2	Servants	8	Tool maker	1
Organ grinders	2	Shepherd	1	Toymaker	1
Ostlers	16	Shipping agent	1	Travellers	15
Packers	9	Ship steward	1	Trimming maker	1
Page	1	Ship watchman	1	Tripe dresser	1
Painters	35	Shoeblocks	4	Turner	1
Paper colourers	2	Shoe finisher	1	Typefounder	1
Paperhanger	1	Shoe fitter	1	Umbrellamaker	1
Paper ruler	1	Shoemakers	12	Umbrella-stick maker..	1
Paper seller	1	Shopkeepers	2	Undertakers	3
Paper stainer	1	Shopmen	6	Upholsterers	8
Pedlar	1	Shorthand writer ..	1	Vellum binder..	1
Pensioners	2	Silk dressers	2	Waiters	20
Perfumer	1	Skinner	1	Warehousemen	25
Photographers.. ..	4	Smiths	23	Watch gilder	1
Pianoforte maker	1	Soap makers	2	Watch makers..	7
Pianoforte tuner	1	Soldiers	14	Watchmen	4
Plasterers and brick-		Sole sewer	1	Watereress seller ..	1
layers	35	Solicitors	2	Weavers	5
Platelayers	5	Stationer	1	Wharfinger	1
Plumbers	35	Stevadores	10	Wheelwright	1
Policemen	6	Stewards	2	Whitesmith	1
Porters..	125	Stick makers	7	Wine coopers	2
Portmanteau makers ..	2	Stockbroker	1	Wine porter	1
Post-office sorter	1	Stokers..	5	Wood carver	1
Postmen	4	Stonemasons	4	Wood cutter	1
Potato salesman	1	Stoveman (drying lead)	1	Wood turner	1
Potboy	1	Students of medicine ..	4		
Potmen	3	Sugar refiner	1		

OCCUPATIONS OF FEMALE PATIENTS.

Actress	1	Envelope folder	1	Pipe makers	2
Alum-maker	1	Factory hands	6	Porter	1
Artificial-flower-makers	11	Farm labourers	2	Pupil teachers	2
Bagmaker	1	Feather sorter	1	Sackmaker	1
Ballet girls	2	Fishwife	1	Schoolmistresses	9
Barmaids	11	Flower sellers	2	Seedbag maker	1
Bead trimmers	3	Furriers	3	Servants	304
Bed manufacturer	1	Glover	1	Shirt dresser	1
Bookbinders	4	Governesses	3	Shoebinder	1
Bookfolders	4	Harlots	262	Shopwomen	18
Bookkeeper	1	Hawkers	16	Silk weavers	4
Bootfitters	2	Hospital nurses	28	Singer	1
Bootmakers	4	Housekeepers	4	Stationery operative	1
Boot translator	1	Housewives	687	Stationer	1
Bottlewasher	1	Jeweller	1	Straw workers	2
Box makers	10	Label maker	1	Tailoresses	5
Brass worker	1	Laundresses	59	Tassel maker	1
Burnishers	2	Machinists	19	Tie makers	4
Cabinetmaker	1	Match maker	1	Tinbox maker	1
Chair caner	1	Metal piercer	1	Turner	1
Charwomen	47	Milliners	10	Umbrella makers	2
Chignon maker	1	Mine girl	1	Valentine maker	1
Cigarette maker	1	Muff stuffer	1	Waistcoat makers	4
Collar makers	5	Needlewomen	25	Watchmaker	1
Cooks	21	Nurses	15	Weaver	1
Corset makers	3	Operative	1	Wood chopper	1
Crinoline makers	2	Paper colourer	1		
Dancing mistress	1	Photographer	1		

MEDICAL REPORT.

TABLE I,

Showing the Total Number of Cases of each Disease under Treatment during the Year 1875, with the Results.

(The numbers after the names of the Diseases refer to the Appendix at the end of the Table.)

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
GENERAL DISEASES, A.										
Chicken pox	3	..	3
Measles	6	5	6	4	1
Scarlet Fever (1)	18	32	14	28	3	1	1	3
Ditto (2) <i>Sequelæ</i> of	17	14	11	12	6	2
Typhus	1	3	1	2	1
Enteric Fever (3)	32	14	26	11	2	1	4	2
Relapsing Fever (4)	1	..	1
Simple Continued Fever	3	4	2	3	1	1
Febricula	3	12	3	12
Ague	5	..	4	1	..
<i>Choleraic Diarrhœa</i>	1	..	1
Diphtheria (5)	8	4	3	4	5
Hooping-cough	2	2	1	2	1
Mumps	1
Erysipelas of the Face	17	12	13	12	2	..	2	..
Pyæmia	1	3	..	2	1	1
GENERAL DISEASES, B.										
Acute Rheumatism (6)	83	106	68	91	1	3	14	12
Ditto, with <i>Pericarditis</i>	24	15	20	13	1	..	3	2
Ditto, with <i>Endocarditis</i>	11	21	9	19	1	1	1	1
Ditto, with <i>Lung Complications</i>	10	8	8	8	2	..
Subacute Rheumatism (7)	9	9	9	9
Gonorrhœal Rheumatism	10	..	10
Synovial Rheumatism	5	5	4	5	1	..
Muscular Rheumatism	1	1	1	1
<i>Lumbago</i>	2	2	2	2
Chronic Rheumatism.. .. .	37	71	35	61	..	7	2	3
Acute Gout	4	2	4	2
Chronic Gout (8)	3	..	1	2	..
Chronic Osteo-arthritis	4	2	4	2
Cancer (9)	18	33	3	15	1	7	14	7	..	4
Scrofula	1	1
<i>Tubercular Meningitis</i>	5	2	5	2
<i>Phthisis Pulmonalis</i> (10)	74	62	37	32	10	4	25	23	2	3
<i>Acute Miliary Tuberculosis</i>	6	4	5	4	1	..
<i>Tubercular Peritonitis</i> (11)	2	2
Rickets.. .. .	1	1	1	1

TABLE I (continued).

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
GENERAL DISEASES, B (continued).										
Diabetes ⁽¹²⁾	5	3	3	2	1	1	1	..
Purpura ⁽¹³⁾	5	2	4	1	1	1
Anæmia	2	9	2	7	2
Chlorosis	3	..	3
General Dropsy	4	2	4	1	..	1
DISEASES OF THE NERVOUS SYSTEM.										
Subacute Meningitis ⁽¹⁴⁾	8	..	3	..	1	..	2	..	2
Cerebral Softening	1	1	1	1
Sanguineous Apoplexy	5	..	1	4
Sunstroke	2	..	1	..	1
Lightning Stroke ⁽¹⁵⁾	1	1
Chronic Hydrocephalus ⁽¹⁶⁾	1	1
Cerebral Tumours	1	1	1	1	..
Cerebral Disease	5	1	2	1	3
Disease of Spinal Cord	4	4	2	4	1	..	1
Paralysis ⁽¹⁷⁾	9	10	5	5	4	3	2
<i>Hemiplegia</i> ⁽¹⁸⁾ — <i>Right side</i>	7	7	4	4	1	2	1	..	1	1
<i>Ditto</i> , <i>Left side</i>	10	9	5	5	2	3	3	1
<i>Paraplegia</i> ⁽¹⁹⁾	8	5	5	1	3	2	..	1	..	1
<i>Locomotor Ataxy</i>	7	..	3	..	4
<i>Infantile Paralysis</i>	3	5	2	3	1	2
Tetanus ⁽²⁰⁾	1	..	1
Convulsions	8	5	7	5	1
Epilepsy ⁽²¹⁾	16	4	11	3	1	1	2	..	2	..
<i>Vertigo</i>	1	..	1
Chorea ⁽²²⁾	10	27	9	26	1	1
Hysteria	33	..	24	..	7	2
Neuralgia ⁽²³⁾	11	9	9	9	2
<i>Sciatica</i>	3	2	3	2
Hypochondriasis	2	..	2
Mania	3	2	3	2
Dementia	4	4
Imbecility	1	1
Hydrophobia	1	1
DISEASES OF THE CIRCULATORY SYSTEM.										
Pericarditis	3	4	1	2	2	2
Valvular Disease	59	81	23	40	8	9	22	30	6	2
<i>Aortic Disease</i> ⁽²⁴⁾	18	12	6	6	2	1	8	5	2	..
<i>Mitral Disease</i> ⁽²⁵⁾	21	36	10	22	2	2	6	11	3	1
Cardiac Dilatation	1	1
Fatty Heart	1	1

TABLE I (continued).

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE CIRCULATORY SYSTEM—continued.										
Angina Pectoris	1	..	1
Aneurism of Aorta	13	1	3	1	2	..	6	..	2	..
Phlebitis	1	..	1
Phlegmasia Dolens	1	..	1
Obstruction to Veins ⁽²⁶⁾	1	1
Goitre (Exophthalmic)	1	..	1
Addison's Disease	1	1
Arterio-Capillary Fibrosis	1	..	1
Cyanosis	1	1
Syncope	1	..	1
DISEASES OF RESPIRATORY SYSTEM.										
Œdema Glottidis	1	1
Croup ⁽²⁷⁾	5	2	2	1	3	1
Laryngitis ⁽²⁸⁾	3	6	2	5	1	1
Laryngeal Catarrh	1	3	1	2	1
Bronchial Catarrh	10	6	10	5	1
Acute Bronchitis	5	2	3	2	1	..	1	..
Chronic ditto, with <i>Emphysema</i> ⁽²⁹⁾ ..	53	40	30	23	2	..	18	14	3	3
Asthma	2	..	2
Pneumonia ⁽³⁰⁾	55	29	32	26	2	1	17	2	4	..
Ditto, <i>Right side</i>	22	10	13	10	6	..	3	..
Ditto, <i>Left side</i>	18	16	10	14	..	1	7	..	1	1
Ditto, <i>Double</i>	9	3	6	2	3	1
Ditto, <i>Lobular</i>	5	..	3	1	..	1	..
Pleuro-Pneumonia— <i>Right side</i>	12	3	9	1	2	2	1	..
Ditto, <i>Left side</i>	16	3	12	3	3	..	1	..
Ditto, <i>Double</i>	3	1	1	2	1
Gangrene of Lung ⁽³¹⁾	4	4
Congestion of Lungs	1	..	1
<i>Hæmoptysis</i>	4	2	2	2	2	..
Pneumonic Phthisis	1	..	1
Pleurisy ⁽³²⁾ — <i>Right side</i>	17	8	14	7	2	..	1	1
Ditto, <i>Left side</i>	21	9	19	7	..	1	2	1
Ditto, <i>Double</i>	5	3	3	2	2	1
Empyema ⁽³³⁾	7	1	3	..	1	..	3	1
DISEASES OF DIGESTIVE SYSTEM.										
Stomatitis	2	..	2
Cancerum Oris	1	1
Stricture of Œsophagus	1	..	1
Chronic Gastritis	1
Gastralgia	2	2	2	2

TABLE I (continued).

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF DIGESTIVE SYSTEM— <i>continued.</i>										
Gastric Ulcer	1	11	1	10	1
<i>Hæmatemesis</i>	3	5	3	4	1
Dyspepsia	5	7	4	3	..	1	1	..
Vomiting	4	5	4	5
Enteritis	1	..	1
Peri-Typhlitis	1	1	..
Dysentery	4	1	3	1	1
Intestinal Ulceration	1	2	1	2
Stricture of Bowel	1	..	1
Obstruction of the Bowel	3	1	1	1	2
Diarrhœa and Vomiting	1	5	..	5	1
Diarrhœa	5	4	5	4
Colic and Constipation	5	13	4	12	1	1
Abscess of Liver ⁽³⁴⁾	2	2	1	1	1	1
Enlarged Liver	5	4	3	2	1	1	1	1
Cirrhosis of Liver	28	9	6	4	1	1	18	4	3	..
Hepatitis	1	..	1
Lardaceous Liver	1	1
Hydatid Disease ⁽³⁵⁾	2	1	1	..	1	1
Jaundice	8	12	6	9	1	2	1	1
Abdominal Tumour ⁽³⁶⁾	4	13	1	6	2	6	..	1	1	..
Enlarged Spleen	1	3	..	1	..	1	1	1
Leuk-æmia	4	..	3	..	1
Peritonitis	5	9	1	2	1	1	3	6
Ascites	5	5	2	3	1	1	2	1
DISEASES OF URINARY ORGANS.										
Vesical Irritation	1	..	1
Nephritis, Acute ⁽³⁷⁾	11	4	9	2	1	2	1	..
Ditto, Chronic ⁽³⁸⁾	50	28	25	13	3	1	19	14	3	..
Pyelitis	2	1	1	1	1	..
Calculus in Kidney	1	1
<i>Hæmaturia</i>	7	..	6	1
Ditto, Intermittent	1	..	1
Renal Tumour	1	1
Retention of Urine	1	..	1
Incontinence of ditto	2	..	2
Cystitis	1	..	1
DISEASES OF THE FEMALE ORGANS OF GENERATION.										
Ovarian Disease ⁽³⁹⁾	13	..	6	..	6	1
<i>Pelvic Cellulitis</i> ⁽⁴⁰⁾	16	..	11	1	..	4
Pelvic Abscess ⁽⁴¹⁾	4	..	2	2

TABLE I (continued).

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE FEMALE ORGANS OF GENERATION— <i>continued.</i>										
Pelvic Peritonitis ⁽⁴²⁾	7	6	1
Pelvic Hæmatocele	11	9	2
Leucorrhœa	2	1	..	1	..	1
Chronic Metritis ⁽⁴³⁾	12	10	2
<i>Elongation of Cervix</i>	2	2
Granular Cervix	3	3
Stricture of Os Uteri ⁽⁴⁴⁾	4	3	1
Uterine Fibrous Tumour ⁽⁴⁵⁾	25	16	..	7	..	1	..	1	..	1
<i>Anteversio</i>	1	1
<i>Retroversio</i> ⁽⁴⁶⁾	14	12	2
<i>Anteflexio</i>	5	5
<i>Retroflexio</i>	2	2
<i>Subinvolution</i>	7	5	2
Prolapsus Uteri	1	1
Tumour of Meatus	1	1
Menorrhagia ⁽⁴⁷⁾	2	2
Dysmenorrhœa	2	1	..	1	..	1
Vaginismus	1	1
Congestion of Uterus	6	6
Abortion	1	1
Arrested Development of Uterus	1	1	..	1
Premature Birth ⁽⁴⁸⁾	1	1
Cyst of Nympha ⁽⁴⁹⁾	1	1
Fibro-Cystic Disease of Cervix ⁽⁵⁰⁾	1	1
Œdema of Pudenda	1	1
Progressive Muscular Atrophy	2	1	1	1	1
Obesity	1	..	1
Marasmus	2	..	2
SKIN DISEASES.										
Elephantiasis Græcorum ⁽⁵¹⁾	1	1
<i>Erythema Nodosum</i>	1	5	1	5
Urticaria	1	5	1	5
<i>Lichen Scrofulosus</i>	1	..	1
Psoriasis	4	..	4
Pemphigus	1	2	1	2
<i>Eczema Chronicum</i> ⁽⁵²⁾	6	9	6	9
<i>Ditto, Acutum</i>	1	1	1	1
Impetigo	1	..	1
Ecthyma	1	..	1
Xeroderma	1	1
Molluscum	1	..	1
Scabies	1	..	1
Pulex Penetrans ⁽⁵³⁾	1	..	1
Tinea Favosa	2	..	1	1	..

TABLE I (continued).

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
POISONS.										
Oxalic Acid ⁽⁵⁴⁾	1	..	1
Lead ⁽⁵⁵⁾	15	..	15
<i>Lead Colic</i>	14	..	14
<i>Ditto Palsy</i>	1	..	1
Ammonia	1	..	1
Sulphuric Acid	1	1
Hydrochloric Acid ⁽⁵⁶⁾	1	1
Carbolic Acid	1	..	1
Arsenic.	1	1
Alcohol— <i>Delirium Tremens</i>	18	4	16	4	2
<i>Ditto Inebriety</i>	5	2	5	2
<i>Ditto Mania a Potu</i>	1	1	1	1
Tin Chloride	1	..	1
Debility	18	34	13	32	2	3	2
Unclassified ⁽⁵⁷⁾	1	5	..	2	1	3

ABSTRACT OF TABLE I.

	Total number of cases under treatment during the year.		Number of medical cases discharged cured and relieved.		Discharged unrelieved.		Died.		Remaining in the hospital at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
GENERAL DISEASES A	118	106	89	93	21	5	8	8
Do. B	280	320	192	235	11	20	54	41	23	24
DISEASES OF THE—	115	143	72	96	27	29	11	9	5	9
Nervous System	29	47	14	9	31	32	8	2
Circulatory System	82	90	144	87	5	4	64	24	13	6
Glands	226	121	54	84	9	14	32	18	8	4
Respiratory System	103	120	42	20	3	2	22	17	5	..
Digestive System	72	89	..	110	..	16	..	6	..	15
Urinary System	..	147	13	30	1	1	1
Female Organs	15	31	13	30	2	3	2
Skin	18	34	13	32	1	2
Debility	58	10	54	7	1	..	3	2	1	1
Poisons, &c.
Unclassified	45	40	38	16	3	1	1	3	3	20
	1132	1201	740	857	76	95	239	157	77	92
	2333		1597		171		396		169	

APPENDIX TO TABLE I.

1. *Scarlet Fever*.—In one female case which was relieved, the scarlatina was complicated with rheumatic fever and pericarditis. In another case pus appeared in the urine on the 31st day, after albuminuria.

2. *Sequelæ of Scarlet Fever*.—In two female cases which were relieved, albuminuria was followed by pericardial and pleural effusions. Both these patients were 8 years old. A male patient, aged 3, had a bubo in the parotid region; another, aged 9, had an abscess in the left side of the thorax. Among the fatal cases were one in which pneumonia and two in which pleuropneumonia was present.

3. *Enteric Fever*.—Seven boys were admitted from the training ship Cornwall. The cases offered some interesting varieties; three had genuine typhoid with rose spots, characteristic stools, and characteristic temperature. Three had no spots, obstinate constipation, and a temperature which was not unlike that of typhoid. One boy who had the above symptoms for 11 days, and went out apparently cured, had a relapse which lasted 14 days. In this the temperature was higher than in the first attack, but there were no spots or diarrhœa. One boy had no typhoid symptoms but had acute pneumonia.

4. *Relapsing Fever*.—The patient was a male aged 24. The fever was dependant on constitutional syphilis, for there was no chance of infection from relapsing fever of the usual kind. On admission the temperature was 105.2, pulse 132; on the same day the fever ended with a critical sweat. During the paroxysm the spleen became swollen. About a week later another exacerbation occurred, the temperature reached 100.8, the next day a defervescence. Twelve days later a more violent relapse followed; temperature 105, pulse 156, respirations 32: the spleen very large. Next day an entire remission, the next day a relapse, and the next a remission. This irregular course was maintained for 4 months, when the fever terminated with the most violent attack of all, the temperature reaching 106.3. There was a history of syphilis, a well marked syphilide and sore throat appeared and remained after discharge from the Hospital. The disease was treated first unsuccessfully with quinine, and then successfully with mercury and iodide of potassium. The recurrence of the fever and its peculiar characters were thought to be due to syphilitic changes in the spleen. (Hospital Reports, vol. xi. 1875, p. 265.)

5. *Diphtheria*.—In two fatal cases tracheotomy was performed.

6. *Rheumatic Fever*.—Among the cases complicated with endocarditis was one with a double mitral and a double aortic murmur; one with a double mitral and a persistent systolic murmur at the left base; and one with a systolic murmur at apex and base. In 6 cases endocarditis was complicated with pericarditis. Of those with lung complications one was a case of double pneumonia, one a case of single (left), and one a case of double pleurisy; one was a case of pleurisy and pericarditis, two were cases of pneumonia and pericarditis; one was a case of endocarditis, pericarditis, and pleurisy (double); one a case of endocarditis, pericarditis, and pneumonia (double); one a case of pleuropneumonia (double), endocarditis and pericarditis; one a case of pleurisy, pneumonia and endocarditis. In one case of rheumatic fever there was also albuminuria, in another tonsillitis, in another otitis interna, in another pelvic cellulitis and pelvic peritonitis, in another scarlatina was contracted in the Hospital.

7. *Subacute Rheumatism*.—One case arose after prolonged lactation; another was complicated with erythema circinatum, and another with atrophy of the choroid.

8. *Gout*.—One case (a female aged 41) had an aortic murmur.

9. *Cancer*.—Of the fatal cases seven were cancer of the liver, four males and three females, the youngest 34, the oldest 78. In six cases cancer of the liver was complicated with cancer of other organs; in two males, aged 42, with cancer of the stomach; in one male, aged 48, and one female, aged 53, with cancer of the

pancreas; in one female, aged 41, with cancer of the lungs; in one male, aged 38, with cancer of the stomach, pancreas, gall bladder, and omentum. One male, aged 53, died of cancer of the cardiac end of the stomach. Four males, of ages varying from 34 to 60, died of cancer of the intestines. There was also one fatal case of cancer of the lungs (a female, aged 28); one of the ribs and sternum (a male, aged 48); one of the thyroid body (a male aged 39); these two last cases will be described at length subsequently; one of the bladder (a male aged 54). There was one fatal case of cancer of the uterus in a woman aged 37. In another case the uterus and vagina were largely affected; the patient was in the fifth month of pregnancy, premature labour came on, craniotomy was performed, but death followed from peritonitis. There were two cases of epithelioma of the cervix uteri, in which amputation of the cervix was performed with the *écraseur*, and the patients left the Hospital relieved.

The case of cancer of the thyroid body, abovementioned, is described at length in the Hospital Reports, p. 265. The enlargement of the neck was noticed only three weeks before admission. There was no pain, but there was *dyspnœa*, a short spasmodic cough with a frothy sputum, and *dysphagia* began five months before admission, and on admission was almost absolute for solids. There was no general enlargement of the glands of the body. The resonance of the apices of the lungs was impaired. There was no leucocythæmia. There was some albuminuria. Eventually the legs swelled; there was nocturnal delirium, and there was absolute *dysphagia* and great *dyspnœa* for 40 hours before death, a month after admission, or six months after *dysphagia* was noticed. The enlargement of both sides of the neck was great, and the cancerous mass involved both lobes of the thyroid, all the cervical, submaxillary, supra- and infra-clavicular and thoracic lymphatics on both sides. The left common carotid was somewhat narrowed by the growth; the trachea, larynx, and vagi nerves showed no signs of disease though imbedded in the mass. The lower half of the œsophagus was impervious and was ulcerating. The liver contained five nodules of cancer; the kidneys were cirrhotic, the other organs healthy.

The other case mentioned in the Hospital Reports, p. 268, is that of osteo-arthritis, with osteoid cancer of the sternum, ribs, right clavicle, glands of the neck, mediastinum and liver, causing fracture of the sternum and right clavicle. The osteo-arthritis was of 12 years' standing, and began in the right knee, but many other parts became affected. The thyroid body, cervical and inguinal glands were hard. Pulsation was felt in the sternal tumour, synchronous with the systole; the tumour was fairly resonant, and the heart sounds were heard over it. There was a systolic murmur at the apex, not heard at the base. Death ensued from pneumonia of the base of the right lung. There were some yellowish-white granules, probably cancerous, in the lungs and liver. The mediastinal glands were affected. Death occurred about five months after the sternal swelling was first noticed.

10. *Phthisis*.—One fatal case had also nephritis and anasarca, another had pneumothorax.

11. *Chronic Tubercular Peritonitis*.—One fatal case; a boy, aged 6, had a discharge from the navel.

12. *Diabetes*.—The fatal male case is described in the Hospital Reports, p. 262. The age was 17. The disease began six weeks before admission, and some seven months before death. The appetite was very ravenous. The chest was natural, the spleen very large, and could be felt some two inches below the ribs. He passed about four pints of urine in 24 hours, sp. gr. 1044. He improved for a month and was then discharged for eating prohibited things, but was readmitted three months later much worse; his appetite was very bad, and he vomited his food. He passed 2½ pints of urine in 24 hours, sp. gr. 1035. The liver was felt slightly below the ribs, but the spleen was no longer enlarged. He died comatose. After death the liver was found enlarged and deeply congested; the spleen congested but not enlarged; the brain tough and pale; no other morbid appearances. There was never any suppression of urine; he passed 5 pints on the day before his death. The transitory enlargement of the spleen is noteworthy.

The female fatal case was also described in the Hospital Reports, p. 263. The patient was 19 years old, a married woman. The disease began three months before admission, or about 6 weeks before death. There was a history of great mental anxiety, also of drinking large quantities of cold water. No family history of consumption or diabetes. The pupils were dilated and several teeth had recently

decayed. The bowels were confined. No pulmonary signs. The appetite gradually diminished, and the bowels became very irregular. Death resulted from coma; the change for the worse occurring only a few hours before death. There was no suppression of urine. The spleen, liver, pancreas and kidneys (otherwise healthy) were congested.

13. *Purpura*.—The female fatal case had a double aortic murmur and an enlarged spleen. The male fatal case (aged 21) is described in the Hospital Reports, p. 65. The patient was a wine cellarman. It was his first attack. For four weeks before admission he had suffered from beating of the heart, rigors and loss of flesh. He had no hæmatemesis. There were large effusions round each optic disk and in both retinae. There was occasional hæmoptysis. Of the petechiæ none were hard; the skin became bruised by slight pinching. The gums oozed blood towards the end, and there was continuous headache for the last five days. After death much serum was found in the lateral ventricles of the brain, and their ependyma was studded with small ecchymoses which were abundant in the choroid plexuses, velum interpositum, soft commissure, corpora striata, optic thalami and posterior wall of the ventricle. The surface of the brain had hardly any ecchymoses. There were abundant meningeal hæmorrhages on the posterior lobes of the brain covering the cerebellum. The thymus was persistent and the foramen ovale open. (Dr. Legg cites Virchow, Canstatt's Jahresbericht 1859, Band iv, p. 267, and Schönlein, Vorlesungen über Pathologie und Therapie, 3 Auflage 1837. Band 2, p. 63.) There was a bloody fluid in the pleuræ and pericardium, and there were many ecchymoses on the visceral pericardium. The lungs were natural, the spleen was very small, the Malpighian bodies distinct. Both kidneys had ecchymoses in the pelvis. The stomach showed a few ecchymoses, the large intestine contained blood.

14. *Meningitis*.—One case in which there was blindness of both eyes, was due to congenital syphilis.

15. *Lightning Stroke*.—This case had optic neuritis.

16. *Hydrocephalus*.—A chronic case, was twice tapped.

17. *Paralysis*.—In a case of spinal paralysis in a female aged 30, there were symptoms of chronic lead poisoning, there was also mitral regurgitation.

18. *Hemiplegia*.—In one case of left, and one of right, hemiplegia there was a præ-systolic mitral murmur. In one of right hemiplegia there was aphasia and agraphia, and in another (left) there was slight aphasia.

19. *Paraplegia*.—In one female case paraplegia followed right hemiplegia. In another (male) there was also left facial palsy. Another case (male) was due to constitutional syphilis. The fatal case had also schirrhus of the mamma.

20. *Tetanus*.—A case of idiopathic tetanus, described in the Hospital Reports, p. 251. The patient was a ticket collector on a railway, and had been wet through. Two days later he had aching pains in the back, and stiffness in the neck. He could not stand or open his mouth, and swallowing was difficult. He had spasms of the back and legs at first two or three times a day with great pain. The tongue could not be protruded, the voice was interrupted with each inspiration. Defecation was very painful, micturition easy. There was no paralysis or impairment of sensation. Not even a scratch could be found anywhere. The spasms at first became more violent, but eventually subsided some six weeks after the commencement of the attack.

21. *Epilepsy*.—In a fatal case with frequently repeated convulsions, which ended in death a few hours after its commencement, disease of the cranial bones was found.

22. *Chorea*.—In one case (a female aged 16) there was also left hemiplegia; in another, the attack was said to have begun on four occasions on July 20.

23. *Neuralgia*.—In one case of intercostal neuralgia the left mamma had been removed thirteen months previously for a hard tumour.

24. *Morbus Cordis, Aortic*.—In one case of systolic aortic murmur there was also a double mitral murmur; in another, in addition to a double mitral murmur there was also a tricuspid systolic murmur. Two aortic murmurs were double. Five had also a mitral murmur, in four of these both murmurs were regurgitant; in one an

aortic regurgitant murmur was associated with a presystolic murmur at the apex. In one aortic case there was angina pectoris.

25. *Mitral*.—In four cases the mitral murmur was double; in one of these there was double pleural effusion. In three cases a mitral murmur was associated with a tricuspid murmur; two of these were diagnosed as mitral stenosis and tricuspid regurgitation. In one case of mitral disease there was hemiplegia, in another tertiary syphilis, in another epilepsy, and another contracted scarlatina in the Hospital.

Among four fatal cases in which mitral incompetence was present, one had adherent pericardium, one had also cirrhosis of the liver, one had mitral and aortic obstruction, and one had acute nephritis. Another had an infarct in the lungs, and another in the brain, followed by softening, also in the kidneys and spleen.

26. *Obstruction to Veins*.—In the vena cava superior.

27. *Croup*.—Two of the fatal cases of croup, males, aged two and one respectively, had tracheotomy performed, death taking place seven and two days after the operations.

28. *Laryngitis*.—One female case was syphilitic.

29. *Chronic Bronchitis*.—Four cases of chronic bronchitis were complicated with phthisis.

In a fatal case (a female aged 49) albuminuria was present.

30. *Pneumonia*.—One case (which recovered) occurred after child-birth, and was thought to be embolic. In another the crisis occurred on the 3rd day.

31. *Gangrene of Lung*.—In one of the fatal cases (a male, aged 34) there was also pleurisy and nephritis.

32. *Pleurisy*.—One case had also albuminuria.

In a fatal case (a male, aged 21) there was also pneumothorax and tuberculosis.

33. *Empyema*.—In one case the empyema pointed, and discharged itself, but the patient died.

In one case which recovered, the empyema was encysted, and discharged itself through the lung.

34. *Abscess of Liver*.—This discharged itself through the lung.

35. *Hydatid of Liver*.—The fatal case (a female, aged 23) had jaundice, bile in the urine, white stools, and xanthops. Her temperature reached 104; the right hypochondrium was full; there was dulness up to the third rib, the liver could be felt down to the navel, and was very tender. She also had persistent shoulder-tip pain. After a time all the symptoms connected with the jaundice abated, but the shoulder-tip pain was constant and the temperature remained high. Some six weeks after admission the resonance was impaired at the base of the *left* lung, with largish râles, and that part of the chest became tender. On the day of her death, some two months after admission, she had a severe and sudden epigastric pain and vomited greenish fluid. After death the right pleura showed signs of recent pleurisy, especially at the base, and the fluid was yellow and turbid; the *left* pleura had a firm adhesion to the diaphragm near the pericardium, on cutting through which pus escaped from below the diaphragm; no opening into the right pleura could be found. Much puriform fluid was found in the pericardium. The *left* lobe of the liver reached nearly to the navel, the right lobe was almost natural. Firm adhesions held the liver to the diaphragm, the liver could not be dissected from it without opening an abscess as large as an orange. No opening into it could be found. The common bile duct was much dilated but showed no obstruction, nor did the cystic duct. Both branches of the hepatic duct were dilated. Near the vena cava the surface of the liver was puckered over a spot about as large as a crown piece, towards which a large branch of the hepatic duct led, opening into a cavity as large as a walnut beneath the puckering; the walls of the cavity were tough, hard, and in places calcified, and the cavity looked like a hydatid cyst. The temporary jaundice and dilatation of the duct were probably due to the passage of the hydatid membrane down the duct. The cause of the suppuration in the liver was not plain. Dr. Legg

cites Murchison, 'Clinical Lectures on Diseases of the Liver,' 1868, p. 94 (Hospital Reports, p. 91).

36. *Abdominal Tumour*.—In one female case, which recovered, the tumour was probably renal. Pus appeared in the urine and was also drawn by tapping from the tumour. The patient refused further operation.

37. *Acute Nephritis*.—One case, which recovered, had also pneumonia, and another pleurisy. One of the fatal cases (a female) also had pneumonia.

38. *Chronic Nephritis*.—One fatal case had also cirrhosis of the liver.

39. *Ovarian Tumours*.—Of those not operated on, two were tapped. One burst into the peritoneal sac and was absorbed.

40. *Pelvic Cellulitis*.—One case which recovered was complicated with pelvic peritonitis and hæmatocele, another with albuminuria.

41. *Pelvic Abscess*.—One of the fatal cases (a female, aged 35) had also general peritonitis, nephritis, and phthisis.

42. *Pelvic Peritonitis*.—One case, which recovered, was complicated by uterine fibroid. In another case the peritonitis was circumscribed, and lay between bladder and uterus. In another case there was also albuminuria, with ascites and anasarca, and in another enteritis, oöphoritis and perityphlitis.

43. *Chronic Metritis*.—One case was due to retained decidua.

44. *Stricture of Os Uteri*.—In one case the cervix was divided with Küchenmeister's scissors, and the dysmenorrhæa cured. Another case was complicated with antelexion.

45. *Uterine Fibroid*.—In one case the sloughing of a fibroid polypus was accompanied by pelvic cellulitis.

46. *Retroversion*.—Complicated in one case with hæmatocele, in another with vaginismus.

47. *Menorrhagia*.—In one case, suffering principally from menorrhagia, there was a general hypertrophy of the subcutaneous cellular tissue, especially of the legs.

48. *Premature Birth*.—A patient with advanced phthisis gave birth to a child in the seventh month, which only survived four hours. After delivery the phthisis rapidly killed the mother.

49. *Cyst of Nympha*.—A sebaceous cyst of the left nympha was removed by the wire écraseur on the day of admission.

50. *Fibro-cystic Disease of the Cervix Uteri*.—Removed by the écraseur.

51. *Elephantiasis Græcorum*.—A native of Calcutta, aged 20, re-admitted. (Hosp. Rep., vol. x., 1874, p. 279). Improved last year by good diet and gurjun oil.

52. *Eczema*.—One case, contracted in the Hospital, scarlatina, followed by erysipelas and an abscess in the arm.

53. *Pulex Penetrans* in the feet.

54. *Poisoning with Oxalic Acid*.—This patient was mad, and was removed to a lunatic asylum.

55. *Lead Poisoning*.—All the patients were males. There were four painters; two plumbers; two envelope-makers; two engaged in a lead factory as "stove-men" (who dry the white lead); two were labourers; one was a cooper; one a paper-stainer; one an excavator.

56. *Poisoning with Hydrochloric Acid*.—This was a suicide. The patient took arsenic also. Death ensued three hours after admission.

57. *Unclassified*.—These include, among those who recovered, one of "poly-sarcia"; two of "pain"; and one of malingering. The fatal cases include one in which, a post-mortem examination having been refused, no certificate was granted, and one of cystocele.

TABLE II,

Showing the comparative Frequency and Mortality of each Disease at different Ages.

DISEASES.	Under 5.		5-10.		10-15.		15-25.		25-35.		35-45.		45-55.		55-65.		65-75.		75 and upwards.		TOTAL.			
	Discharged.		Discharged.		Discharged.		Discharged.		Discharged.		Discharged.		Discharged.		Discharged.		Discharged.		Discharged.		Discharged.			
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Chickenpox	3	3
Mensles	5	2	6	4
Scarlet Fever	3	2	3	10	2	5	10	..	1	3	1	14	28	3	1
Ditto, Sequelæ of	1	2	3	10	8	3	..	1	..	1	11	12	6	2
Typhus	..	1	..	1	..	1	..	1	1	2	..	1
Enteric Fever	2	1	..	3	3	14	5	2	2	1	2	3	1	..
Continued Fever	3	12
Febricula	2	1	..	1	..	3	1	6	..	1	..	1	4
Ague	1	1
Relapsing Fever.	1	1
Choleric Diarrhœa	1	1
Diphtheria	2	..	1	2	..	2	1	..	1	1	..	1	3	4	5	..
Whooping-cough	1	1	..	2	1	2	1	..
Mumps	1	1	2	1	..
Erysipelas of Face	1	1	1	1	3	4	..	4	1	..	1	1	2	13	12	2	..
Pyæmia	1	1	..	1	68	91	1	3
Acute Rheumatism	3	7	..	3	7	32	51	1	24	14	2	5	13	68	91	1	3
Ditto, with Pericarditis	11	7	1	7	3	..	1	20	13	1	..
Ditto, with Endocarditis	2	..	1	3	..	5	13	1	..	1	9	19	1	1

Table showing the Average Stay of the Medical Cases in Hospital, &c.

Within—Weeks of admission.	No. of Patients discharged.		Died.		M. F.	Text
	M.	F.	M.	F.		
						The deaths which took place within one week of admission were on the days as shown to the right, No. 1 being the day of admission.
1	79	91	80	38	1 11 5	The total number of deaths during the year was . . . 392
2	149	156	46	27	2 17 1	Of these there occurred within 24 hours of admission . . . 17 = 4 per cent.
3	140	141	29	14	3 12 7	Within one week of admission 118 = 30 „
4	120	134	21	12	4 13 5	„ a fortnight „ 191 = 48 „
5	69	115	16	13	5 16 11	„ a month „ 267 = 68 „
6	41	65	6	13	6 4 4	The average time of fatal cases in Hospital was { Males, 21·9 } 27·72 in days { Females, 32·64 }
7	44	65	6	7	7 7 5	
8	32	38	5	4		The total number of discharges during the year was . . . 1,758
9	26	33	8	4		Of these the number whose stay in the Hospital did not exceed a fortnight was } 475 = 27 per cent.
10	16	11	1	5		Those whose stay did not exceed three weeks . . . 756 = 43 „
11	8	22	..	3		„ „ „ four „ . . . 1,010 = 57 „
12	8	10	5	2		„ „ „ six „ . . . 1,300 = 74 „
13	3	10	1	4		The average stay in Hospital of patients discharged was in days { Males, 26·45 } 27·02 { Females, 39·17 }
14	1	6	2	2		The average time of all the medical cases in the Hospital within the year 1875 was in days . . . { Males, 25·71 } 27·15 { Females, 27·88 }
15	2	2	1	1		
16	4	1	3	..		
17	1	4	1	2		
18	1	2		There are 230 beds in the medical wards, giving the number of possible pernottations for the year, 230 × 365, or 83,950; but of the ten medical wards five were wholly closed during a portion of the year. This would take away about 7,050 from the possible number of pernottations, leaving 76,900
19	1	..	1	..		
20	..	1		
21	..	2		
22	..	1	1	..		
23	1	1		The actual number of pernottations has been as follows:
24	1	2		Of cases admitted in 1874 or 1875 and discharged in 1875 47,509
25	1	1	..	1		Of cases remaining in at the end of the year 1875 . . . 5,415
26	1		Of fatal cases 10,867
27	..	2	..	1		<u>63,791</u>
29	..	1		Giving a difference of 13,109
30	..	1		This gives a nightly average of 36·13 empty beds—about 16 for males and 20 for females—and the time between successive occupants about 136 hours on an average, or about 5·65 days a month.
31	1	..		
40	1		

SURGICAL REPORT.

TABLE I,

Showing the total Number of Cases of each Disease under Treatment during the year 1875, with the Results.

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
GENERAL DISEASES.										
Phagedæna	3	1	2	1	1
Erysipelas—										
<i>a.</i> Simple	34	17	29	14	1	1	4	2
<i>b.</i> Phlegmonous	39	24	36	19	2	4	1	1
<i>c.</i> Diffuse Inflammation	2	1	1	1	1
Pyæmia	3	7	2	2	1	3	..	2
Syphilis—										
<i>A.</i> Primary Syphilis—										
Hard Chancre	29	14	27	13	2	1
Soft Chancre	53	39	47	34	1	2	5	3
Phagedænic Sore	14	5	12	5	2	..
<i>B.</i> Hereditary Syphilis	1	3	1	3
<i>c.</i> Secondary Syphilis—										
Local Syphilitic Affections—										
Brain	2	1	2	1
Tongue	4	..	4
Palate and Pharynx	1	13	1	12	1
Larynx	4	3	4	2	1
Rectum	1	3	1	2	1
Anus	6	42	6	41	..	1
Bone	4	3	2	3	2
Skin	70	82	61	71	2	3	7	8
Eye	7	4	7	3	1
Cancer—										
<i>A.</i> Scirrhus—										
Rectum	1	1
Female Breast	25	..	15	..	6	..	3	..	1
Lymphatic Glands	2	1	1	1	1	..
Sup. Maxilla	3	1	3	1
Pharynx	1	1
Scapula	1	1

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1876.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
GENERAL DISEASES (continued).										
B. Medullary Cancer—										
Female Breast	2	1	..	1	..	1
Testis	3	..	2	1
Glands	2	1	..	1
Spine	1	1
Femur	2	2
C. Epithelial Cancer—										
Lip	6	..	4	..	2
Tongue	14	2	6	2	5	..	2	..	1	..
Mouth	3	1	1	1	2
Face	1	1	1	1
Sup. Maxilla	1	1
Leg	3	..	2	1	..
Serotum	6	..	4	..	1	..	1
Rectum	2	..	1	..	1
D. Colloid—										
Breast	1	..	1
Lupus	2	3	1	3	1
Serofula										
Lymphatic Glands ..	3	3	3	3
Testicle	9	..	7	..	1	1	..
Bone	2	1	2	1
Joints	11	3	9	1	1	..	1	1	1	1
Skin	3	..	2	..	1
DISEASES OF THE NERVOUS SYSTEM.										
Malingering	1	..	1
Neuralgia	1	..	1
Hysteria	1	3	..	3	1	..
Tetanus, Traumatic ..	1	1
Spinal Meningitis	1	1
Radial Paralysis ..	1	1
DISEASES OF THE EYE.										
A. Conjunctiva—										
Catarrhal Ophthalmia ..	5	1	5	1
Pustular	1	..	1
Purulent	1	2	1	2
Strumous	2	1	2	1
Gonorrhœal	1	1	1	1

TABLE I (continued).

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1873.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE EYE (continued).										
Rheumatic Ophthalmia ..	2	..	2
Neonatorum	1	..	1
Trachoma	3	..	3
Lippitudo	2	..	2
B. Cornea—										
Keratitis	6	10	6	8	2
Do. Interstitial ..	2	8	1	7	1	1
Hypopyon	2	3	2	3
Ulcers	7	5	7	4	1
Opacity	5	5	4	5	1	..
Staphyloma	2	2	2	2
Corneo-Cyclitis ..	1	..	1
C. Iris—										
Iritis	2	4	2	4
Rheumatic Iritis ..	3	1	3	1
Irido-choroiditis ..	1	1	1	1
Synechia	6	5	6	5
D. Crystalline Lens—										
Cataract—										
Hard	10	3	9	2	..	1	1	..
Soft	3	1	2	1	1
Traumatic	5	1	5	1
Congenital	6	..	6
Dislocation of Lens ..	1	..	1
E. Diseases of Retina and Optic Nerve—										
Detached Retina	1	1
Retinitis Albuminurica ..	1	..	1
Optic Neuritis	3	3	2	2	1	1
Neuro Retinitis	3	1	2	1	1
Retino-Choroiditis	2	2	1	1	1	1
White Atrophy of Optic Discs	4	1	..	1	4
F. Diseases of the Choroid—										
Choroiditis	1	1	1	1
G. Hyphæma										
	2	..	2
H. General Affections of the Eye—										
Glaucoma	5	10	4	10	1
Sympathetic Ophthalmia ..	1	1	1	1
Total Disorganization of Eye	..	1	..	1
Melanotic Sarcoma	1	..	1

TABLE I (continued).

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE EYE (continued).										
I. Strabismus—										
Internal	12	8	12	8
External	3	2	2	1	1	1
J. Hypermetropia and Asthenopia 3 .. 2 .. 1										
K. Diseases of the Lachrymal Apparatus—										
Lachrymal Obstruction	6	..	6
Abscess and Fistula	2	3	2	3
Dacryo-Cystitis	1	1	1	1
L. Diseases of the Eyelids—										
Entropion	1	1	1	1
Distichiasis	2	..	2
Pannus	1	1	1	1
Symblepharon	1	1
M. Diseases of Orbit—										
Defective Cicatrix	1	1
Abscess in Frontal Sinus	1	..	1
N. Diseases of Cranial Nerves 1 2 1 2										
DISEASES OF THE EAR.										
Otorrhœa	2	1	2	1
Polypus	1	..	1
DISEASES OF THE NOSE.										
Sinus	1	1
Polypus	2	3	1	3	1	..
Epistaxis	4	5	2	5	1	..	1	..
DISEASES OF THE CIRCULATORY AND ABSORBENT SYSTEMS.										
Aneurism—										
Femoral Artery	1	..	1
Popliteal Artery	3	..	2	1
Varicose Veins	4	8	4	7	1

TABLE I (continued).

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE CIRCULATORY AND ABSORBENT SYSTEMS (continued).										
Thrombosis	4	1	4	1
Secondary Hæmorrhage	1	..	1
Angioma	1	..	1
Lymphatic Glands—										
Abscess	7	3	7	3
Enlarged Glands	3	..	3
Lymphangitis	8	1	7	1	1	..
DISEASES OF THE LIPS.										
Malformations—										
Single Harelip	4	5	4	3	..	1	1
Double „	4	..	4
DISEASES OF MOUTH AND CHEEK.										
Stomatitis	1	1	1	1
Ranula	1	..	1
Cancrum Oris	2	2	2	1	1
Congenital Orifice below Mouth	1	..	1
DISEASES OF GUMS AND JAWS.										
Necrosis	10	8	9	5	1	2	1
Epulis	1	4	1	4
Fibrous Tumours	1	1
Sarcoma	1	..	1
Osteoma	1	1
DISEASES OF PALATE AND FAUCES.										
Enlarged Thyroid	2	2	1	1	1	1
„ Tonsils	2	2	2	2
Tonsillitis	8	19	7	19	1
Cleft Palate	8	9	7	4	1	4	1
Naso-Pharyngeal Polypus	1	..	1
DISEASES OF THE TONGUE.										
Ulcer	1	1
Hypertrophy	1	1	1	1
Glossitis	1	..	1

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF SALIVARY GLANDS.										
Cyst	2	..	2
Parotid Glandular Tumour	1	..	1
Sarcoma	1	..	1
DISEASES OF ŒSOPHAGUS AND LARYNX.										
Stricture of Œsophagus	1	2	..	1	1	1
Laryngitis	1	1	..	1	1	..
DISEASES OF THE INTESTINES.										
Hernia—										
Umbilical	6	..	4	2
Inguinal	30	2	22	1	4	1	4	..
Femoral	4	24	3	11	1	10	..	3
Ventral	1	..	1
Internal Strangulation	2	2
Fæcal Fistula	1	..	1
Abscess of Abdomen	2	..	2
Tumour	1	1
DISEASES OF RECTUM AND ANUS.										
Fistula in Ano.. .. .	19	7	17	6	2	1
Hæmorrhoids	18	5	17	5	1	..
Fissure of Anus	1	4	1	4
Fibrous Stricture	5	..	3	..	2
Imperforate Anus	1	1	1	1
Prolapsus Ani.. .. .	5	2	5	2
Ulcer of Rectum	1	1
Ischio-Rectal Abscess	5	2	5	2
DISEASES OF URINARY SYSTEM.										
Cystitis—										
Chronic	6	..	4	2
Renal Calculus	3	1	..	2	..
Abscess about Kidney	1	1	1	1
Calculi Vesicæ—										
a. Uric Acid	11	1	9	1	2
b. Oxalate of Lime.. .. .	2	..	2
c. Phosphatic	1	..	1

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF URINARY SYSTEM (continued).										
Urethral Calculus	4	..	3	1	..
Irritable Bladder	9	..	8	..	1
<i>Diseases of Prostate Gland.</i>										
Enlarged Prostate	9	..	2	..	2	..	4	..	1	..
Calculus	1	1
<i>Gonorrhœa and its Complications.</i>										
Gonorrhœa	23	124	21	115	1	2	7
Phimosis	6	..	4	..	1	1	..
Paraphimosis	5	..	5
Bubo	17	24	15	22	1	1	2
Verruœ	5	14	4	13	1	1
Abscess in Labium	5	..	4	1
Perineal Abscess	5	..	5
Orchitis	22	..	19	..	2	1	..
<i>Diseases of Urethra.</i>										
Stricture—										
<i>a.</i> Organic	48	..	37	2	..	9	..
<i>b.</i> Inflammatory	6	..	5	1	..
<i>c.</i> Traumatic	1	1
<i>d.</i> Spasmodic	1	..	1
Urinary Fistula	13	..	10	..	1	2	..
Extravasation of Urine	16	..	11	5
Incontinence of Urine	2	..	2
Retention of Urine	4	..	4
<i>Diseases of the Penis and Testis.</i>										
Malformation—										
Phimosis	31	..	30	..	1
Paraphimosis	2	..	2
Hæmatocele	2	..	2
Hydrocele	16	..	14	..	2
Encysted Hydrocele	1	..	1
" " of Cord.. .. .	1	..	1
Sarcoma Testis	1	..	1
Enchondroma Testis	1	..	1
Varicocele	4	..	3	..	1
Inflamed Scrotum	3	..	3

TABLE I (continued).

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF FEMALE ORGANS OF GENERATION.										
Diseases of the Ovary	6	5	1
<i>Diseases of Round Ligament.</i>										
Cyst in Canal of Nuck	1	1
<i>Diseases of the Vagina.</i>										
Vesico-Vaginal Fistula	2	1	..	1	..	1
Recto-Vaginal Fistula	1	1
Congenital absence of Vagina and Uterus	1	1
<i>Diseases of Vulva.</i>										
Cystic Tumour of Labium	2	2	..	2
Noma	6	3	..	1	..	1	..	1	..	1
Fibrous Tumour	2	2
<i>Affections connected with Parturition.</i>										
Ruptured Perinæum	7	7
DISEASES OF THE FEMALE BREAST.										
Abscess	26	24	1	..	1
Chronic Eczema about Nipple	1	1
Non-Malignant Tumours—										
Cysts	4	4
Mammary Glandular	8	7	..	1
DISEASES OF THE ORGANS OF LOCOMOTION.										
<i>Diseases of Bones.</i>										
Ostitis	1	1	..
Periostitis	9	5	6	5	2	1	..
Chronic Abscess	2	1	1
Diffuse Periostitis—										
Acute Necrosis	3	1	2	1	1
Caries	23	19	19	8	3	1	2
Necrosis	36	13	30	9	3	2	3	2

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE ORGANS OF LOCOMOTION (continued).										
<i>Diseases of Bones (continued).</i>										
Tumours—										
<i>a.</i> Exostosis	4	5	3	4	1	1
<i>b.</i> Enchondroma	1	1	1	1
<i>c.</i> Sarcoma	4	1	2	1	1	..	1
Old Amputations	5	1	4	1	1	..
Deformities after Fracture, &c.	4	2	4	2
<i>Diseases of Joints.</i>										
Acute Synovitis	40	17	35	17	1	..	4	..
Chronic do.	79	69	54	47	6	7	4	1	15	14
Ulceration of Cartilage ..	11	8	8	5	1	..	2	3
Ankylosis (Fibrous).. .. .	3	7	1	3	..	2	2	2
Loose Cartilage	5	..	4	1	..
Knock-Knee	8	4	5	4	1	2	..
Weak Ankles	1	..	1
Bow Legs	1	1	..	1	1	..
Old Resections	3	..	3
<i>Diseases of the Spine.</i>										
Caries	2	3	1	3	1
Psoas, Lumbar, and other Abscesses	7	3	3	2	1	3	1
Angular Curvature	11	9	7	7	1	2	1	..	3	..
Lateral Curvature	4	2	3	2	1	..
Spina Bifida	2	1	2	1
<i>Diseases of Muscles, Tendons, &c.</i>										
Tenonitis	1	1
Contraction of Tendons, Fasciæ, or Muscles	5	2	5	2
Club-Foot—										
<i>a.</i> Talipes Equinus.. .. .	3	2	2	2	1	..
<i>b.</i> Do. Valgus	3	4	2	3	1	1
<i>c.</i> Do. Equino-varus	21	17	11	14	3	1	7	2
Wry-neck	1	..	1
Enlarged Bursa Patellæ	1	6	1	6
Inflammation and Suppuration of										
Bursa Patellæ	12	28	12	25	3
Do. do. of other Bursæ	2	1	1	1	1	..
Ganglion	1	7	1	5	..	1	1
Diffuse Palmar Ganglion	3	..	3

TABLE I (continued).

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE CELLULAR TISSUE.										
Abscess	107	44	95	41	1	..	5	..	6	3
Connective Tissue Tumours—										
<i>a.</i> Fatty	9	11	9	10	..	1
<i>b.</i> Fibro-cellular	3	..	2	..	1
<i>c.</i> Sarcoma	5	2	2	2	1	..	2
<i>d.</i> Fibrous	2	1	2	1
Sebaceous Cysts	9	10	8	9	..	1	1	..
Dermoid Cysts	1	1	1	1
Elephantiasis	2	2
DISEASES OF THE CUTANEOUS SYSTEM.										
Ulcer	31	31	20	24	..	1	..	1	11	5
Carbuncle	8	4	6	3	2	1
Whitlow—										
Thecal Abscess	2	3	2	3
Gangrene	5	3	2	3	3
Nævus	4	7	4	5	2
In-growing Toe-nail	2	7	2	6	1
Bed Sores	3	..	2	1
Cicatrix	2	5	2	1	..	3	1
Papilloma	3	3	3	2	..	1
Keloid	1	..	1
Chilblain	1	..	1
GENERAL INJURIES.										
Burns and Scalds	48	38	34	23	1	1	11	9	2	5
Contusions	23	8	22	7	1	1
Railway Shock	1	1	..
LOCAL INJURIES.										
<i>Injuries of the Head—</i>										
Contusion	2	..	2
Scalp Wound	29	15	25	15	2	..	2	..
Concussion of Brain..	56	5	51	4	3	..	2	1
Gunshot Wounds	2	..	1	1

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1876.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
<i>LOCAL INJURIES (continued).</i>										
<i>Injuries of the Head (continued).</i>										
Fracture of Vault of Skull—										
Simple	4	2	..	2	4
Compound	4	..	1	2	..	1	..
Fracture of Base of Skull	4	1	1	..	1	..	2	1
<i>Of the Ear.</i>										
Wound	1	..	1
Bead in Ear	1	1
<i>Of the Face.</i>										
Contusion	2	1	2	1
Wound	6	1	5	1	1	..
Fracture of Facial Bones	6	2	6	2
Do. Lower Jaw	4	2	4	2
Dislocation	1	..	1
Foreign Body in Cheek	1	..	1
<i>Injuries of the Eye.</i>										
Wound of Orbit	1	1
Burns	5	..	5
Wound of Eye	15	4	14	2	1	2
<i>Injuries of the Neck.</i>										
Contusion	2	..	2
Cut Throat	6	1	5	1	1
Foreign Body in Oesophagus	1	..	1
Wound of Pharyngeal Artery	1	1
Glass in Neck	1	..	1
<i>Injuries of the Chest.</i>										
Contusions	11	1	8	1	3
Fractured Ribs and Sternum, Lung not Wounded	20	1	18	1	2	..
Do. Lung Wounded	1	1
Incised Wound (Stab)	2	..	2
<i>Injuries of the Back.</i>										
Contusion	8	6	8	6
Fracture of the Spine	5	1	4	1	1	..
Injury to Spine, without Fracture	6	..	3	3	..
Fractured Coccyx	1	..	1

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
<i>LOCAL INJURIES (continued).</i>										
<i>Injuries of the Abdomen</i>										
Contusion	20	4	19	4	1
Do. with Rupture of Viscera	3	1	1	2	1
Wound	1	1	..
<i>Injuries of the Pelvis.</i>										
Contusion	1	..	1
Wound of Scrotum	4	..	4
Wound of Vulva	2	..	2
Ruptured Urethra	4	..	3	1	..
Fracture of Pelvis	5	1	2	1	2	..	1	..
Do. do. with Injury to Viscera	1	1	1	1
Hairpin in Bladder	2	..	2
<i>Injuries of the Upper Extremity.</i>										
Contusion	1	..	1
Sprains	1	2	1	2
Wound—										
Of Arm	7	..	6	1	..
Of Forearm	17	7	16	7	1	..
Of Hand	32	2	30	2	2	..
Injuries of Vessels	3	1	3	1
Fracture of—										
Clavicle	12	2	10	2	2	..
Humerus—										
Simple	8	7	8	6	1
Compound	6	2	5	2	1	..
Forearm—										
Simple	11	3	11	3
Compound	6	..	6
Bones of Hand—										
Compound	1	1	..
Dislocation of—										
Clavicle	3	..	3
Humerus	1	2	1	2
Co. Dislocation of Humerus	1	1	..
Radius and Ulna	1	..	1
Foreign Body in Hand	2	1	1	1	1

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
LOCAL INJURIES (continued).										
<i>Injuries of Lower Extremities.</i>										
Contusions	18	8	17	8	1	..
Sprained Ankle	13	7	12	6	1	1
Do. Knee	4	1	4	1
Wounds—										
Of Thigh	9	2	9	2
Over Knee	6	6	6	4	2
Of Leg	8	..	6	2	..
Of Foot	10	..	9	1	..
Of Knee Joint	2	..	1	1	..
Foreign Bodies removed	1	3	1	3
Wounds of Vessels	2	..	1	1	..
Fracture of Femur—										
Simple	55	17	50	16	1	..	4	1
Compound	3	..	1	1	..	1	..
Fracture of Cervix Femoris—										
Intracapsular	2	9	2	8	1
Extracapsular	7	2	5	1	1	1	1	..
Fracture of Patella	23	10	19	7	1	4	2
Fracture of both Bones of the Leg—										
Simple	93	31	79	24	1	1	13	6
Compound	20	7	12	4	4	1	4	2
Fracture of Tibia alone—										
Simple	27	7	26	6	1	1
Compound	4	..	3	1	..
Greenstick	1	..	1
Fracture of Fibula alone—										
Simple	32	17	30	16	2	1
Fracture of Bones of Foot—										
Simple	5	1	4	1	1	..
Compound	1	1	..	1	1	..
“Pott's Fracture” of Leg	11	6	9	5	2	1
Dislocations of—										
Leg	1	1	1	1
Patella	2	..	1	1	..
Foot	1	..	1

TABLE I (continued).

DISEASES.	Total number of cases under treatment.		Discharged cured and relieved.		Unrelieved.		Died.		Remaining in at the end of the year 1875.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
<i>LOCAL INJURIES (continued).</i>										
<i>Dislocations of (continued).</i>										
Co. of Foot	1	1	1	1
Semi-lunar Cartilage ..	2	1	2	1
<i>Diseases and Injuries not classified.</i>										
Rupture of Muscle and Tendon	3	1	3	1

APPENDIX TO TABLE I.

GENERAL DISEASES :

Pyæmia.—Of the cases admitted into the Hospital, three females suffered from puerperal pyæmia. In a fourth case a woman, æt. 24, had suffered from what appeared to be pyæmic abscesses occurring at intervals during a period of two years. She died of an attack of erysipelas with sloughing of the integuments of the face.

Cancer.—A man, 63 years old, suffered from hard carcinoma of the pharynx and larynx, chiefly of the posterior wall of the former. In consequence of dyspnoea tracheotomy was performed. He died two or three weeks after the operation from exhaustion, produced by suppuration extending from the wound of the operation into the axilla, opening in its course into the sterno-clavicular articulation.

One well-marked case of colloid carcinoma of the breast occurred in a woman, about 40 years of age. It was thought before removal to be an ordinary case of hard carcinoma.

DISEASES OF THE NERVOUS SYSTEM :

Spinal Meningitis.—A woman, æt. 54, had been attending the out-patient rooms at St. Bartholomew's and King's for about five months on account of an abscess in the right loin. A month before her admission into the Hospital the discharge from the abscess began to diminish, but the patient became generally worse and more feeble. On the 11th November she found on getting up in the morning that she was unable to move the right leg. The next morning could not move the left leg. Was admitted on the 14th with paraplegia and loss of sensation in both lower extremities, with retention of urine and constipation. A small sinus discharged very slightly in the right lumbar region. On the 15th the right upper extremity was paralysed. On the 16th, loss of power in the left upper extremity. On the 18th she gradually sank and died. The autopsy disclosed pus between the arachnoid and pia mater of spinal chord from the cauda equina as far up as the cervical enlargement. The chord was exceedingly soft, rapidly decomposing. A large collection of dirty-brown pus was found over the right side of the sacrum. No diseased bone. Matter could not be traced along the course of the sacral or lumbar nerves directly into the spinal canal, but the inference was obvious that it had thus made its way.

DISEASES OF THE NOSE :

Epistaxis.—A man of intemperate habits, 54 years old, was admitted with hæmorrhage from the nose. The left nostril was plugged in front and behind. Two days after his admission he presented the symptoms of delirium tremens, and would not allow the plugs to be removed or re-adjusted. On the third night, during sleep after a small dose of anodyne, his breath became laboured, and before the assistance of the House Surgeon could be obtained he expired. The trachea and bronchi were found to contain a large quantity of blood,

which would appear to have flowed down into them as he lay in the supine posture.

DISEASES OF THE CIRCULATORY AND ABSORBENT SYSTEMS:

Aneurism of the Popliteal.—A labourer, 32 years old, was admitted with an aneurism of the popliteal artery. On the 3rd July the femoral artery was tied with carbolised catgut. On the 8th July there was slight return of pulsation in the middle of the tumour. On the 10th the catgut ligature was sufficiently absorbed to break and come away. The tumour was smaller and firmer, pulsation very slightly increased, but not distending the tumour. On the 14th hæmorrhage (5ij) from upper part of wound, ceasing spontaneously. It occurred whilst the patient was straining at stool. Pad and bandage. On the 24th recurrence of hæmorrhage. Wound opened up; ulcerated opening found at seat of ligature; vessel tied above and below the opening. On the 31st, about 6oz. of hæmorrhage from the wound, controlled by compress and bandage. Pressure over vessel above by means of weight and air-cushion. On the 2nd August about Oj of blood lost. Amputation just below the trochanters was then performed, but the patient died on the 3rd.

DISEASES OF MOUTH AND CHEEK:

Congenital Orifice below Mouth.—A very singular case of a female infant having a small orifice about half an inch in diameter (when open) just below the corner of the mouth. This orifice was surrounded by mucons membrane, furnished with a sphincter, opening and shutting synchronously with the mouth. It led into a small cavity lined by mucons membrane, separated from the mouth by a thin septum. Saliva appeared to flow into this cavity. The orifice was closed by operation, an opening being at the same time made through the membranous septum into the mouth.—(See *Lancet*, Vol. I, 1876, p. 13).

DISEASES OF THE INTESTINES:

Internal Strangulation.—Two cases occurred, the one in a young man, æt. 24, in whom the symptoms commenced suddenly on the 16th of October. He did not come to the Hospital until the 24th, when it was thought inadvisable to operate, as his symptoms had slightly abated. He died on the 27th, having become collapsed a few hours previously. A portion of the ileum was found strangulated by a long fibrous cord extending between the mesentery and a diverticulum of the small intestine. The bowel had given way, and there was extravasation of fæces.

The second case occurred in a labourer, 57 years old. The symptoms had existed ten days before admission. He was too ill, when admitted, to give any proper and reliable account of his case. Death occurred about forty-eight hours after he came into the Hospital. After death several coils of the ileum just above the ileo-colic valve were found to be firmly bound together, and so constricted that, with a little pultaceous matter contained in the gut, so total an obstruction was produced that even water, forcibly injected, would not pass through.

Inguinal Hernia.—A man, 47 years old, had been in Lazarus Ward with constitutional syphilis for nearly a month, when one morning (February 27) an inguinal rupture, from which he had suffered for some years, came down whilst he was at stool. He put it back himself. On the 29th it came down again. He again put it back. That same night it came down again and he could not return it. It was partially reduced by the House Surgeon, completely reduced a little later in the day. But the symptoms of strangulation from which he had suffered before it was reduced continued. On the 2nd February a consultation

was held; slight fulness was thought to be felt high up in the inguinal canal; exploratory operation was performed. A small sac was found high up in the canal close to the internal ring, but not strangled there. A knuckle of intestine was strangulated within the sac at its neck. It was returned, and the wound was closed. He made a good recovery; by the 14th February the wound was quite healed and he was ready for a truss. Before he left the Hospital he was attacked by pleurisy with effusion, from which he died on the 6th March. This case is, however, entered as one of the deaths from hernia.

DISEASES OF URINARY SYSTEM :

Lateral Lithotomy.—Both the deaths occurred from hemorrhage two or three days after the operation. One of the patients was 20 years old, the other 27 years.

DISEASES OF THE FEMALE BREAST :

Chronic Eczema about Nipple.—A woman, aged 52, suffered amputation of the breast on account of long continued eczema about the nipple with slight induration in the upper part of the mammary gland. The operation was performed in the hope of averting the formation of cancer. (For further account of this case, see Med. Chir. Transactions, Vol. LIX.)

DISEASES OF THE ORGANS OF LOCOMOTION :

Acute Necrosis.—A young man, 18 years old, was admitted with acute necrosis of the lower portion of the femur. About a month after admission the disease extended to the knee-joint. Amputation of the thigh was performed, but death occurred about 52 hours after the operation without any very evident cause.

In a boy, 12 years old, also suffering from acute necrosis of the femur, repeated hemorrhages into the suppurating cavity between the dead bone and the newly-forming bone, necessitated amputation of the thigh. He made a good recovery, although he was much weakened by loss of blood.

An infant, 15 months old, was admitted in November with acute necrosis of the lower half of the femur. The mother was allowed, at her express desire, to take it home at the end of four days. It was re-admitted on December 15, much worse, and having a spontaneous fracture of the femur. Consultation was held. It was thought that the only chance for the child lay in amputation of the thigh. This was accordingly done on the 16th, but the child sank and died on the 17th.

DISEASES OF THE CELLULAR TISSUE :

Sarcoma.—An enormous spindle-celled sarcoma of the buttock was removed from a clerk, 46 years old. Secondary hemorrhage occurred on the 12th and 14th days after the operation. During the second hemorrhage he died. There had been very little bleeding at the time of the operation, the tumour shelling out easily.

Elephantiasis.—Two cases occurred, one of elephantiasis of the scrotum, the other of nævoid elephantiasis of the lower extremity.

INJURIES OF THE FACE :

Foreign Body in Cheek.—A girl, 10 years old, suffered from the continual

re-opening of a sinus in the cheek, following a wound received $2\frac{1}{4}$ years previously by falling upon an earthenware pot. The trouble was finally found to be due to a small portion of the pot, which had lain deep in the substance of the cheek ever since the accident. This was removed and the sinus at once healed.

INJURIES OF THE EYE :

Wound of Orbit.—A youth, 16 years old, whilst standing on a chair cleaning a glass globe, fell. The globe was broken and some of the glass entered his right orbit, cutting through the inner portion of the upper and lower lids. He was admitted July 19, placed under chloroform, and three portions of glass were removed from the orbit, one of which was firmly impacted in frontal and sphenoidal bones. Severe inflammation of the orbit followed, with ulceration of the cornea. On the 22nd he was drowsy and complained of intense pain in the head. Also delirious. Head symptoms continued for a week, then gradually subsided. By August 23 was so much improved as to be able to go out into the square. September 11 and 12 headache returned. On the 13th was delirious, but soon became drowsy, and died comatose. No paralysis of cranial nerves previously. No convulsions. The autopsy discovered a small quantity of pus between the dura mater and arachnoid in the anterior and middle fossæ of the skull, also a piece of glass 1 inch by $\frac{3}{8}$ inch imbedded in the anterior lobe of the cerebrum, and several small portions beside this piece. These portions of glass lay in an irregular cavity surrounded by blood-clot and softened brain substance. There was scarcely any meningitis.

INJURIES OF THE NECK :

Wound of Pharyngeal Artery.—A greengrocer, 23 years old, while drunk on the 17th September, fell forwards with a clay pipe in his mouth. The pipe broke and grazed (as was thought) the left tonsil. On the 19th came to the Hospital with inflammatory œdema of the fauces. An incision was made in the back of the soft palate. He was admitted and went down for a bath. Whilst there he vomited a quantity of blood. In the evening several ounces more of blood escaped from the mouth. On the 21st a sudden rush of nearly a pint of blood occurred. Cavity found in left side of throat, plugged. On the 22nd two hæmorrhages closely following each other. Examination of the throat made under anæsthetic. A piece of tobacco-pipe $\frac{3}{8}$ in. long found in tonsil. This was removed and cavity plugged. The common carotid was then tied, but the patient sank and died in three hours.

Glass in Neck.—A woman, 23 years old, had a portion of glass removed from the side of the neck, where it projected in front of the sterno-mastoid muscle. It was $1\frac{1}{2}$ in. long by $1\frac{1}{4}$ in. The wound through which it had entered was completely closed. There was not the slightest inflammatory irritation, although the glass had been impacted in the neck for six months. It was a portion of a cab window. The cab had overturned, and the broken window had cut the patient's neck.

INJURIES OF THE CHEST :

Incised Wound (Stab).—On the 1st June a wood turner, æt. 33, stabbed himself in the head, chest, and abdomen with a chisel. The wounds of the head and abdomen were not very severe. But the wound of the chest was situated just to left of the sternum, between the fifth and sixth ribs. It was three inches long. Through it the lung could be easily seen wounded and bleeding. The wound in the chest was closed with strapping and lint soaked in carbolic oil. On the 3rd there was emphysema. This soon cleared up, and he had scarcely any further trouble, being up and dressed in four weeks from the date of his admission.

INJURIES OF THE ABDOMEN :

Contusion, with Rupture of Viscera.—A man, 48 years old, was admitted with several broken ribs on the left side from a fall through a skylight. He was suffering much from collapse. About twenty hours after the injury he died. The commencement of the jejunum was almost completely torn across; the fœcal matter was extravasated into the abdominal cavity; and there was general peritonitis, the intestines being just glued together by quite recent lymph.

INJURIES OF THE PELVIS :

Fracture of Pelvis with Injury to Viscera.—A girl, 15 years old, was knocked down and run over by a hansom cab, the wheel of which passed over her thighs and the lower part of the abdomen. On the 30th June, the day after admission, she lay on her back, very still, complaining of great pain about the lower part of the abdomen. There was very little mark of injury. She passed urine which was very dark, almost sooty. Until July 3 the urine was quite clear, apparently normal; signs of peritonitis were well marked. On July 3 the urine contained a good deal of blood. On the 8th began to complain of pain in micturition. On the 14th a large fluctuating swelling was noticed in the left side of the abdomen and iliac region. On the 15th this swelling was punctured, about 6 oz. of clear, glairy fluid let out, followed by some reddish-grey fluid, thicker than the first, and ammoniacal. On the 17th she gradually sank from exhaustion. After death a large cystoid cavity was found to the left side of the bladder, behind the peritoneum, communicating with the bladder through a rupture in its left side. The cystoid cavity had been tapped during life: it was surrounded by very considerable diffuse inflammation. There had been general peritonitis, but not very severe.

INJURIES OF THE UPPER EXTREMITY :

Injuries of Vessels.—In one case of the brachial artery; in the remaining cases of the radial and ulnar arteries.

Dislocations of Humerus and of Forearm.—The dislocations of the humerus were, two sub-coracoid, one sub-spinous. The dislocation of the forearm was backwards and outwards.

INJURIES OF THE LOWER EXTREMITY :

Compound Fracture of the Leg.—A young man, 20 years old, was admitted on the 1st of March with a severe compound comminuted fracture of both bones of the right leg. As the chief vessels and nerves appeared to be sound, it was decided not to amputate. On the 3rd a small blue spot of gangrene appeared on the third toe. On the 4th the whole leg was gangrenous. On the 5th and 6th the disease extended up to the groin. On the 7th he died.

A second case of gangrene occurred in a waiter, æt. 49, who had suffered a compound fracture of the leg. The disease was of much later occurrence than in the last case, not showing itself for more than a fortnight after the accident.

Dislocations.—The dislocations of the leg were backwards and outwards.

Those of the patella were outwards.

The dislocation of the foot was inwards.

The compound dislocations at the ankle-joint were outwards, and backwards and outwards. An attempt was made in each case to save the limb. Secondary amputation was performed at the end of a month in the first case (a boy). The second patient (a woman, 40 years old) died in a few days of phlegmonous inflammation and gangrene.

DISEASES.	Under 5.		5-10.		10-15.		15-25.		25-35.		35-45.		45-55.		55-65.		65-75.		75 and upwards.		TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.			
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE ORGANS OF LOCOMOTION.																								
Periostitis ..	1	1	1	3	2	2	2	2	2	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Chronic Abscess. .	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Diffuse Periostitis— Acute Necrosis ..	1	1	2	7	5	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4
Caries ..	1	1	4	7	5	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4
Necrosis ..	2	1	1	9	3	4	2	4	2	6	2	1	1	1	1	1	1	1	1	1	1	1	1	1
Tumours—																								
a. Exostosis ..	1	1	1	2	3	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
b. Enchondroma ..	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
c. Sarcoma ..	1	1	1	2	1	2	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Old Amputations, &c. .	1	1	1	3	2	1	3	2	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
<i>Diseases of Joints.</i>																								
Acute Synovitis ..	1	1	2	9	6	8	6	8	6	8	1	8	1	2	1	1	1	1	1	1	1	1	1	1
Chronic do. .	7	7	19	12	19	11	6	5	4	3	4	3	4	3	4	3	4	3	4	3	4	3	4	3
Ulceration of Cartilage.	1	1	1	3	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
Ankylosis ..	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Knock-Knee, &c. .	1	1	2	5	2	1	5	2	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Weak Ankles ..	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Loose Cartilage in Knee	1	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2

DISEASES.	Under 5.		5-10.		10-15.		15-25.		25-35.		35-45.		45-55.		55-65.		65-75.		75 and upwards.		TOTAL.					
	Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.					
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F		
DISEASES OF THE CUTANEOUS SYSTEM.																										
Ulcer ..	1	1	2	..	5	1	2	3	5	7	3	8	3	3	2	20	25	1	1
Carbuncle ..	1	1	..	1	..	1	..	1	6	3	2	1
Thecal Abscess	1	..	2	2	1	1	1	1	2	3
Gangrene	1	1	1	1	1	2	3
Cicatrix	1	1	1	1	2	2	2	4
Nævus ..	4	4	1	4	5
In-growing Toe-Nail	1	3	3	1	2	6
Bed Sores	1	1	2	2	1
Chilblain	1	1
Papilloma	2	2	1	3	3
Keloid	1	1
GENERAL INJURIES.																										
Burns and Scalds ..	21	13	9	4	5	1	1	3	2	4	1	4	1	1	..	2	1	35	24	11	9
Contusions	1	..	1	2	..	6	3	4	4	4	4	4	4	4	3	2	2	2	22	7	1	..
LOCAL INJURIES.																										
<i>Injuries of the Head.</i>																										
Contusions	1	2

DISEASES.	Under 5.		5-10.		10-15.		15-25.		25-35.		35-45.		45-55.		55-65.		65-75.		75 and upwards.		TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
LOCAL INJURIES (contd.)																								
<i>Injuries of the Head</i>																								
<i>(continued)</i>																								
Scalp Wounds ..	1	..	3	1	7	1	4	2	1	3	1	5	3	1	2	1	4	26	15	3	
Concussion of Brain ..	2	..	16	1	9	2	..	6	1	1	5	1	4	1	2	..	1	51	4	3	
Fractures of Skull	1	2	1	4	1	1	1	..	2	3	2	8	
<i>Injuries of the Face.</i>																								
Contusions	
Wounds ..	1	1	1	1	2	1	..	
Fractures, &c.	5	1	
Injuries to Ear	3	2	1	..	3	2	1	11	5	..	
<i>Injuries of the Eye.</i>																								
Contusions ..	1	..	1	..	4	1	..	4	1	..	6	..	1	..	2	1	2	
<i>Injuries of the Neck.</i>																								
Cut Throat	19	2	1	
Other Injuries ..	1	1	2	..	2	1	1	5	1	1	
<i>Injuries of the Chest.</i>																								
Contusions	
Wounds	3	1	1	..	1	..	1	8	1	3	
Fractured Ribs	3	8	1	..	4	1	2	
	18	1	1	

AGE AND SEX.

OPERATIONS.	Under 5 Years.		5—		10—		20—		30—		40—		50—		60—		70—		TOTAL.		Cured and Relieved.		Not Relieved.		Died.		
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	
	EXCISION OF JOINTS AND BONES (continued).																										
Ankle	1	1	..	1
Elbow	1	1
<i>Removal of Sequestra :</i>																											
From Head and Face	3	..	1	3	3	1	1	8	5	8	5
" Upper Extremity	2	..	1	2	2	5	2	5	2
" Lower Extremity	7	2	6	1	7	1	1	..	2	26	4	20	3	6	1
AMPUTATIONS.																											
<i>Primary :</i>																											
Arm	2	1	2	1	2	1
Forearm	1	..	1	2	..	2
Parts of Hand	1
Thigh	1	1
Leg	1	1
Parts of Foot..
<i>Secondary :</i>																											
Arm	1	1	..	1
Leg	1	1	..	1

During the year 1875 Anæsthetics were administered 1587 times.

Of these Chloroform was administered..	..	617	times.
Nitrous Oxide Gas alone	86	„
Ether alone	120	„
Ether, preceded by Nitrous Oxide Gas		764	„
		<u>1587</u>	

Youngest Patient	24 hours.
Oldest Patient	77 years. No Death.

APPENDIX
TO
TABLE OF OPERATIONS.

EXCISION OF JOINTS AND BONES :

Hip.—Child died many months after operation from gradual exhaustion and amyloid disease.

Knee.—In two cases amputation of the thigh was performed within a short period after the operation of resection ; in a boy, *æt.* 4, on account of malposition of the fragments and exhaustion ; in a girl, *æt.* 14, on account of necrosis of a large portion of the lower end of the femur.

Removal of Sequestra.—In several cases amputation was afterwards performed.

AMPUTATIONS :

Primary—Arm.—Of these cases a boy, 15 years old, suffered amputation of the right arm and of the left forearm just below the elbow.

Death occurred in a woman, 39 years of age, shortly after the operation. Amongst other injuries she had sustained a fracture of the base of the skull.

Thigh.—The only case was a clerk, *æt.* 48, who had sustained a compound fracture of the femur as well as other severe internal injuries. He died two days after admission.

Leg.—This patient had both his legs amputated about the junction of the middle with the lower third.

For Disease—Arm.—Both cases of malignant disease.

Forearm.—Of a woman, *æt.* 27, for malignant disease. In the other cases, for deformity consequent on old injury.

Thigh.—In eight males and three females amputation was performed for chronic disease of the knee-joint.

In a male, *æt.* 64, for a spindle-celled-sarcoma of the tibia. Pyæmia. Death.

In a male, *æt.* 32, on account of secondary hemorrhage from the femoral (described in Appendix to Table I). Death.

AMPUTATIONS (*continued*).

In a woman, 36 years old, on account of ulceration, &c., due to phlegmonous inflammation.

In an infant of 16 months, on account of acute necrosis of the shaft of the femur, with spontaneous fracture and profuse suppuration. Death two days after the operation.

And in two other cases of acute necrosis of the femur, in males of 13 and 18 years.

A delicate man, æt. 29, lost his thigh on account of rapidly spreading inflammation and gangrene following sequestrotomy performed upon the 5th metatarsal bone.

Leg.—The leg was twice amputated on account of ulcers, twice on account of disease of the ankle-joint, twice for epithelioma, in three cases for deformity.

Ankle-Joint.—In all cases Symes' amputation: in every case for disease of bones or joints.

REMOVAL OF TUMOURS.

Testicle Removed.—Male, æt. 22, for pure enchondroma; æt. 24, for sarcoma; æt. 35 and 49, for soft carcinoma.

REMOVAL OF CALCULI:

Lithotomy.—Both deaths occurred from hæmorrhage, within the first few days after the operation, in patients aged respectively 18 and 27 years.

LIGATURE OF VESSELS:

Common Carotid.—Case related in Appendix to Table I.

SUB-TABLE, SHOWING THE NUMBER OF CASES OF ERYSIPELAS, PYÆMIA, &c.

DISEASES.	Under 5.		5-10.		10-15.		15-25.		25-35.		35-45.		45-55.		55-65.		65-75.		75-85.		Total.		Deaths.			
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F		
	Erysipelas— Admissions.. .. . Occurring in Hospital .. " after Operations	1	1	2	1	6	2	4	3	10	2	6	2	3	2	..	2	32	15	1
Phlegmonous Inflammation— Admissions Occurring in Hospital	7	3	9	2	12	6	6	3	3	8	3	40	22	4	3	
Diffuse Cellulitis— Admissions..	1	..	2	1	..	2	2	1	1	..	2	8	4	2	2	
Pyæmia and Septicæmia— Admissions Occurring in Hospital " after Operations	1	..	2	3	4	1	2	1	
Phagedæna— Admissions..	1	1	2	3	5	1	3	
	1	1	..	1	2	1	..	1	..	
	1	2	

TABLE OF AMPUTATIONS WITH THE PERCENTAGE OF DEATHS DURING THE
TEN YEARS from 1866 to 1875, inclusive.

OPERATIONS.	CASES UNDER TREATMENT.										PERCENTAGE OF DEATHS.										Total Number of		Average Percentage of Deaths.
	1866		1867		1868		1869		1870		1871		1872		1873		1874		1875		Cases.	Deaths.	
	1866	1867	1868	1869	1870	1871	1872	1873	1874	1875	1866	1867	1868	1869	1870	1871	1872	1873	1874	1875			
PRIMARY.	1866	1867	1868	1869	1870	1871	1872	1873	1874	1875	1866	1867	1868	1869	1870	1871	1872	1873	1874	1875	9	4	44.44
Thigh	1	..	2	3	2	..	1	100.	..	50.	33.33	100.	..	4	..	44.44
Knee-Joint ..	1	1
Leg ..	4	3	3	1	3	1	33.33	15	6	40.	..
Ankle-Joint	1	1
Shoulder-Joint	1
Arm ..	2	1	2	1	2	1	1	3	3	3	50.	19	2	100.	10.52
Forearm ..	2	1	7	4	1	3	5	2	25	1	4.	..
SECONDARY.	13	4	30.76	..
Thigh ..	4	2	1	1	2	1	1	..	1	..	50.	2
Knee-Joint ..	1	..	1	2
Leg	1	3	..	1	1	2	50.	66.66	16	5	31.25	..
Arm ..	1	..	5	1	1	6
Forearm	1	..	1	1	1	4

OPERATIONS.	CASES UNDER TREATMENT.												PERCENTAGE OF DEATHS.												Total Number of		Average Percentage of Deaths.
	PERCENTAGE OF DEATHS.												Total Number of														
	1866	1867	1868	1869	1870	1871	1872	1873	1874	1875	Cases,	Deaths,															
FOR DISEASE.	1866	1867	1868	1869	1870	1871	1872	1873	1874	1875	1866	1867	1868	1869	1870	1871	1872	1873	1874	1875	Cases,	Deaths,					
Hip-Joint	4	2	50				
Thigh	128	28	21.87				
Knee-Joint	4	1	25				
Leg	60	11	18.33				
Ankle-Joint	49	4	8.16				
Shoulder-Joint	4	1	25				
Arm	13	3	23.07				
Forearm	18	1	5.55				

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