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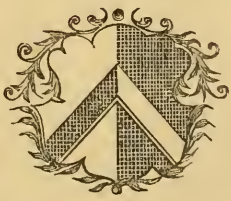

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SAINT BARTHOLOMEW'S HOSPITAL REPORTS.

ON PRÆSYSTOLIC MURMURS AT THE HEART'S APEX.

BY

JAMES ANDREW, M.D.

So much has been written of late on this subject, the nature, mode of production, and clinical characters of these murmurs have been so fully discussed, that any further statement about them might well seem to be superfluous. But, as a teacher, I find that the very wealth and variety of cardiac literature throw difficulties in the way of students, difficulties still further increased by the unnecessarily elaborate, not to say grotesque, nomenclature which many even of the best writers employ. My purpose, therefore, is not to add another to the many exhaustive papers already in existence, but to express, as shortly and simply as possible, what I hold to be the true doctrine of præsystolic murmurs.

First, with regard to nomenclature. For clinical use, it is, to say the least, highly convenient to apply the terms, "Systole" and "Diastole," "Systolic" and "Diastolic," to the action, the entire action, of the ventricles only, and thus to get rid of such complicated expressions as "Auricular-Systolic," "Ventricular-Systolic" or "Post-Diastolic," to some of which it is only too easy to attach an erroneous meaning.

There is no question here of the natural healthy action or sounds of the heart, to which terms such as these may or may not be applicable, but of certain abnormal sounds occupying a

part only of a complete cardiac revolution, and which, therefore, do not require for their description the use of each and all of the names which physiologists have arranged with so much ingenuity, like the points of a mariner's compass, about their circular diagrams.

For whatever may be the true theory of the production of murmurs, whether by fluid veins or otherwise, no one, I imagine, believes that a murmur can be produced at either auriculo-ventricular orifice when the blood in it is at rest. The existence of an endocardial murmur necessitates the simultaneous existence of some blood-movement at the spot where it is produced. But if the blood is in movement, it must be passing either into or out of the ventricle; if the former be the case, the ventricle must be dilating; if the latter, it must be contracting; for the blood never passes in a continuous stream through the ventricles, as it does through the auricles. All mitral and tricuspid murmurs must therefore be, in the proper sense of the words, either systolic or diastolic. It makes no difference whether they occupy the whole, or a part only, of the systolic or diastolic periods; their nature does, and their name ought to, depend upon the state of the ventricles at the time of their production, and upon this alone. The words "Systole" and "Diastole" have primarily nothing whatever to do with the sounds of the heart which occupy a part only, and a constantly varying part, of these periods, and the attachment of such reference to them by some writers is, I know, a frequent source of great confusion in the minds of students.

All murmurs, then, produced at the mitral orifice during the flow through it of blood into the ventricle are diastolic, although in practice, and for purposes of accurate diagnosis, it is convenient to distribute them further into three main classes, according to the portion, more or less, of the diastolic period which they may chance to occupy. Thus, a diastolic murmur at the apex may take place at the beginning or at the end, or it may be prolonged throughout the whole, of the diastolic period, but in each case it remains equally and always diastolic. To the murmur which occurs at the end of the diastole, and often seems to be continuous with the first sound of the heart, the name "Præsystolic" has been given, and it would no doubt be highly inconvenient to relinquish a name, sanctioned by long usage, for a murmur which is generally of peculiar character and significance: only be it always remembered that præsystolic are nothing more than one of the varieties of diastolic murmurs.

A well-marked præsystolic murmur, indicating obstruction at the mitral orifice, has the following characters:—

1. It occurs during the latter part of the diastole.
2. Its point of greatest intensity is a little within and above the apex-beat; it fades towards the left axilla, and is not heard behind.
3. A diastolic thrill is present over the left ventricle.
4. The murmur has a peculiar, harsh, droning sound, and becomes louder towards its close, which is abrupt.
5. It is generally difficult to distinguish an interval between it and the first sound.
6. The first sound is shortened and intensified.
7. Reduplication of the first sound is of frequent occurrence.
8. The second sound is very feeble, or even entirely absent at the apex.
9. The second sound is intensified at the left base, and very frequently reduplicated.
10. The physical signs of dilatation of the left auricle and of the right side of the heart are present.
11. The murmur varies much in loudness, length, and pitch, may disappear entirely, or even be replaced by a systolic apex murmur. These changes are of common occurrence as the case approaches its termination.
12. The pulse is often small, rapid, and very irregular.

These characters are the natural consequences of the passage of the blood through the obstructed or roughened mitral orifice, and of the interference with the simultaneous action of the ventricles by the delay of the blood stream on the left side of the ventricular septum. In fact, all, or nearly all, of them might have been predicted before they had been actually observed. There are, however, one or two the full significance of which is not, I believe, as yet universally recognised, and on these I wish to make some short comment.

And first, it ought always to be kept in mind that a murmur with the greater part, or even with the whole, of these characters does not of necessity indicate mitral stenosis. Doubtless it generally does so; but roughening, without constriction, will produce an onward murmur at the mitral not less than at the aortic orifice. And the instances in which a præsystolic murmur has been present during life, and no narrowing of the mitral orifice found after death, no more disprove the frequent causal connection of the narrowing and the murmur in the case of mitral, than in that of aortic disease. What they do prove is that "præsystolic murmur" and "murmur of mitral stenosis" are not synonymous.

The statement in sign 2, that the præsystolic murmur is not heard behind, requires qualification. I have often heard it there, but then it has either been so loud as to be heard over the whole

chest, or some other abnormal condition, *e.g.*, consolidation of the lower lobe of the left lung, has been present.

As regards sign 5, viz., the continuity of the murmur with the first sound, how are we to explain the disappearance of the short pause? I think in two ways. The murmur is often very loud, and increases greatly in intensity towards its termination, and thus the ear has not time to lose the impression of the diastolic murmur before the systolic sound falls upon it. Especially would this be the case if the normal duration of the short pause were at the same time diminished, as indeed it probably is. For the left ventricle, filling slowly during its diastole from the obstruction at its feeding orifice, will be roused to sudden and energetic contraction at the beginning of its systole by the twofold stimulus of the already commencing movement of the right ventricle and of the sudden afflux of blood caused by the systole of the auricle. These two considerations, viz., first, an apparent, and, secondly, a real shortening of the normal interval between the first and second sounds, seem to me to offer a sufficient explanation of the seeming continuity of the præsystolic murmur with the first sound of the heart. And again, in many cases the effect of these causes must be further increased by the alteration in the first sound itself, sign 6, which begins more suddenly and abruptly than in health; partly, as already mentioned, from the abrupt beginning of the systole of the left ventricle, partly, and in some cases, from certain changes in the valve. On examining a good specimen of a so-called "button-hole" mitral valve, it is seen that the two curtains, but for a short narrow slit between them, form together a loose septum between the auricle and ventricle, the tension of which by the sudden pressure of the ventricular systole must produce a sharp flap, like that of an ill-managed sail. And it is just this condition of things, viz., stricture of the orifice by adherence of the edges of the cusps without calcification, which is found very frequently after death in typical instances of præsystolic murmur. It must not, however, be supposed that the short cardiac pause is always absent. If the murmur be double, *i.e.*, if there be regurgitation as well as obstruction, there is always an interval between the two sounds, for the reversal of the current cannot take place without an appreciable moment of rest; and again, the præsystolic murmur itself not unfrequently becomes less, instead of more, loud towards its close, which is then followed by a distinct period of silence.

There is yet one other point in this connection which requires notice. The diagnosis of a præsystolic murmur is so easy, its signs appear to be, nay, are, so distinct and characteristic, that the apparent impossibility of making a mistake is the very cause of

its being made. Systolic murmurs, like diastolic ones, very frequently do not occupy the entire cardiac period within which they occur. They may, indeed, do so, or they may be audible only at the beginning, or towards the end, of the time during which the ventricle is contracting. Now a murmur at the beginning, and the beginning only, of the systole is, I believe, very common. Any condition of the valve, *e.g.*, a little thickening along its line of attachment, which interferes with the natural position of the cusps when the systole begins, is sufficient to produce one. After death, even in a healthy heart, if a stream of water be allowed to flow gently into the left ventricle from the aorta, a certain amount will generally pass right through into the auricle, but on increasing the intra-ventricular pressure, the valve becomes at once water-tight. Now it is difficult, if not impossible, to distinguish by the ear alone between a murmur at the end of the diastole and one at the beginning of the systole, and which each stand in all but the same relation to the following first sound. A little carelessness, a little too implicit reliance on the seemingly pathognomonic sounds alone, is all that is wanting to mislead the auscultator, and then another instance is put on record in which a præsystolic murmur did not indicate mitral contraction or roughening. Of course, the mistake is one which ought not to be frequently made, but unless in each case the time of the murmur be accurately determined by the pulse in the great vessels of the neck, its presence or absence behind ascertained, and every available sign duly weighed, a murmur at the beginning of the systole is sure to be mistaken, sooner or later, for one at the end of the diastole, and this, probably, in some very simple case.

The explanation, whatever it may be, of the facts relating to the second sound at the apex, sign 8, raises questions of the highest physiological interest, but the case, from the clinical point of view, admits of very brief statement, and seems to point most distinctly to one conclusion. The problem is to explain the complete absence or great feebleness of the second sound at the apex, when at the same time it is to be heard at the base, distinctly on the right, and on the left of the sternum even more distinctly than usual. There is no doubt about the facts; a single day's hospital practice ought to suffice to convince any one that in many cases of præsystolic murmur the second sound is inaudible at the apex, and as loud as ever at the base. It cannot be that the second sound is masked by the co-existence of a murmur, for the murmur which is present is separated from the period of the second sound by the whole of the systole and something like one half of the long pause. Again, accepting for the time the valve theory of the second sound, it seems to me

impossible to admit that the second sound, as heard at the left apex, is derived entirely from the closure of the aortic valves, and that the sound produced by the pulmonary valves, intensified as it generally is in mitral obstruction, would not be heard there; so that great feebleness of the second sound at the aortic orifice would be sufficient to account for its absence at the apex. For the sounds produced in one ventricle are, when of a certain strength, heard most distinctly over the other. Thus the murmur of mitral regurgitation, loudest it is true at certain points, is yet heard over both ventricles, and the murmur of aortic regurgitation often covers completely the clack of the pulmonary valves. If the sole factor of the second sound, as heard at the apex, is the closure of the sigmoid valves, it seems to me all but impossible to explain its absence there in these cases. Further, it must be remembered that the facts at present under review are not the only ones which it is difficult to explain by the commonly received theory of the second sound. Here we have a second sound, of at least average loudness at the base, inaudible at the apex; in other instances, *e.g.*, in the later stages of typhoid fever, the second sound is often distinctly louder at the apex than it is at the base; and again, in aortic regurgitation a second sound is sometimes to be found at the apex when no trace of one can be detected at the base. These common facts taken together prove that there is no definite relation between the loudness of the second sound as heard at the base and at the apex; they do not of themselves establish the precise cause of that want of relation. But they are very perplexing on the theory which assigns the second sound to the closure of the sigmoid valves and to nothing else; very simple, indeed just what was to be expected, on that held by many writers, *e.g.*, by Pettigrew[1] and by Jaccoud,[2] that the principal factor of the second sound, as heard at the apex, is the sudden entry of blood into the ventricle at the beginning of its diastole, and in this connection it is noteworthy that a very slight murmur at the beginning of the diastole at the apex is incompatible with the existence of the second sound at that point. No doubt this might be accounted for by the supposition that the murmur masks the normal sound. But when, as often happens, the murmur is very slight, the blood-theory of the second sound, according to which the murmur replaces it, gives a much more complete and satisfactory explanation. It is unnecessary here to enter on detailed proof that sudden blood movement is sufficient to produce a sound undistinguishable from

[1] Physiology of the Circulation, 1874, p. 290.

[2] Leçons de Clinique, 1869. Sixième Leçon. Skoda also admits the possibility of the same view: "On Auscultation," translated by Markham, 1858, p. 198.

the second sound of the heart. I would merely mention the fact, which any one may easily verify, that the sudden pulse-wave in aortic regurgitation is attended in the larger arteries, *e.g.*, in the femorals, by a sound closely resembling the second, which in this case is not produced at, and therefore cannot be propagated from, the aortic valves.

Sign 9. The intensification of the second sound over the pulmonary valves is often, in the absence of murmur, the most direct proof of mitral obstruction—the form of heart disease which more frequently than any other is, though latent, the cause of increased tension in the pulmonary artery. I have elsewhere[1] spoken of the clinical significance of an intensified pulmonary second sound: the time and space now at my disposal only allow me to insist dogmatically on the paramount importance of accurately determining the relative loudness of both sounds at as many different points as possible. By this we are often enabled to detect otherwise latent disease, or to estimate the amount of mischief which many of the more obvious physical signs betray but do not measure.

The value of reduplication of the sounds, one or both (signs 7 and 8), depends upon the other conditions, local or general, with which it is associated. One of the commonest auscultatory phenomena, it may mean nothing more than a trifling attack of indigestion or of hysteria; but if mitral disease be present, then the reduplication, the result of a want of harmony between the action of the ventricles, becomes a most useful index of the amount of obstruction to which that want of harmony is due, and in this respect deserves to be placed on the same line with the intensification of the second sound, aortic or pulmonary.

I have purposely refrained from the easy task of supporting my statements by a long array of authorities. Every position in this short paper might, I well know, be completely buried beneath a list of references to writers who have either attacked or defended it. My wish is to state, as briefly as possible, the views which I have held and taught for some years past, on one of the most interesting points in cardiac pathology.

Without asserting the least claim to originality against those who may challenge any part or the whole of what I have said as their own, I would yet venture to quote the old saying, that it is possible to have a glass of water which you have not stolen from your neighbour's well.

[1] St. Bartholomew's Hospital Reports, vol. i. p. 34.

ON

THE ESSENTIAL PHÆNOMENA OF JAUNDICE.

BY

J. WICKHAM LEGG, M.D.

In the last volume of these Reports I attempted to review the different theories held in different times as to the causes of jaundice. I propose in the following paper to describe the appearances seen in jaundice; and the invariable appearances, not the variable. Thus I do not propose to discuss such phænomena as xanthopsy, the hæmorrhagic diathesis, the temperature of the body, or the slow pulse. Nor do I intend to speak of those diseases in which the jaundice is but a secondary symptom, such as pyæmia or acute yellow atrophy, which being general disorders, not local, may be discussed elsewhere; but I wish rather to describe the phænomena which follow any simple obstruction to the passage of the bile into the duodenum.

The first change from health to jaundice is seen in the colour of the conjunctiva. It becomes yellow. This is the symptom commonly first noticed by the friends of the patient, a day or two before the skin has changed its hue. In the lower orders it may often be found that the change of colour in the face is the first to draw attention, the yellowness of the eyes having been passed by. With the better classes, the eyes are commonly the first to be noticed. When the eyes show the first appearance of yellowness, however slight it may be, the urine will also commonly give a good reaction with Gmelin's test, showing the presence of bile pigment. How soon these symptoms set in after the obstruction to the flow of bile is first set up, there are at present no very certain means of judging. It is usually said that they appear

after the third day of obstruction;[1] but this conclusion is drawn from observations upon dogs. In these animals the conjunctiva has a brownish appearance, very unlike the pearl-white of a human sclerotic, and I look upon this as a hindrance to the early detection of changes of colour. From observations upon men, I should be inclined to put the first appearance of jaundice much earlier after obstruction, perhaps within the first twenty-four hours. In this opinion I am supported by several clinical observers.[2] The serous exudations will oftentimes give a reaction with Gmelin's test after death, when little or no trace of jaundice exists in the skin.

After the conjunctiva, the skin of the face becomes yellow. At first it may be no more than a sallow tinge, hard in some persons to tell from the natural tint of the skin. This is especially the case with persons of dark complexion. Those who are fair show the change earlier. From the face it spreads over the upper part of the trunk, and thence over the belly and limbs, the legs being the last to show the yellow appearance.

As a rule, the yellow colour of the skin is spread all over the body. It is, however, not uncommon to see, in cases of slight jaundice, the upper part of the body yellow, while the belly and legs are free from unnatural colour. Frerichs has made the same observation,[3] and Hecker has noticed a case of acute yellow atrophy in which the upper part only of the body was coloured yellow.[4] But in all cases of severe jaundice which have fallen under my observation, the whole of the body has been nearly equally jaundiced.

In some old authors,[5] a jaundice limited to the right half of the body and to the face and hands[6] has been described. I scarcely feel able altogether to reject these statements when an observation

[1] Frerichs, Klinik d. Leberkrankheiten, Braunschweig, 1858, Bd. i. p. 99. In my own experiments upon cats, the conjunctiva did not become coloured for many days after the operation (St. Bartholomew's Hospital Reports, 1873, vol. ix. p. 161).

[2] Siebert, Diagnostik d. Krankheiten des Unterleibs, Erlangen, 1855, p. 283. Audigné (Gaz. méd. de Paris, 1874, in Virchow and Hirsch's Jahresb. f. 1874, Bd. i. p. 349) found that bile pigment could be detected in the urine in three to four hours after ligature of the common duct of a dog. As the urine of dogs so often contains in health a body which gives a reaction precisely similar to that of bile pigment, this observation can be scarcely looked upon as conclusive.

[3] Frerichs, *op. cit.*, Bd. i. p. 112.

[4] Hecker, Monatsschrift f. Geburtsk., 1863, Bd. xxi. p. 212.

[5] Behrens, Ephemerides Nat. Cur. Noribergæ., 1715, Centioria iii. Obs. lxiv. p. 145. A man seventy years old, seized with right hemiplegia, had jaundice of the same side of the body as the hemiplegia: the right side of the nose was yellow, the left natural in colour. Behrens quotes from Ettmüller (Op. Med. Francof. ad Moen. 1697, t. ii. p. 844) a similar case. I have been unable to verify the reference. Morgagni (De Sedibus, &c., Ep. xi. sec. 14) mocks at Behrens' account.

[6] Strack, Journal de Médecine, 1768, t. xxviii. p. 163.

of a like kind has been recorded by so credible a witness as Joseph Frank. He found a lady to have at first jaundice of the right side only, which in two weeks spread over the whole body.[1] Surely this observer possessed as good means of forming an opinion on this point as any at the present day. But, at any rate, the appearance must be extremely uncommon, and be one of the rarest phænomena in medicine. Peter Frank, on the other hand, says he has never seen such a thing, and thinks it very hard to explain,[2] in which every one who is able to form an opinion on the matter must agree with him. Pouzol attempts to explain it by bringing into notice the varying vascular supply of different parts of the skin, which would cause a like variation in the supply of bile pigment.[3] This may serve to explain some cases of a yellow colour in spots; these have, however, still to be proved to be jaundice. It does not explain the appearance of jaundice in one half of the face while the other half remains natural. Morgagni can only explain Behrens' case by supposing a slowness of the circulation in the paralysed side and thus no time being given to tinge the parts.[4]

The older physicians were delighted to enumerate numberless species of jaundice according to the amount of bile pigment present in the skin. Aretæus describes two species, the white and the black jaundice, *melas icterus*, a word which remains in use in our own day, and says there are myriads of stages between the two.[5] Many of the so-called species of jaundice are quite different diseases, *icterus albus*, *ruber*, and *cæruleus* being the names for chlorosis, erythema, and cyanosis.

The mucous membranes do not share in the yellow tint of the skin. The lips, for example, retain the redness of health, and the inside of the mouth shows the usual colour.[6] A yellow colour may be got, however, if pressure be made so as to drive all the blood out of the mucous membrane, and allow the tint which is underneath that of the blood vessels to appear. There is one part of the mucous membrane of the mouth, the part under the tongue, which is often yellow, almost constantly so, an appearance noticed by Hippocrates,[7] but to which little attention

[1] Joseph Frank, Praxeos Medicæ Universa Præcepta, Leipsig, 1843, pars ii. vol. ii. sec. ii. fasc. i. p. 278.

[2] Peter Frank, De Curand. Hom. Morb. Epitome, Vienna, 1821, lib. vi. pars. iii. p. 308.

[3] Pouzol, Essai sur l'Ictère, Paris, 1872, p. 63.

[4] Morgagni, *loc. cit.*

[5] Aretæus, Adams' ed., p. 83.

[6] Villeneuve (Dict. des Sciences méd., Paris, 1818, vol. xxiii. p. 405) says the lips become a deep yellow, and Lonjon (Gaz. méd. de Paris, 1845, p. 231) that the inside of the mouth is yellow.

[7] Hippocrates, De Morb. lib. ii., Littré's ed., t. vii. p. 54.

has been paid. Lonjon thinks that in every case of jaundice the *velum palati* shows a deep and constant yellow colour.[1] And Pupier says that the roof of the mouth is the first to show the yellow colour, and the last to lose it.[2]

The secretions of mucous surfaces and those of the glands which open upon mucous surfaces or the skin, saving always the notorious example of the kidneys, do not as a rule contain bile pigment. The saliva is colourless. I have often tested it in cases of severe jaundice, without being able to detect any reaction, with nitric acid.[3] In like manner, the tears, the sweat, and the milk of jaundiced patients are as colourless as in health. To this statement there are rare exceptions. The sweat is the most common exception.[4] The linen near the armpits may sometimes be seen stained yellow as if wetted with the urine. This is certainly the most common place for the sweat to be coloured, although the appearance of colour in other parts has been recorded. Thus, in an old case of Chomel's, a woman seized with an epilepsy, jaundice came on; a thick sweat broke out, and the linen with which they wiped the patient was tinged of a saffron colour.[5] Sir Henry Marsh says that Cheyne had met with a patient whose handkerchief was stained yellow when she wiped her face.[6] Andral mentions a really curious case in which the stools were white, but the skin and conjunctiva colourless; nevertheless the urine was bilious and the linen used to wipe the sweat off the head was coloured yellow. After death the liver was found softened, not fatty; the cystic and hepatic ducts free from bile, and a colourless serous fluid in the gall-bladder; nothing unnatural was found in the duodenum.[7] Textural changes, like those in the kidneys, do not appear in the sweat glands. Frerichs figures brownish granules within the glands, but nothing like the great masses of pigment seen in the kidney.[8]

There are some early notices of a change of colour of the

[1] Lonjon, *loc cit.* Decaisne (Comptes rendus des Séances de l'Académie des Sciences, 1871, t. lxxiii. p. 1486) noticed the same appearance in an epidemic of jaundice at Paris.

[2] Pupier, Gaz. hebd., 1875, p. 307.

[3] Mosler (Berliner klin. Wochenschrift, 1866, p. 173) could find neither bile pigments nor bile acids in the saliva of the parotid gland from three jaundiced patients.

[4] Noticed by Galen, De Sanitate Tuenda, lib. iv. cap. iv., Kühn's ed., vol. vi. p. 250.

[5] Chomel, Histoire de l'Académie royale des Sciences, année 1737, Paris, 1740, p. 49.

[6] Henry Marsh, Dublin Hospital Reports, 1822, vol. iii. p. 269.

[7] Andral, Clinique méd., Paris, 1839, 4e éd., t. ii. p. 374; *cf.* p. 320, and t. i. p. 600.

[8] Frerichs, Path.-anat. Atlas zur Klinik der Leberkrankheiten, Tafel i. fig. 6.

saliva in jaundice,[1] and one of the best of these is due to an excellent observer, John Huxham. A gentleman forty years old, jaundiced, took overnight, with some other medicines, gr. viii. of calomel. The next day a very green saliva poured out of the man's mouth, exactly like green bile, only thinner. This flow of green saliva lasted forty hours, and very nearly equalled two quarts in amount. The green colour of the saliva passed into yellow, which lasted another forty hours, and then the salivation disappeared as suddenly as it came on. Huxham does not think it due to the mercury, on account of the smallness of the dose; the patient had before been salivated, apparently without mercury.[2] But the observer who has paid the most attention to the saliva, whether in health or disease, is undoubtedly Samuel Wright; and to him is owing the most complete account on record of the saliva in jaundice. He says that in jaundice, the saliva is seen in two forms, colourless and coloured, and that the coloured bilious saliva is of various shades, from a golden yellow to a deep olive; it is always abundant, and though it sometimes alternates with that of other secretions, it rarely permanently diminishes until the patient be free from the jaundiced tinge. The salivary glands are usually tinged, and of a dull red colour, but they are not painful, and they show no signs of active inflammation.[3] Wright thinks a discharge of bilious saliva may precede, accompany, or even hinder an attack of jaundice. He gives three cases of coloured saliva, in all of which salivation was present; and in the two first mercury was given; in the third, the treatment was by "salines and aperients." The saliva tasted bitter to the patients themselves. Budd likewise speaks of a case of jaundice in which salivation was caused by mercury, and the saliva was deeply coloured with bile.[4] Since, however, the custom of giving mercury for jaundice has fallen out of use, such cases seem to be less common. Leyden, by means of giving two grains of calomel every three hours, begat a salivation, and with the salivation a distinct yellow appearance of the saliva.[5] The records of a bilious saliva without salivation are rare, a yellow appearance of the buccal mucus being always excepted.[6] There is indeed a case of Dr. Hilton Fagge's, in which the saliva just before death had the colour of bile. The patient, a woman, had

[1] Nuck, Sialographia, Lugd. Batav. 1690, p. 49. Riedlinus, Lineæ Medicæ, August Vindelic. anni 1697, p. 88.
[2] Huxham, Phil. Trans. 1724, vol. xxxiii. p. 63.
[3] Wright, Lancet, 1842–43, vol. i. p. 559.
[4] Budd, On Diseases of the Liver, London, 1857, 3d ed., p. 469.
[5] Leyden, Beiträge zur Pathologie des Icterus, Berlin, 1866, p. 208.
[6] Andral, Clinique méd., Paris, 1839, 4e éd., t. ii. p. 320.

for six months suffered from a macular syphilide, and for three months from jaundice. She denied having used mercury; but during the ten days of her stay in Guy's Hospital she had the $\frac{1}{16}$ gr. of perchloride of mercury given three times a day, and on the day before death three grains of calomel. It does not appear that there was any notable salivation.[1]

The milk of women suckling sometimes contains bile pigment. This was first proved by means of chemical tests by Gorup-Besanez.[2] Older cases of a yellow appearance of the milk in jaundice have likewise been recorded.[3] Hervieux found quite lately, in an epidemic jaundice in the Maternité at Paris, that in all the women who were suckling, the milk was coloured yellow.[4] Underwood says that he has met with some striking instances in which women, suckling and jaundiced, have communicated the disease to their offspring, which has not been cured until the child were weaned or the nurse recovered.[5]

I have never been so fortunate as to see the milk coloured in jaundice. A patient of mine, suckling, who had symptoms of simple jaundice, told me that the milk was coloured green. Yet on some being pressed out of the nipple, no colour was apparent; neither, on applying Gmelin's test, could any certain reaction be got; the layer of milk above the nitric acid became of a dirty yellow, markedly different from the white of the milk, but no play of colours or distinct green was seen; nor did a green colour develop in the milk after it was kept three days.

As to the other secretions, they seem to be very rarely coloured. Heberden is commonly quoted as an authority for the statement that the tears sometimes become yellow; all, however, that he does say is, that one man assured him his tears were tinged in a jaundice.[6] Some French writers speak of the tears in the jaundice of the new-born being coloured; but I think this must clearly be a mistake—the pus of an ophthalmia being

[1] Hilton Fagge, Trans. of the Path. Soc. of London, 1867, vol. xviii. p. 136.

[2] Gorup-Besanez, Arch. f. phys. Heilkunde, 1849, Jahrg. viii. p. 719.

[3] Peter Frank, De Curandis Hominum Morbis, Vienna, 1821, lib. vi. pars. iii. p. 305. The vaginal secretion was likewise coloured. Henry Marsh, Dublin Hospital Reports, 1822, vol. iii. p. 263. Bright, Guy's Hospital Reports, 1836, vol i. p. 623. Wunderlich, Arch. d. Heilkunde, 1869, Jahrg. i. p. 35. Marsh's, Bright's, and Wunderlich's cases are from observations on the mammary gland after death. There are some other cases, sometimes set down as instances of bile being present in the milk, which merely show that the milk had, from some cause or other, a bitter taste (Ol. Borrichius, in Bartholini Acta Med. Hafniensia, 1673, vol. ii. p. 164, Obs. lxii.; and Samml. aus. Abh., 1792, Bd. xv. p. 121, note).

[4] Hervieux, Union méd., 1872, t. xiii. p. 610.

[5] Underwood, A Treatise on the Diseases of Children, London, 1805, 5th ed., vol. i. p. 27.

[6] Heberden, Commentaries, London, 1806, 3d ed., p. 247.

coloured yellow, and thus thought to be tears.[1] Sander says that the nurse attending one of his cases which died of acute yellow atrophy reported that the tears were yellow.[2] This suffers the same want of proof as Heberden's case, as the doctor himself did not see the tears.

The mucus of the nose has been seen to be yellow by one or two observers.[3] Joseph Frank says he has seen the secretion of the ears altogether suppressed in jaundice.[4] It is likewise asserted that the vaginal mucus is sometimes coloured.[5] Bouisson says he has found the semen in the *vesiculæ seminales* manifestly yellow.[6]

The fluids poured into the intestinal tract are likewise devoid of colour in jaundice; otherwise the fæces themselves would be coloured. I am not acquainted with any well-authenticated case in which these fluids were coloured; indeed, it would be hard to prove the presence of bile pigment in them apart from the fæces.[7] Osborne speaks of colourless fæces floating in a bilious fluid,[8] but this seems to me best explained by supposing an admixture of urine.

Although the mucous secretions remain uncoloured in jaundice while the mucous membranes and glands remain healthy, yet the secretions may become coloured if the glands be diseased. This has been already noticed in the case of the saliva. Simple catarrh does not seem to beget much change; but the croupous exudations seem very liable to be coloured in jaundice. For example, in simple catarrh of the bronchial tubes, the mucus is rarely coloured, although I have met with such an instance in one case; yet in bilious pneumonia, where a croupous exudation takes place, the sputa are very commonly coloured.

The fluid exudations into the serous cavities are among the first to show the presence of bile pigment.

The extra-vascular tissues are not coloured in jaundice. There are old stories quoted of the hairs sometimes changing

[1] Billard, Traité des Maladies des Enfans nouveau-nés, Paris, 1828, p. 647.

[2] Sander, Deutsche Klinik, 1860, Bd. xii. p. 32.

[3] Kercksig, Hufeland's Journal, 1799, Bd. vii. Stück iii. p. 98. There is also an old case by Riedlinus, Lineæ Med., anni 1679, August. Vindel. p. 88.

[4] Joseph Frank, *op. cit.*, p. 282.

[5] Peter Frank, De Curand. Hom. Morb., Vienna, 1821, lib. vi. pars. iii. p. 305. Reclus, Gaz. des Hop., 1872, p. 259. Stokes, Lond. Med. and Surg. Journal, 1834, vol. v. p. 199.

[6] Bouisson, De la Bile, Montpellier, 1843, p. 151. A similar instance is quoted by Joseph Frank (*op. cit.*, p. 281) from Petermann (Obs. Med., Dec. i. No. 9).

[7] Bouisson (De la Bile, Montp., 1843, p. 150) says that Voigtel (Handb. d. path. Anat., Halle, 1804, Bd. i. p. 552) records a case in which the pancreatic fluid was yellow in jaundice. What Voigtel does say is that a yellow fluid was found in the pancreas of a jaundiced person.

[8] Osborne, Dublin Quarterly Journal, 1853, vol. xv. p. 106.

colour, which, on examination, do not bear out the purpose for which they are quoted; the case quoted by Schenck, to wit, suggesting chlorosis rather than jaundice,[1] and that of Riedlinus being simply that a jaundiced woman told him that whenever she pulled out a hair, a yellow drop appeared at the spot whence the hair came out.[2] Stokes, however, tells us that many of his friends considered that the hairs were unquestionably coloured in some cases of jaundice. He says he has never seen such cases.[3] Neither have I.

Huxham's statement that the teeth were coloured green for a fortnight after convalescence in his case of bilious saliva[4] is confirmed by Wright, who says that in like cases he has seen the teeth coloured permanently yellow, green, brown, or black, according to the amount of bile present in the saliva.[5]

There is a curious case mentioned by Bleicher of a man of forty, who, on the eighth day of a jaundice with fever, passed urine quite blue, and at the same time the lips and tongue, and with them the teeth, became blue. Eight days after he was cured.[6]

Pain is not a symptom of jaundice. Jaundice caused solely by the pressure of a tumour upon the hepatic ducts, or a plug of mucus within them, is not accompanied by any pain due to the jaundice; if pain be present, it is due to some other complication of the disease. It is, however, otherwise when the jaundice is due to the passage of a rough angular gall stone down the ducts; the friction and forcible dilatation of the ducts sometimes causes agony so great that it is necessary to give the patient chloroform to still the pain.

Pouzol, speaking of the slight troubles and absence of pain which in general accompany jaundice, says that he has noticed a headache sometimes attend this disease; the headache is frontal or general, sometimes lancinating, sometimes giving a feeling of weight.[7]

Patients with jaundice appear to me to be drowsier than others. They often sleep much during the day. Sleeplessness, unless due to the itching, is not at all a common symptom, although mentioned as one.[8]

[1] Schenk, Obs. Med. lib. iii., De Ictero, Obs. i., quoted from Cornelius Gemma Cosmocrit. lib. i. cap. 7.

[2] Riedlinus, Linea Med. anni 1697, August. Vindel., p. 88.

[3] Stokes, Lond. Med. and Surg. Journal, 1834, vol. v. p. 199.

[4] Huxham, Phil. Trans., 1724, vol. xxxiii. p. 63.

[5] Wright, Lancet, 1842–43. vol. i. p. 559.

[6] Bleicher, Schmidt's Jahrbb. 1839, Bd. xxi. p. 48.

[7] Pouzol, Essai sur l'Ictère, Paris, 1872, p. 78. Portal (Maladies du Foie, Paris, 1813, p. 133) mentions shortly that heat and weight of the head are often seen in jaundice.

[8] Pouzol, *loc. cit.*

Heberden speaks of hiccup being seen in jaundice, but without denoting any present or future mischief.[1]

The blood early suffers in jaundice. Saunders showed that, within two hours after ligature of the bile duct, the blood gave evidence of the presence of bile pigment.[2] I have repeated this experiment, following Saunders' directions exactly, but without attaining his results.[3] Frerichs, in like manner, could only detect bile pigment in the blood at the end of forty-eight hours after the ligature of the ducts.[4] I quite agree, however, with his statement that bile pigment may be found after death in the serous effusions of the chest or belly, in cases where none can be detected in the urine, nor any change seen in the skin. I have several times verified this statement, taking care to use only the fluids from the chest, as it may be objected to those from the belly that the bile in the gall bladder may have transuded after death.

It is granted by all that the bile pigments exist in the blood in jaundice.[5] The same cannot be said of the bile acids. For many years a fruitless search was made for them, only one chemist,[6] since Pettenkofer's discovery, and that not in cases of disease of the liver, being able to detect them in the blood by their peculiar reaction. In 1858 came Hoppe-Seyler's discovery of the bile acids in the urine,[7] and Kühne soon after found them in the blood of jaundiced dogs, even so soon as twenty-four hours after complete occlusion to the ducts.[8] Huppert found them in the blood of a rabbit who lived only six and a half hours after the bile ducts were tied, and in the blood of a cat who lived more than sixteen hours after.[9] Also in the blood of a man who died of acute yellow atrophy. But Ernst Bischoff could not find them in the blood, muscles, brain, or cerebro-spinal fluid of a patient who died with intense jaundice from cancer of the liver.[10]

When it was the custom to make analyses of the blood in mass, analyses to which little regard is now paid, several examinations

[1] Heberden, Commentaries, London, 1806, 3d ed., p. 246.

[2] Saunders, Treatise on the Structure, Economy, and Diseases of the Liver, London, 1803, 3d ed., p. 111.

[3] Wickham Legg, St. Bartholomew's Hospital Reports, 1873, vol. ix. p. 176.

[4] Frerichs, *op. cit.*, Bd. i. p. 99.

[5] See Lecanu, Transactions méd., 1831, t. vi. p. 113, note, for the bibliography of the older researches into the presence of the colouring matter of the bile in the blood. I have looked through these references, but the authors do not communicate any important results. Kane (Dublin Journal, 1833, vol. ii. p. 346) confirms Lecanu's statements.

[6] Lehmann, Lehrb. d. phys. Chem., Leipzig, 1853, 2te Aufl., Bd. i. p. 122.

[7] For history of this, see the section on the urine, p. 26.

[8] Kühne, Arch. f. path. Anat., 1858, Bd. xiv. p. 347.

[9] H. Huppert, Arch. d. Heilk., 1864, Jahrg. v. p. 253.

[10] Ernst Bischoff, Zeitschrift f. rat. Med., 1864, III. Reihe, Bd. xxi. p. 142.

of the blood from jaundiced persons were made. Lecanu[1] and Simon[2] found the amount of colouring matter of the corpuscles decreased, while Denis[3] found the globules natural in amount but the albumen decreased. Gorup-Besanez[4] found an increase of the water, and decrease of the blood corpuscles, and, at the same time, no increase of fatty matters, and no cholestearin. The absence of any increase of fat or cholestearin was noticed also by Kane[5] and Simon.[6] On the other hand, Becquerel and Rodier found an increase in the fatty matters and cholestearin,[7] Frerichs a large increase in the fatty matters, which were chiefly composed of cholestearin, and which rose sometimes to 4 or 5 per cent. Leucin and tyrosin were also found in the blood.[8]

Budd, though he seems to have made no analyses, says that, after jaundice has lasted some time, the globules of the blood are always less in proportion than in health. This probably results not so much from the mere presence of the principles of the bile in the blood as from a diminution of those reparative changes which the blood naturally undergoes in its passage through the liver, and to the impaired digestion which results from the absence of bile in the bowel.[9] Leyden seems to incline to believe that the poverty of the blood is due to the action of the bile acids upon the corpuscles.[10] For my own part, I feel inclined to agree with Budd and attribute the state of the blood to the impaired action of the liver and impaired nutrition, rather than to the solvent action of the bile acids upon the corpuscles.

The fæces in jaundice are often entirely wanting in colour.[11] They have a grey or clay coloured appearance, and are less consistent than the stools seen in health. Frerichs, however, says that the stools are firmer and more consistent.[12] To this statement I fear I cannot assent, or to that, that constipation is the rule. In simple jaundice, which is, I suppose, the most common of all kinds of jaundice, diarrhœa is very often seen. In chronic jaundice, diarrhœa is less common. In this position I am supported by no less profound an observer than Heberden, who,

[1] Lecanu, Trans. méd., 1831, t. vi. p. 109. The blood which he examined did not clot till long after it had cooled.

[2] Simon, Animal Chemistry, Day's trans., 1845, vol. i. p. 330.

[3] Denis, Essai sur l'Application de la Chimie à l'Etude physiologique du Sang, Paris, 1838, p. 309.

[4] Gorup-Besanez, Arch. d. phys. Heilk., 1849, Bd. viii. p. 530.

[5] Kane, Dublin Journal, 1833, vol. ii. p. 346. [6] Simon, *loc. cit.*

[7] Becquerel and Rodier, Recherches sur la Composition du Sang, Paris, 1844, p. 106. [8] Frerichs, *op. cit.*, Bd. i. p. 103.

[9] Budd, On Diseases of the Liver, London, 1857, 3d ed., p. 468.

[10] Leyden, Beiträge zur Pathologie des Icterus, Berlin, 1866, p. 117.

[11] Hippocrates describes the stools as yellow-white and stinking (De Morb. int., Littré's ed., t. vii. p. 252.

[12] Frerichs, *op. cit.*, Bd. i. p. 119.

granting that costiveness might be looked for in jaundice, says that, in fact, icteric persons are often disposed to have a purging. He looks upon the diarrhœa as a help to distinguish jaundice from ileus.[1]

Sometimes the fæces are parti-coloured, some part being more deeply stained with bile than the other, or even one part free from bile altogether while the other is coloured. Graves explains this appearance by supposing that the liver secretes bile during a certain part of the digestive process, then stops, and then secretes again.[2] It is rare in jaundice for the fæces to be unchanged in colour or show more staining with bile than natural. This last has given rise to theories of polycholia, or an excess of bile, as a cause of jaundice; and is one of the points relied upon by the defenders of the theory of hæmatogenous jaundice. Doubtless, the appearance of highly coloured fæces is sometimes hard to explain upon the theory of an obstruction to the duct, if the admixture of foreign colouring matters such as blood or drugs can be disproved. Still it is well to keep in mind the facts disclosed by morbid anatomy: some of the small ducts may be obstructed high up in the liver, while the large ducts and branches leading into them are quite free; also the small amount of colouring matter that need be in the blood to bring about the appearance of jaundice.

If the fæces be colourless, there can be little doubt that no bile enters the intestine; in other words, that the obstruction of the bile duct is complete. But the contrary does not hold good. The fæces may owe their colour to other sources than the bile, as the administration of drugs like charcoal, bismuth, or iron. Likewise the appearance of hæmorrhages into the intestinal track in jaundice is far from uncommon, and blood mixed with the stools may readily give the deceptive appearance of a dark colour to the fæces. The preparations of mercury given by the mouth may cause a green appearance of the fæces from the presence of the green sulphide of mercury. I cannot, however, argue with Osborne that it is possible that the secretions from the bowels may be coloured while the fæces are white. He says that the fæces in jaundice may be seen to be of a white colour like putty surrounded by a green or yellow liquid.[3] This seems to me to be due possibly to some error of observation. The fæces may not unfrequently be seen surrounded by a coloured liquid, but this is in cases where the bilious urine has been

[1] Heberden, Commentaries, London, 1806, 3d ed., p. 246.

[2] Graves, Clinical Lectures on the Practice of Medicine, Dublin, 1864, p. 342, reprint of 2d ed., edited by Neligan.

[3] Osborne, Dublin Quarterly Journal, 1853, vol. xv. p. 106.

suffered to mix with the uncoloured stools; thus a deceptive appearance is caused.

The fæces in jaundice do not seem to have been examined with much attention by the chemists. Dr. Austin Flint, jun.,[1] has found that they contain no stercorin, a substance which he says is derived from the decomposition of cholestearin, and which always appears in natural fæces. Dr. Flint thinks that no cholestearin is found in natural fæces. Hoppe-Seyler, commenting on Dr. Flint's researches, says that stercorin is nothing more than an impure cholestearin—cholestearin itself being abundantly found in the fæces of mammalia, including man.[2] A large amount of fat was also found by Dr. Flint in the fæces of jaundice.[3] Farines, in an old analysis, found the greater part of the fæces in jaundice to be formed of a fatty matter, soluble in æther.[4] A like result was attained by Trommer,[5] who analysed the fæces of one medical student who was jaundiced, and compared the results with those obtained from the fæces of another medical student who was healthy, but ate the same food. Far more fat was found in the fæces of the former than of the latter. Jaundiced persons, it is well known, dislike fat.

The Urine.—The urine very early shows changes in jaundice. It becomes high coloured.[6] It stains yellow linen or white paper dipped into it. This is an appearance often noticed by the patients themselves. The froth on the surface likewise shows a yellow look, and is long in disappearing.

The colour may be scarcely darker than natural, and need a chemical test to show the presence of bile, or the urine may be almost as dark as porter, of a green-brown, or brown-black. The reaction is acid, and the specific gravity is not low, seldom falling below 1010.[7] The urine is commonly clear, but may sometimes be turbid, owing to the presence of urates, which does not seem to be dependent upon the temperature of the patient.

The quantity of urine passed in the twenty-four hours in

[1] Austin Flint, jun., Recherches exp. sur une nouvelle Fonction du Foie, Paris, 1868, p. 101 ; also in American Journal of the Medical Sciences, 1862, vol. xliv. pp. 358, 364.

[2] Hoppe-Seyler, Virchow's Jahresbericht f. 1868, Bd. i. p. 97.

[3] Flint, *loc. cit.*

[4] Farines, in Orfila, Elém. de Chimie appliquée à la Méd. et aux Arts, Paris, 1836, t. iii. (quoted by Bouisson, De la Bile, Montpelier, 1843. p. 151).

[5] Trommer, reported by Niemeyer, Text-Book of Practical Medicine, trans. by Humphreys and Hackley, New York, 1870, vol. i. p. 679.

[6] The change in colour was noticed by Hippocrates (De Morbis, lib. ii., Littré's ed., t. vii. pp. 252 *et seq*).

[7] Gorup-Besanez (Arch. f. phys. Heilkunde, 1849, Jahrg. viii. p. 713) says that the specific gravity of the urine of jaundice is commonly high, about 1031.

jaundice is subject to considerable variations. Becquerel found in three cases in which he carefully noted the appearances of the urine, that in the first, a man, the amount in twenty-four hours was 1419 c.c.; in the second, a man jaundiced after a violent dispute, it was 634 c.c.; and in a third, a pregnant woman, it was 640 c.c.[1] The observations, however, were only made on one day, and are therefore less valuable than those which follow and were made in succession.

Leyden found in one of his cases that the urine was increased in quantity, while the temperature was normal, but sank in amount during a paroxysm of fever.[2] A. Vogel found in his case of jaundice, lasting for many months, and due to cancer of the liver, that the amount of urine varied from 1350 c.c. to 2000 c.c.[3] Kölliker and Müller, in their observations on a woman of twenty-five suffering from simple jaundice, note that the mean amount of the urine during the jaundice was 1670 c.c.; during the convalescence, 1401 c.c.; when quite well, 1370 c.c.[4] So that in this case there seems some evidence that the amount of urine passed was really increased in amount.

Julius Jacobs found the amount in three cases to be 2409 c.c., 1638 c.c., and 1332.[5]

I have measured the amount of urine passed in the twenty-four hours in cases of jaundice due to various causes. The highest mean was in a case of complete obstruction to the ducts by a hydatid cyst, and equalled 2790 c.c. in the twenty-four hours. The lowest mean was in a case of cancer of the pancreas and liver, and equalled 570 c.c. In two other cases of jaundice, due to the same cause, the means were 1971 and 1120 c.c. In the remaining six cases the means of the urine passed were 1100, 1355, 1433, 1481, 1788, and 2234 c.c., amounts not above or below the standard of health.[6]

In some cases of jaundice it has been noticed that, as the patient was dying, the amount of urine passed became very small, or indeed sank to almost nothing. Such a case has been noticed by Devay, in which the urine was suppressed three

[1] Becquerel, Séméiotique des Urines, Paris, 1841, pp. 413 and 393.

[2] Leyden, Beiträge zur Path. des Icterus, Berlin, 1866, p. 205. He likewise gives cases of increased or abundant flow of urine at pp. 114 and 184, although in the former of these two the temperature was high.

[3] A. Vogel, Zeitschrift f. rat. Med., 1854, Bd. iv. (neue Folge), p. 391.

[4] Kölliker and Müller, Verhandlungen d. phys. med. Gesellschaft in Würzburg, 1856, Bd. vi. p. 497.

[5] Julius Jacobs, Arch. f. path. Anat., 1877, Bd. lxix. p. 487.

[6] Wickham Legg, Med. Chir. Trans., 1876, vol. lix. I have noticed that dogs whose bile ducts have been tied pass a greatly increased quantity of urine. Feltz and Ritter found that after the injection of bilirubin into the veins, the amount of urine was much increased (Robin's Journal de l'Anat. et de la Phys., 1875, t. xi. p. 158).

days before death after a seven years' jaundice from gall stones.[1]

Albumen is rarely absent from jaundiced urine. The amount is indeed small, and some care is needed to detect it. No cloudiness is given by heat as a rule; an opalescence, however, appears on dropping a little nitric acid into the boiling urine. I look upon this as evidence of the presence of albumen in the urine. No opalescence is commonly given where a layer of nitric acid and the urine touch.

According to Kühne, the cause of the presence of this small quantity of albumen in jaundiced urine would be due to the action of the bile acids upon the red corpuscles.[2] It is well known that the red corpuscles, whenever they come in contact with the bile acids, are dissolved. Free hæmoglobin thus becomes present in the plasma of the blood, and is thrown out by the kidneys. The bile acids must be present in but small quantity in the blood or a much larger amount of albumen would be thrown out by the kidneys.

Sugar is not often met with in jaundiced urine. Indeed, though I have carefully looked for it in nearly all the cases which I have examined, I have never met with it. The test which I have used has been Trommer's. On theoretical grounds the appearance of sugar should be rare, as one important form of diabetes does seem to arise from the liver. I have shown[3] that after ligature of the bile duct no glycogen is made in the liver, and that, under the same circumstances, when the fourth ventricle is irritated, no sugar appears in the urine. So in jaundice, if the obstruction to the ducts be complete, no sugar from the liver should appear in the urine. There are, however, a few cases in which sugar has appeared in the urine of jaundice. In Golowin's case, a dog was artificially jaundiced, and each time that milk was given to the animal, sugar appeared in the urine.[4] Now this experiment is rather in favour of my own observations, for when sugar of milk is in health absorbed from the stomach into the liver, it is split up into glycogen, and does not appear in the urine as sugar. But in Golowin's case the liver failed to break up the sugar, and thus it was excreted by the kidneys. There is an important case of this kind recorded by Dr. Bright in his paper on diseases of the pancreas. A man, forty-nine years of age, first felt symptoms of diabetes in the month of March. In the beginning of September he began to be jaundiced, and at the end of December all symptoms

[1] Devay, Gaz. méd. de Paris, 1843, p. 263; *cf.* Frerichs, *op. cit.*, Bd. i. p. 417.
[2] Kühne, Lehrb. d. phys. Chemie, Leipzig, 1868, p. 545.
[3] Wickham Legg, St. Bartholomew's Hospital Reports, 1873, vol. ix. p. 161; and Arch. f. exp. Path., 1874, p. 384.
[4] Golowin, Arch. f. path. Anat., 1871, Bd. liii. p. 428.

of diabetes had disappeared. The man died on March 1, and after death the common duct was found to end in a *cul de sac* in the diseased substance of the pancreas, complete obstruction having taken place.[1] The theory of this case would be that the gradual obstruction of the ducts caused the liver to cease its glycogenetic function, and the diabetes to cease; while at last the patient died from the effects of the jaundice.

Frerichs has reported a case somewhat similar in appearance. After death the head of the pancreas was found enlarged and completely obstructing the ducts. Old capillary apoplexies were found in the pons Varolii. The man was fifty years old, and began to be yellow in December. No attention seems to have been especially paid to sugar in the urine until about March 15, when it was discovered that the patient was passing sugar. The quantity was never more than 5000 c.c. in the twenty-four hours, and the specific gravity varied from 1009 to 1018. The amount of sugar was estimated by Soleil's polarisation apparatus, and varied from ·822 to 2·88 per cent. Three days before death the sugar disappeared from the urine, and after death none could be found in the substance of the liver.[2] Dr. George Harley says that on two occasions he found a little sugar in the urine of a jaundiced man,[3] but he does not give his methods, and the analyses seem not to be very accurate.

It is a matter of doubt among physiological chemists whether the bile pigments be not natural constituents of the urine. It is acknowledged generally that they may be found in the urine of healthy men in summer weather, after much drink, long fasting, &c. In the urine of many animals, such as dogs and cats, there is a substance commonly present which closely imitates the reaction of bile pigment with nitric acid.

The best way of testing for bile pigment in the urine is as follows: Ordinary nitric acid, in which some nitrous acid is nearly always present, is poured into a test-tube for the depth of an inch. On the surface of this the urine is gently poured by means of a pipette along the side of the tube, so that the two fluids may touch but not mix. A red line forms at the place of contact in every urine. If the urine contain bile pigment, however, a zone above becomes green, then blue, violet, and lastly red, the uppermost ring being green. This colour is certainly the most characteristic, and without it the reaction must always be thought dubious. In most bilious urines only the green colour is seen. The varying shades of colour answer to the stages of oxydation of the bile pigment. The reaction

[1] Bright, Med. Chir. Trans., 1833, vol. xviii. p. 3.
[2] Frerichs, *op. cit.*, Bd. i. p. 153.
[3] George Harley, Jaundice, London, 1863, pp. 78, 79.

was first noticed by Gmelin, and therefore known by his name.[1]

Carefully practised, it is by far the best test for bile pigment. Other tests have been proposed, as that of Maréchal, who mixes two or three drops of tincture of iodine with the urine, and finds an emerald-green colour given to the fluid.[2] This test, however, is lacking in delicacy. It is hard to perceive the green colour when the amount of bile pigment is small, though easy to find when the colour of the urine is so deep as not to be mistaken for anything else but bilious urine. At the beginning and end of an attack of jaundice, the iodine often fails when the nitric acid gives a distinct green. Dr. Walter Smith, however, thinks it a delicate reaction.[3]

Another test for the presence of bile pigment has been lately brought forward, *violet de Paris*, or methyl anilin violet. It appears, however, that the reaction is seen equally well with urine coloured with senna or rhubarb, or the carbolic acid treatment. It is thus worthless for the purpose for which it has been introduced.[4]

Städeler believes that the chief pigment in jaundiced urine is biliprasin, because the urine becomes green with acids and brown with alkalies, and solutions of biliprasin show like reactions.[5] On the other hand, Schwanda, from numerous observations, asserts that quantities of bilirubin may be found in jaundiced urine, and only traces of biliprasin, so that it is the bilirubin which is the chief source of colour, not biliverdin.[6] Städeler's test is not nearly so delicate as Gmelin's. It is useful, however, in determining if the high colour of the urine be due solely to drugs. Rhubarb and santonin both give a high colour to the urine, hardly to be distinguished from that of jaundice. Alkalies, however, deepen to a red the urine passed after rhubarb and santonin, but give a brown colour to the urine of jaundice.

Schwanda has attempted the estimation of the bilirubin passed in the urine, and if his results may be trusted, the amount is

[1] Rosenbach (Centralblatt f. d. med. Wissenschaften, 1876, p. 5) proposes a modification of Gmelin's test. Some of the urine is filtered through filtering paper, and then a drop of nitric acid let fall on the paper as it lies in the funnel. A fine green circle is developed.

[2] Maréchal, Journal de Pharmacie et de Chimie, 1869, 4e série, t. ix. p. 189.

[3] Walter G. Smith, Dublin Journal of Medical Science, 1876, Dec. p. 449.

[4] Rosenbach, Centralblatt f. d. med. Wiss., 1876, No. 1 ; *cf.* Gubler, Gaz. hebd., 1876, No. 21.

[5] Städeler, Annalen d. Chem. u. Pharm., 1864, Bd. cxxxii. p. 341. According to Städeler, the formula for biliprasin is $C^{32}H^{22}N^2O^{12}$; for bilirubin, $C^{32}H^{18}N^2O^6$. Maly suggests that biliprasin and biliverdin are identical (Journ. f. prak. Chem., 1868, Bd. civ. p. 32).

[6] Schwanda, Wiener med. Wochenschrift, 1865, p. 692.

extremely small, even in cases where the urine is deeply coloured. The greatest amount estimated was ·015 grm. in the twenty-four hours. In one case it was as little as ·002 grm.[1] Thus the largest amount which the urine contained was one part of bile pigment in 100,000 parts of urine. Even if these results be only approximatively true, the amount of pigment passed would be very small in comparison with the pigment excreted in health by the liver.

In some cases of jaundice, even where the urine is high coloured, and clearly contains abundance of some kind of pigment, no reaction, or no distinctive reaction, with nitric acid can be seen. Prussak believes that this absence of reaction is due to the presence of fever, and that the bile pigments are burnt off or oxydised in the blood, so that they can no longer be detected in the urine.[2] Huppert, again, says that in many cases of jaundice, no pigment but biliprasin can be found during the whole course of the disease, and it is to the absence of any other pigment but biliprasin that the trouble in applying the nitric acid test is due.[3] These two statements are not contrary, as biliprasin is but a more highly oxydised body than bilirubin; yet Städeler affirms that biliprasin gives all the reactions of bile pigments with nitric acid, saving only that the blue colour is indistinct.[4] Frerichs likewise attributes the failure of the nitric acid reaction to an oxydation either in or out of the body.[5] Nitric acid only gives in this case a red colour.

Lewin states that when jaundiced urine gives no reaction with nitric acid, the reaction may sometimes be got by cooling the urine with ice, thus causing a precipitation of the urates, collecting the urates on a filter, dissolving them in warm water, and testing this solution with nitric acid. A beautiful Gmelin's reaction may thus be seen.[6] These observations closely agree with those of Ernst Bischoff, who found a large amount of pigment thrown down with the uric acid after acidulation.

Although the presence of one element of the bile, the pigment, is plain almost to the unaided senses without chemical help, yet

[1] Schwanda, *op. cit.*, p. 989.

[2] Prussak, Centralblatt f. d. med. Wiss., 1867, p. 97.

[3] Huppert, Arch. d. Heilkunde, 1867, Jahrg. viii. pp. 351 and 476. This writer finds that the best plan of recognising even traces of bile pigment in the urine is to precipitate with milk of lime, let the mixture stand, and filter. Some of the precipitate, the size of half a hazel-nut, is put into a test-tube, and the test-tube half filled with absolute alcohol, and dilute sulphuric acid added till the fluid has a distinct acid reaction. A precipitate is slowly thrown down of a greenish colour; and if the fluid be gently warmed, the alcohol itself becomes of a greenish colour.

[4] Städeler, *loc. cit.*

[5] Frerichs, *op. cit.*, Bd. i. p. 106.

[6] Lewin, Centralblatt f. d. med. Wiss., 1875, p. 81.

it is quite otherwise with another important element of the bile, the conjugate acids. Before the days of Pettenkofer[1] and Strecker[2] it was well-nigh hopeless to search for the crystalline elements of the bile in the urine. Nevertheless some observers thought they had detected them both in the blood and urine of jaundice. After Pettenkofer, none succeeded[3] in finding the bile acids in the urine, notwithstanding many attempts by observers in every way worthy of credit. It was therefore assumed that the bile acids were absent from the urine in jaundice, and it became a matter of importance to explain this absence. Frerichs and Städeler found that after the injection of colourless bile into the veins of animals, the urine was tinged of a high colour, and gave a well-marked reaction with Gmelin's test, showing the presence of bile pigment,[4] and yet no bile acids could be found in this urine. The same observers afterwards found that when sulphuric acid was allowed to act upon the bile acids, a coloured substance was got, which gave with nitric acid the same play of colours as bile pigment.[5] Here, then, seemed a clear way out of all difficulties. The bile acids do not appear in the urine, because they are oxydised in the circulation into bile pigment.

But in 1858, two years after the publication of this last discovery, Hoppe detected in jaundiced urine a notable quantity of choloidinic acid, an acid derived from the bile acids, but containing no nitrogen, and yet giving Pettenkofer's reaction.[6] Later on he demonstrated the presence of nitrogenous derivatives from the bile acids,[7] and last of all, the presence of glycocholic and taurocholic acids themselves, together with cholalic acid, in the urine of jaundice.[8]

This discovery of the bile acids or their derivatives in the urine was soon after confirmed by Kühne,[9] and although at first doubted by some,[10] yet these acids have been so repeatedly found by those who have used Hoppe's improved method of searching for them, that their presence in the urine of jaundice is now generally admitted.

[1] Pettenkofer, Annalen d. Chemie u. Pharm., 1844, Bd. lii. p. 90.

[2] Adolph Strecker, ibid., 1848, Bd. lxv. p. 1.

[3] Lehmann, however, says (Lehrb. d. phys. Chemie, Leipzig, 1853, 2te Aufl., Bd. i. p. 122) that he has found substances which give Pettenkofer's reaction in the blood and urine of persons whose livers did not seem immediately concerned in the disease.

[4] Frerichs, *op. cit.*, Bd. i. p. 404. I have elsewhere remarked upon the incorrectness of this observation.

[5] Frerichs and Städeler, Arch. f. Anat. Phys., &c., 1856, p. 55.

[6] Hoppe, Arch. f. path. Anat., 1858, Bd. xiii. p. 101.

[7] Idem, ibid., 1862, Bd. xxiv. p. 1.

[8] Hoppe-Seyler, Centralblatt f. d. med. Wiss., 1863, p. 337.

[9] Kühne, Arch. f. path. Anat., 1858, Bd. xiv. p. 315.

[10] Neukomm, Arch. f. Anat. Phys., &c., 1860, p. 368.

It may be doubted if the total amount of bile acids in the urine have ever been properly estimated. It is, at all events, so small, that Pettenkofer's test applied directly to the urine gives no certain result. Hoppe in a case of acute yellow atrophy found about ·03 per cent. of cholonic acid;[1] and Ernst Bischoff gives as the result of three estimations, ·02, ·04, and ·05 per cent. This latter observer looks upon ·3 grm as the maximum of bile acids excreted in jaundice by the urine during the twenty-four hours,[2] a result which agrees generally with that of Hoppe-Seyler.

If Pettenkofer's test be applied directly to many urines, a reaction is got which may readily be confounded with that seen in a pure solution of bile acids. Sulphuric acid by itself, without the presence of sugar, will often beget a fine red or purple colour, by inexperienced eyes easy to be mistaken for Pettenkofer's reaction. This is most often the case in a high-coloured or albuminous urine, and not uncommon in perfectly natural urine. Nothing, therefore, but disappointment and utterly untrustworthy results can be looked for from the direct application of Pettenkofer's test to jaundiced urine. It is a method which should never be adopted. The bile acids must be separated out from the jaundiced urine before their presence can be definitely asserted.

To detect the bile acids in jaundiced urine a long preparation is needed. In Hoppe's first experiments, the urine was boiled with milk of lime, then filtered, and again boiled with hydrochloric acid. This undoubtedly causes a large destruction of the bile acids, and the method which is now recommended is as follows: Precipitate the bile acids with acetate of lead from the urine made faintly ammoniacal; wash the precipitate with a little water, boil it with alcohol, and filter hot. The lead salts of the bile acids are soluble in hot alcohol; a few drops of soda solution should next be added, and the whole evaporated to dryness. This should now be boiled with absolute alcohol, filtered, and evaporated to a small volume, and then poured into a great excess of ether. In this way the bile acids may often be crystallised out.[3]

This process is troublesome, and ill fitted for the wants of the practitioner. Modifications of it have been proposed. A rapid way of testing has been set forth by Strassburg. A piece of filtering paper is dipped into the urine to be tested, to which a little cane-sugar has been added. The paper is then dried, and on the

[1] Hoppe, Arch. f. path. Anat., 1862, Bd. xxiv. p. 1.
[2] Ernst Bischoff, Zeitschrift f. rat. Med., 1864, Bd. xxi. p. 140.
[3] Hoppe-Seyler, Handb. d. phys. u. path. chem. Analyse, Berlin, 1868, 2te Aufl., p. 274.

centre is let fall a drop of concentrated sulphuric acid. Around the drop of acid a beautiful violet colour forms.[1] In my own hands, however, this test has proved very unsatisfactory. I have seen a purplish colour, identical with that seen with jaundiced urine, arise with natural urine, or with urine from patients not known to have anything amiss with their livers.

By some observers the bile acids are now looked upon as natural constituents of the urine. Naunyn collected large quantities of natural urine, and was able to detect an appreciable amount of bile acids by the process of precipitation with lead.[2] Fudakowski [3] and Vogel [4] have repeated and confirmed these observations, but their value has been doubted by Wolff.[5]

The presence of the bile acids in natural urine, if granted, is probably due to an absorption of the bile acids from the intestine, a very small quantity passing through the liver into the general circulation.

It has, however, been asserted of late that in long-continued jaundice the liver loses the power of secreting the bile acids, and thus none appears in the urine. Golowin found none either in men or animals.[6] Huppert found the amount much decreased towards the end of a case of acute yellow atrophy,[7] and Dr. Pye Smith could find no bile acids in 14,000 c.c. of the urine of a patient jaundiced a long time, probably from gall stones.[8]

Neither dyslysin, nor glycocol, nor taurin has ever been found in the urine.[9] The first is a product of the decomposition of the bile acids, the two last are important constituents of glycocholic and taurocholic acid.

The amount of urea excreted in jaundice has of late years been more studied than formerly. For it would seem likely, if Meissner's theory of the formation of urea in the liver be true, that the amount of urea in jaundice would be decreased, and on these grounds: if albuminous substances be split up in the liver

[1] Strassburg, Arch. f. d. ges. Phys., 1871, Bd. iv. p. 461.

[2] Naunyn, Arch. f. Anat. Phys., &c., 1869.

[3] Fudakowski, Centralblatt f. d. med. Wiss., 1869, p. 129.

[4] Vogel, Schmidt's Jahrbb., 1872, Bd. clvi. p. 36. Dragendorff was able to get ·7 or ·8 grm. of bile acid from 100 litres of urine.

[5] F. A. Wolff, Zur Pathologie des Ikterus, Diss. Inaug, Königsberg, 1869, abstract in Henle and Meissner's Bericht f. 1869, p. 196.

[6] Golowin, Arch. f. path. Anat., 1871, Bd. liii.

[7] H. Huppert, Arch. d. Heilk., 1864, Bd. v. p. 255.

[8] Pye Smith, Trans. of Path. Soc. of Lond., 1873, vol. xxiv. p. 251. He does not, however, give the method followed.

[9] Frerichs, *op. cit.*, Bd. i. p. 102, note. Ernst Bischoff, Zeitschrift f. rat. Med., 1864, III. Reihe, Bd. xxi. p. 146. Kühne, Arch. f. path. Anat., 1858, Bd. xiv. p. 322. I attribute no value to the finding of taurin in the urine by Raczieiewsky (Leyden, Beiträge zur Path. des Icterus, Berlin, 1866, p. 33), as the microscope only was used, and no chemical test.

into glycogen, bile acids, and urea, then, if the formation of glycogen cease in jaundice, the formation of urea should cease likewise. I have shown that within a few hours after the ligature of the bile ducts glycogen ceases to be found in the liver,[1] and this statement has been confirmed by Von Wittich.[2] If, then, in jaundice no glycogen be formed by the liver, it would seem likely that the urea and bile acids would likewise cease to be made. Now Golowin seems to think that in long-continued jaundice the bile acids no longer appear in the urine;[3] and the fluid found in the dilated gall ducts after death certainly gives no reaction with Pettenkofer's test. But these are all cases in which the jaundice has been long-continued, and it is denied by none that early in jaundice bile acids may readily be found in the urine and in the biliary passages. So that, notwithstanding Golowin's statements, it would seem likely that in jaundice, at all events early in the disease, the bile acids continue to be formed. As to the urea, much doubt formerly existed whether the amount were unusually low or natural. Becquerel analysed the urine of three jaundiced patients. In the first, a man, he found that 17·923 grm. were excreted in the twenty-four hours; in the second, a man jaundiced, after a violent dispute, he found only 4·037 grm.; and in a third, a pregnant woman, markedly jaundiced, 7·933 grm.[4]

A. Vogel found in a case of cancer of the liver with jaundice that the urea varied from 6·75 to 9·5 grm., a very low amount.[5] Kölliker and Müller found in a woman, aged twenty-five, with simple jaundice, that the mean of urea during the jaundice was 20 grm., during convalescence 19 grm., and when in health 26 grm.[6] Leyden, on the other hand, has recorded a case in which the urea once rose to 50 grm. in the twenty-four hours; three times it is noted between 30 and 40 grm., and it never seems to have fallen below 23 grm.[7] There seems to have been in this case some relation between the amount of water excreted and the urea, the highest readings of urea corresponding to the highest readings of water and *vice versa*. Bouchardat has recorded an extraordinary case in which the readings of the urea were enormously high. A man with simple jaundice, following a

[1] Wickham Legg, St. Bartholomew's Hospital Reports, 1873, vol. ix. p. 161.
[2] Von Wittich, Centralblatt f. d. med. Wiss., 1875, p. 291.
[3] Golowin, Arch. f. path. Anat., 1871, Bd. liii. p. 433.
[4] Becquerel, Séméiotique des Urines, Paris, 1841, pp. 413 and 393. There is an old analysis of Braconnet in which he says he found more urea and less phosphate of lime than usual (Journal de Chimie méd., 1827, t. iii. p. 480).
[5] A. Vogel, Zeitschrift f. rat. Med., 1854, Bd. iv. (neue Folge), p. 391.
[6] Kölliker and Müller, Verhandlungen d. phys. med. Gesellschaft in Würzburg, 1856, Bd. vi. p. 497.
[7] Leyden, Beiträge zur Path. d. Icterus, Berlin, 1866, p. 205

sudden great joy, passed 3750 c.c. of urine in the twenty-four hours: the specific gravity was 1031. The amount of solid matter passed was 220·87 grm.; the urea, 133·6 grm. The jaundice next day was less, and the urea only 89·18 grm. On the third day the jaundice was still less, and the urea only 46·94 grm.[1] The same observer has recorded another instance of uncomplicated jaundice in which the urea ruled high, 57·2 grm. in the twenty-four hours.[2]

Genevoix has published seven cases of simple jaundice, in which, during the height of the jaundice, the urea was markedly increased, falling to the natural standard as the jaundice disappeared. In three of these cases the urea was over 66 grm. in the twenty-four hours; in two, 55 grm.; and in the remaining two, 45 grm. When convalescence was established the amount fell to 23 or 24 grm. There was no rise of temperature.[3]

Brouardel has analysed the urine of three cases of simple jaundice and found no increase or decrease of urea. His figures vary from 26 grm. to 32·8 grm. daily.[4] Julius Jacobs has estimated the urea in three cases: in one, complete obstruction of the ducts from a mass of cancer was present, and the amount of urea was on an average of seven days 56·525 grm. in the twenty-four hours. In two other cases it was 28·249 grm., and 27·527 grm. on an average.[5]

In three cases of jaundice with clay-coloured stools, J. C. Lehmann found the amount of urea decreased during the obstruction to the gall ducts, while it became greater as soon as the obstruction was removed. In the third case, the amount of urea was increased.[6] Lehmann explains the diminution by supposing an imperfect digestion of the albuminous matters, probably because the parapeptones are not thrown down. What, however, would be the explanation of the case in which the urea was increased in amount? Dr. A. W. Foot also found no decrease of the amount in one of his cases.[7] Nor did Dr. George Harley, in a case of jaundice lasting eighteen months, find any decrease until a fortnight before the death of the patient.[8]

[1] Bouchardat, Annuaire de Thérapeutique, 1846, p. 328.

[2] Idem, ibid., 1869, p. 237.

[3] Genevoix, Essai sur les Variations de l'Urée et l'Acide urique dans les Maladies du Foie, Paris, 1876, p. 63.

[4] Brouardel, Archives de Physiologie, 1876, p. 416.

[5] Julius Jacobs, Arch. f. path. Anat., 1877, Bd. lxix. p. 487.

[6] J. C. Lehmann, Ugeskr. for Läger, 1868, 3 R. vi. No. 24–26, abstract in Virchow's Jahresb. f. 1868, Bd. ii. p. 143.

[7] A. W. Foot, Dublin Journal of Medical Science, May 1876. The amount excreted was 439·687 grains.

[8] George Harley, Jaundice, its Pathology and Treatment, London, 1863, p. 74, *et seq.*

I have made observations upon the urea in ten cases of jaundice. The two cases in which the largest amount of urea was passed were likewise those in which the largest amount of water was passed, and in which the jaundice had been longest and most intense. The mean of the urea in these two cases was 28·387 and 29·574 grm. The lowest amount of urea was met with a case of somewhat uncertain diagnosis, in which the jaundice had lasted about two months, and the stools were of a light brown. The mean of five consecutive observations was 15·975 grm. In two other cases of jaundice from cancer of the pancreas and liver, the mean amount of six observations was 18 grm. In the remaining five cases, the means varied from 19·8 to 27·28 grm.[1]

Upon a review of these cases, it seemed to me that the amount of urea was the greatest in those patients whose general health was the best, or who were allowed the most generous diet. I do not think there is any proof that obstruction of the gall ducts causes a lessened excretion of urea, although the earlier recorded cases may at first seem to encourage such a belief. The estimations of the urea in simple jaundice lately undertaken by the two French observers, apparently suggested by Charcot, seem to destroy such a notion. It is, however, a hard matter on any theory to explain the great increase of urea which Bouchardat and Genevoix have noticed in their cases. I must be content to note that in jaundice the urea may be low, natural, or greatly in excess.

There are not many observations on the amount of uric acid daily excreted in jaundice. Becquerel found in one of three cases the uric acid in excess, as much as 1·153 grm. in quantity,[2] but in the remaining two it was natural. Kühne seems to think that it is always much increased.[3] And, on the other hand, Ernst Bischoff, whose observations lay claim to far greater accuracy than those of Kühne, found the uric acid not increased.[4] He explains Kühne's statement by saying, that when jaundiced urine is acidulated with hydrochloric acid, an amazing quantity of crystals is thrown down. If these crystals be collected on a filter, they dissolve when well washed with water, and the wash-water gives an intense reaction with nitric acid, so that it is highly probable that the crystals are mostly made up of bile pigment. Dr. A. W. Foot has, however, in one case found an increase of the uric acid, the amount being 27·13 grains.[5] Dr.

[1] Wickham Legg, Med. Chir. Trans., 1876, vol. lix. p. 149.
[2] Becquerel, Séméiotique des Urines, Paris, 1841, pp. 393 and 413.
[3] Kühne, Arch. f. path. Anat., 1858, Bd. xiv. p. 320.
[4] Ernst Bischoff, Zeitschr. f. rat. Med., 1864, Bd. xxi. p. 151.
[5] A. W. Foot, *loc. cit.*

George Harley found ·511 and ·266 grm., and lastly none, in three analyses made one after another in a case of permanent obstruction.[1] In these two last cases, the uric acid seemed to diminish or increase *pari passu* with the urea. The like was noticed by Genevoix, who found that the uric acid was increased in seven cases of simple jaundice with the urea, and that as the jaundice diminished, the urea and uric acid decreased likewise.[2] Julius Jacobs did not find the uric acid diminish or increase with the urea; his lowest reading is ·997 grm., while the urea is 56·525 grm., while in the two other cases the uric acid is 1·0038 and 1·466 to 28·249, and 27·527 of urea.[3]

Kühne stated, in the midst of his essay on jaundice, that no hippuric acid could be found in the urine of jaundiced men; and that after benzoic acid, or its soda salts, had been given to jaundiced patients, no hippuric acid appeared in the urine, but only unchanged benzoic acid.[4] It is well known that if benzoic acid be given to men in health, an equivalent quantity of hippuric acid appears in the urine. Several observers have repeated Kühne's experiments, but all with a negative result.[5] The source of error in Kühne's work seems to be that the urine was not examined while quite fresh, and that the hippuric acid was changed again into benzoic. The hippuric acid may in all cases be found in jaundiced urine, if looked for soon after the urine has been voided, and by the method recommended by Schultzen.

Less attention has been paid to the chlorides than to the uric acid. A. Vogel found them in his case greatly decreased. They were only ·16 to ·3 grm. in the twenty-four hours.[6] In nine cases of jaundice in which I estimated the chlorides, I found a tolerably close relation to the urea, the chlorides being equal to about one half of the urea in six out of the nine. In two of the three cases the chlorides were decreased; they were both cases of cancer of the pancreas; the chlorides were about 6 grm. to 18, and 22 grm. of urea. In the remaining case, of doubtful diagnosis, of which I have spoken before while treating of urea, the chlorides were estimated on five following days. The first day the urea was 18 grm. to 21 grm. of chlorides; the second

[1] George Harley, *loc. cit.* [2] Genevoix, *loc. cit.*

[3] Julius Jacobs, Arch. f. path. Anat., 1877, Bd. lxix. p. 487.

[4] Kühne, Arch. f. path. Anat., 1858, Bd. xiv. p. 319.

[5] Folwarczny, Zeitschr. d. k.k. Gesellschaft d. Aerzte zu Wien, 1859, p. 225. Schultzen, Arch. f. Anat. Phys., &c., 1863, p. 204. Neukomm, Frerichs' Klinik d. Leberkrankheiten, Bd. ii. p. 537. Huppert, Arch. d. Heilkunde, 1865, p. 93. Horace Chase, Arch. f. Anat. Phys., &c., 1865, p. 392. In two of Chase's cases, however (dogs in whom the bile ducts had been tied), he could not find hippuric acid after the benzoate of soda had been given by the mouth.

[6] A. Vogel, *loc. cit.*

day it fell to 17 grm. of urea and 14 grm. of chlorides, and maintained this proportion through the rest of the observations.[1] I think the first day's very high reading may be caused by some error, or perhaps accidental admixture, seeing the abundance of chloride of sodium in the world.

Julius Jacobs in his three cases found an increase in the chlorides in nearly all—10·717 grm. in the first, 17·818 in the second, and 14·366 in the third.[2]

There are also few observations upon the amount of sulphates excreted in jaundice. Kölliker and Müller found in an artificially jaundiced dog that more sulphuric acid was excreted than in a sound dog; a fact which seems to them to show that the sulphur of the taurocholic acid passes into the urine. In a case of simple jaundice in a woman aged twenty-five they found, however, the sulphuric acid, as well as the urea, diminished.[3] It seems doubtful, however, if the decrease of the sulphuric acid be not due to the general state of the patient. Julius Vogel gives as evidence of the state of chronic disease, that in a jaundiced man the amount of sulphuric acid excreted in the twenty-four hours was only 1·4 grm.[4] Ernst Bischoff likewise found the amount of the sulphates diminished, clearly he thinks owing to the decreased diet.[5] But he also noticed another more interesting fact. It is well known, from Voit's observations, that the sulphuric acid in the urine does not account for all the sulphur, and that a body containing sulphur is present, probably derived from taurin. Now the amount of sulphur, not oxydised, present in the urine of jaundice is much in excess of what is natural, and the amount of excess is exactly equal to the amount of sulphur in the fæces which Bischoff calculates should be passed during health. So that Bischoff is inclined to think that the same amount of taurin is secreted by the liver in jaundice as in health, and that therefore the same amount of taurocholic acid is secreted by the liver in jaundice as in health; a conclusion with which I am not prepared to agree.

I have been able to find only a few observations upon the amount of phosphates excreted. Von Haxthausen found that a jaundiced man, thirty-six years of age, excreted as a mean of twenty-four observations 1·93 grm. of phosphoric acid in the twenty-four hours; the lowest being 1·364 grm., the highest

[1] Wickham Legg, Med. Chir. Trans., 1876, vol. lix. p. 149.
[2] Julius Jacobs, Arch. f. path. Anat., 1877, Bd. lxix. p. 487.
[3] Kölliker and Müller, Verhandlungen d. phys. med. Gesellschaft in Würzburg, 1856, Bd. vi. p. 492.
[4] Neubauer and Vogel, Anleitung zur qual. u. quant. Analyse des Harns, Wiesbaden, 1863, p. 327.
[5] Ernst Bischoff, *op. cit.*, p. 150.

3·508 grm. The excretion of phosphates appears to be less before noon than after noon, and more in the night than during the day.[1] Dr. A. W. Foot found, as a result of one observation upon a jaundiced woman, that the amount of phosphoric acid excreted was 58·625 grains in the twenty-four hours.[2] Julius Jacobs in one case found 1·183 grm. in the twenty-four hours.[3]

Except in cases of acute yellow atrophy, leucin and tyrosin are not present in the urine of ordinary jaundice. Great caution should be used in asserting that they are present in the urine in any case. The mere finding of a sediment in the urine which shows under the microscope crystals like those of leucin and tyrosin is no proof whatever that these bodies exist in the urine. Chemical tests must be applied to the bodies crystallized out from the urine, otherwise no reliance can be placed upon the statement that they are present.[4]

Budd,[5] Murchison,[6] and Pouzol[7] have noticed the occasional presence of casts of the renal tubes in jaundiced urine. Nothnagel looks upon their presence as a constant phænomenon in all cases of intense jaundice, whatever the cause may be. He thinks casts in the urine are more often present than itching of the skin; they are usually hyaline, and are then not coloured yellow; epithelial casts are the next commonest, and are deeply yellow coloured; very rare are the so-called fibrinous casts. In two-thirds of the cases albumen was not found by the ordinary tests, nor any of its modifications. The cases observed were catarrhal jaundice, jaundice from gall stones, compression of duct by new growth, pyæmia, and bilious pneumonia.[8] Dr. Finlayson, in some observations upon the presence of casts in urine not containing albumen, says that tube casts are almost invariably found in marked cases of jaundice, and this as a rule occurs without albuminuria,[9] so that this observer agrees with

[1] Von Haxthausen, Schmidt's Jahrbb., 1863, Bd. cxx. p. 160. Braconnet (Journal de Chimie méd., 1827, t. iii. p. 480) says he once found the phosphate of lime decreased.

[2] A. W. Foot, *loc. cit.*

[3] Julius Jacobs, Arch. f. path. Anat., 1877, Bd. lxix. p. 487.

[4] A friend of mine, the late Dr. C. E. Squarey, well known for his experience in the analysis of the urine, was able to get abundant evidence with the microscope of the presence of leucin and tyrosin in nearly all cases of jaundice. Pushing his results further, he got the same shaped crystals in all kinds of urine, diseased and healthy, but he was in no case able to produce any evidence of the presence of these bodies in the urine of jaundice, or of any other disease, when using chemical tests.

[5] Budd, On Diseases of the Liver, London, 1857, 3d ed., p. 288.

[6] Murchison, *op. cit.*, p. 287.

[7] Pouzol, *op. cit.*, p. 58.

[8] Nothnagel, Deutsch. Arch. f. klin. Med., 1874, Bd. xii. p. 326.

[9] Finlayson, British and Foreign Med. Chir. Rev., 1876, vol. lvii. p. 184.

Nothnagel. I have been unable, however, in many case in which I have carefully looked for casts to find these bodies; nor can I agree with the statement that jaundiced urine is very commonly free from albumen. Dr. Finlayson appears to attribute the presence of casts in the urine to the irritation caused by the passage of pigment.

BRIEF NOTES

ON

THE OUTBREAK OF SCURVY IN THE LATE ARCTIC EXPEDITION.

BY

HARRY LEACH.

This is a well-worn subject. Some doubtless think that it is well-nigh worn threadbare, and that little or nothing more can be said about a disease that, except in times of war or famine, ought to be seldom seen in civilised communities. The disease is indeed so rare in our public hospitals that many students pass through their entire curriculum without seeing a case, and there are some middle-aged, if not elderly, practitioners in the kingdom, who would probably fail, at all events at first, to diagnose the malady. The events of the past eighteen months have, however, brought scurvy again to the front in an unpleasantly prominent manner. An Arctic Expedition started from our shores in the summer of 1875, bountifully and even lavishly furnished with all the varieties of food, clothing, and other material that science and art could suggest. For the sympathies of the public were with the explorers, and they, as well as the Government, gave helping hands without stint. The expedition returned home in the autumn of last year, and before the news of the arrival of the ships had been announced many hours, accounts came that the men, both of the "Alert" and the "Discovery," had been severely affected with scurvy. Rumours soon gave place to certainty, and it was found that sixty of the men had been attacked, and that four had died. A great deal of natural indignation was exhibited and expressed, for it was felt either that our old antiscorbutics, lime and lemon juice, were no longer to be trusted, or that some neglect had occurred, for which either

the executive or the medical officers must be held responsible. Much newspaper war, good, bad, and indifferent, was waged about the matter, and our, so to speak, "antiscorbutic beliefs" appeared in danger. The Admiralty were ultimately induced by the force of public opinion to institute a special inquiry into the circumstances attending the outbreak. The evidence given before the Committee was of a very exhaustive character, and the Blue-Book containing it undoubtedly furnishes the most complete scientific, as well as practical, essay on scurvy that exists at the present time. The writings and experiences of all good authors are utilised, and in some cases summarised by the writers themselves, many of whom gave evidence before the Committee. But this Blue-Book is a very cumbrous mass of literature, and is very voluminous. It appears, therefore, that some good may be done by endeavouring to put this large mass of evidence into a small compass, and to compare it impartially with events, and with the literary labours of those who have written on the subject during the past half century, so as to see whether or not the practical conclusions arrived at by the Committee are in accord with the majority of scientific opinion on the subject generally, and as to this outbreak in particular.

The total complement carried in the "Alert" and the "Discovery" consisted of 122 men of all ranks, chosen with great care—the standard of age being between twenty-five and thirty-two, and the standard of height from 5 ft. 5 in. to 5 ft. 8 in. The diet scale on board ship, a copy of which is appended, was stated by all witnesses examined on this point to be a good diet, and quite sufficient for the purpose. Samples of the articles brought home were examined by the Committee, and some were analysed, and the quality of all was found excellent, except the beef, which was described as "very salt." It was proved that the men remained in good health, and even gained weight, while subsisting upon it, and scurvy did not occur among any of the men who had been restricted to this class of diet during the winter, with the exception of one case on board each of the two ships.

The expedition started in the summer of 1875. The winter was spent to a great extent in darkness, for the sun was not seen from the "Alert" for 142 days, and from the "Discovery" for 138 days. The men were during this time breathing an exceptionally impure atmosphere, the space allotted to each man in the "Alert" being 107, and in the "Discovery" 140 cubic feet. Various analyses of the air made showed in the "Alert" an average of 0·3314 per cent. of carbonic acid, or more than five times as much as the maximum quantity in a sufficiently pure

air; and on the "Discovery" 0·415 per cent., or nearly seven times as much as the maximum quantity. A great amount of dampness prevailed almost constantly between decks, because the air was not renewed sufficiently, the ventilating arrangements being (perhaps unavoidably) defective; and there was also a deficiency of fresh meat, the dietary of the crew of the "Discovery" having rations of it on 53 days, and the men of the "Alert" on only 14 days. The crews were also necessarily confined in the lower deck during the greater part of the twenty-four hours. The conditions above mentioned may properly be classed, or at all events suggested, as predisposing causes of scurvy. But, with a single exception, all remained free from the disease throughout the winter. The exception was in the person of a cooper, who, according to the report of Dr. Ninnis, surgeon of the "Discovery," had been a hard drinker, disliked preserved meat and vegetables, and surreptitiously abstained from them and the fresh beef and lime-juice, whenever he had an opportunity. For several weeks previous to the departure of the sledging parties, a double allowance of lime-juice was served out to the men, and was in most cases taken by them. Hence it appears clear that at the end of the winter, in spite of the very unfavourable conditions epitomised above, the health of the men was good. The minimum temperature of the outer air for twenty-four hours during this time, as registered on the "Alert," was 70° 31′ below zero, and that of the air between decks 34° Fahr.

In considering the requirements of sledge travelling, weights are of course a primary consideration. As the weight of many articles of food may now be greatly reduced (without any apparent impairment of their nutritive properties) by the abstraction of water, which is returned in cooking, condensed foods are of course largely used in such journeys. The sledging diet adopted on this occasion is appended (see p. 40), and it will be seen that when the foods used on board ship and the sledge dietaries are reduced to the state of water-free solids and compared, the board-ship dietary is really less liberal than the sledging dietary, the former containing 27·44 ounces of water-free solids, while the latter contains 35·65 ounces. It was also estimated by one of the witnesses examined before the Committee that 340 foot tons of productive work could be done without loss of weight on the former diet, and as much as 585 foot tons on the latter. The following table, compiled by the same witness (Dr. De Chaumont), contrasts the more important nutritive constituents of the ship and sledging dietaries.

SHIP DIET.

Constituents.	Ounces Water Free.	Ratio of Albuminates.
Albuminates . . .	4·76	1·00
Fats	4·18	0·88
Carbo-Hydrates . . .	16·84	3·54
Salts (Mineral) . . .	1·66	0·35
Total	27·44	

SLEDGE DIET.

Constituents.	Ounces Water Free.	Ratio of Albuminates.
Albuminates . . .	7·99	1·00
Fats	11·65	1·43
Carbo-Hydrates . . .	15·22	1·90
Salts (Mineral) . . .	0·79	0·10
Total	35·65	

The report suggests whether this excess of fat, taken in conjunction with the somewhat large total quantity of nutritive material, renders this diet a sufficient one for men engaged in severe physical work in extreme cold. But it appears that the same dietary succeeded well in former expeditions, where the cold experienced did not differ very materially from that undergone by the recent expedition, but the amount of physical exertion undergone on the recent expedition appears to have been comparatively much greater. The average work done daily per man was equivalent to 534 foot tons, or less than that estimated as the productive work to be got out of the sledge diet. But the evidence shows that the men were sometimes not able to take their full rations, and it is also probable that the physical work was underestimated, as the difficulties encountered by the men were altogether exceptional.

Having thus endeavoured to summarise all the predisposing causes of scurvy in this case, let us turn again to a further and general comparison of the ship and sledge dietaries. The report says, "The ship dietary provides for a liberal allowance of vegetable food. Preserved vegetables, including potatoes, were issued on board in rations of eight ounces once or twice a week, and of four ounces four times a week; compressed vegetables in rations of two or six ounces respectively twice a week; while vegetables were present in several of the tinned meats." It is to be remarked particularly that analyses of the preserved vegetables showed that all retained their nutritive properties unim-

paired except the dried compressed vegetables, which were found deficient in mineral matter, "especially of the saline material termed alkaline phosphate." There was, besides, a daily ration of one ounce of lime-juice, which was increased to two ounces on board the "Alert" during the month of March. In the sledge scale of rations vegetables are represented by only two ounces of preserved potato, which nearly all the witnesses very decidedly deemed insufficient. The scale of this article for convicts includes (or did include) four ounces per head per day; Messrs. Green's seamen's scale is four ounces weekly; Messrs. Wigram's scale, eight ounces weekly; and the Emigration scale, eight ounces weekly, besides preserved meats, peas, currants or raisins, and a daily ration of one ounce of lime or lemon-juice. Other authorities agree that not less than four ounces is a proper ration of this article under any circumstances, so that the antiscorbutic value of the quantity given must have been exceedingly small. It will be seen that a small quantity of onion powder and curry paste ($\frac{1}{8}$th ounce of each) was included in the sledge dietary. Such were the dietary conditions under which the sledge expeditions were organised, and fifty-eight cases of scurvy out of the sixty occurred in men who had been deprived for a greater or less period of adequate vegetable material in their food. Practically they had lived without vegetables, or its substitute, lime-juice. Some of this juice was indeed carried by two or three of the minor sledge parties, especially by those which started from the ships towards the latter part of the sledging season, or visited the depots where lime-juice was stored. But, as we know, it was not given systematically in any case, and the sledge parties which suffered most severely from scurvy were not provided with it. It appears, therefore, to be proved conclusively that, as regards the circumstances of this expedition, the one invariable antecedent to scurvy existed, viz., the absence of vegetable food. The late Dr. Parkes, who wrote on scurvy in the "British and Foreign Medico-Chirurgical Review" in 1848, and whose article on the subject has during the last thirty years never been surpassed, says in conclusion, "True scurvy is caused by a deficient supply of the organic vegetable acids or salts of fresh vegetables." Budd, in the "Library of Medicine," appears to show the same primary cause, which is in like manner worked out by Buzzard in Reynold's "System of Medicine," as well as in the last edition of Cooper's "Surgical Dictionary," and Dr. Ralfe has lately published some valuable notes on the general pathology of scurvy, which will be found in "The Lancet" of June and July 1877, all of which tend to the same result. The siege of Paris, the Irish famine, and the early events of the Crimean

War all gave terrible examples of scurvy with the same antecedent, a dearth of vegetable food. And writing at this date (September 1877), we may unhappily predicate with almost certainty the advent and speedy spread of the disease among the Eastern armies in Bulgaria and Asia Minor, soon after the grape crops are exhausted. In quoting examples of this antecedent to scurvy, I have confined myself to those of comparatively recent date. But the detailed practical experiences of Captain Cook, Lind, Woodall, Trotter, Gilbert Blane, and others, all point to the same single condition among others that invariably existed.

We are rightly reminded, however, in the Arctic report, that the converse does not always hold good, inasmuch as scurvy occasionally occurs where fresh vegetable food has been eaten. In such cases, however, it is probable that other abnormal causes were in operation, preventing the vegetable variety in common with the other kind of food taken, from being properly digested and assimilated.

The invariable antecedent cause appears indeed now to be so well established, that it is somewhat unfortunate to find in official reports hypotheses so palpably wide of the mark. A large Blue-Book was published under the auspices of the Board of Trade last year (Parl. Paper 117 of 1876) containing particulars as to inquiries on outbreaks of scurvy in merchant ships. In one or more cases the medical inspector attributed the outbreak to the eating of "scouse," which is a mixture of cook's fat or slush, biscuit, and water, to which minced salt meat is usually added. The inspector on these occasions would find it difficult to prove that any of the articles above mentioned were directly or indirectly scorbutic in their effects.

Some three years ago an ingenious paper was published in Paris by M. Villemin, who attempted to prove that scurvy was purely a contagious disease. These views, which received no sort of confirmation from any other authority, were successfully combated at the time by M. Alfred le Roy de Mericourt, medical chief of the French navy, and have since been ably contested by M. J. B. Foussagrives (who now holds the chiefship) in a very able treatise on Naval Hygiene that appeared in the spring of the current year. Among points worthy of notice was the fact that the officers of the expedition suffered less than the men. Sir George Nares says in his evidence, "No case of scurvy (among the officers) was actually on the sick list on board the 'Alert.' Lieutenant Beaumont of the 'Discovery' suffered from scurvy." This fact entirely agrees with experience gleaned in the mercantile marine, where the masters and those officers who

live aft (including usually the cook and steward) enjoy an immunity, this immunity being probably due to advantages in way of surroundings and less labour and exposure, as well as a more varied diet.

It has been often stated, but incorrectly, that the Esquimaux do not suffer from scurvy. There is no doubt that they are often affected, and that scurvy has been prevalent in Greenland for many years. Hence the inspector of North Greenland, residing at Disco, was very glad to receive a present of lime-juice made to him by the commander of the expedition.

Bad water, whether brackish or otherwise foul, and condensed water, have sometimes been cited, and, indeed, may perhaps be still classed as a predisposing cause of scurvy. It was, however, brought out in evidence, that the water used in the "Alert" was exceptionally good, as fresh-water ice was always obtainable, instead of snow, which is usually more or less brackish in the Arctic Seas. There is little doubt that in some severe outbreaks of scurvy that occurred in the mercantile marine about twenty years ago, bad or condensed water might be classed as a predisposing cause, notably among the vessels trading between Aden and Callao.

One of the most important questions raised, and, as it is hoped, well-nigh disposed of, in the course of this inquiry, was the value of alcohol as an antiscorbutic. The physiology of the subject entirely negatives the supposition. As is observed in the "Lancet" of 2d December 1876, alcohol is "well known to paralyse the vaso-motor centres, and, as a consequence, to cause dilatation of the capillaries, allowing a freer current of blood to pass through them, which leads again to undue loss of heat by radiation, evaporation, and conduction, by no means compensated for by its capability of undergoing combustion." The practical results of the Arctic Expedition go very far to prove the physiological aspects of the question. The report says, "It is a significant fact in the history of the recent expedition, that the first two cases of scurvy occurred in men who were addicted to an immoderate use of alcohol, and who had not been exposed to the deteriorating conditions that existed during sledge travelling." In former Arctic Expeditions scurvy occurred in men who indulged to excess in alcohol, although at the time the disease was not prevalent among the rest of the crew. The officers of the Hudson's Bay Company rarely drink alcohol in any form, and enjoy an almost complete immunity from scurvy. A ration of $2\frac{1}{2}$ ounces of rum was served out daily to each man on board ship during the recent expedition, and the same quantity to the sledging parties. The great majority of the Arctic voyagers

examined were agreed that tea was more acceptable than the ration of rum during the working hours, but most of the scientific as well as the other witnesses urged that a small quantity of rum should be carried for issue at night. We may, however, safely dismiss alcohol from the list of direct or indirect antiscorbutics, as in this case the ration of rum was recommended merely as a "comforting" agent, but by no means as a prophylatic against scurvy.

Dirt is said to be a predisposing cause of scurvy. But in this expedition, it appears that a fair amount of cleanliness was practised, and Dr. Colan, the surgeon of the "Alert," says, that "with most men the examinations as to cleanliness were satisfactory."

Idleness is also sometimes classed as a predisposing cause. But it does not appear from the evidence that the men suffered on this account, for the day's work on board ship was mapped out for them, and was, of course, regularly executed; and, as regards the sledging expeditions, there can be little doubt that the severe muscular exertion undergone, after comparative inaction during the winter, was in verity and truth one predisposing cause of the outbreak.

A great deal was said and written previous to the inquiry as to the prevalence and degree of severity of scurvy on former sledging expeditions. The evidence adduced on this important point is very unsatisfactory and altogether inconclusive. It is well known that in M'Clintock's expedition with the "Fox," scurvy attacked Captain Hobson and others with great severity, and there is good reason to believe that, with some few exceptions, the disease appeared in a greater or less degree among the men of all former extended sledge expeditions, *i.e.*, when no antiscorbutics were carried and taken systematically. Indeed, nearly all the leading witnesses, including Sir George Nares, agreed on this head; and in the cases of the "North Star," "Pioneer," and "Intrepid," the medical evidence goes to prove that the immunity from scurvy was due to the regular serving out of lime-juice, with frequent rations of fresh meat and vegetables.

If we turn aside to seek for outside evidence as to the influence of vegetable diet, or its substitute, lime-juice, as a prophylactic, the most recent and most satisfactory results are to be found in connection with the mercantile marine. For some years prior to 1867 it was found by the medical officers of the Dreadnought Hospital Ship that the admissions for scurvy were increasing. It occurred to them to obtain samples of the lime or lemon-juice used on board these scurvy-stricken ships,

and analyses showed that, in many cases, an entirely spurious article was carried. Mixtures of citric acid and water, weak solutions of sulphuric and citric acids, coloured in some cases with brown sugar, were found as substitutes for lime-juice. This discovery led to legislation under the auspices of the Board of Trade. By the terms of the Merchant Shipping Act of 1867, commonly called the Duke of Richmond's Act, the quality of all lime and lemon-juice carried in British merchant ships is now assured, the juice being officially tested and afterwards kept in bond ; and, as a practical result, it is found that during the last ten years scurvy has decreased in the mercantile marine 50 or 60 per cent. In point of fact, the large majority of cases now admitted into the Seamen's Hospital at Greenwich are those in which the disease has appeared as "secondary" to some other malady, usually dysentery, Mauritius or West Coast fever, or some variety of syphilitic disease. The increase of steamships, the opening of the Suez Canal, the consequent shortening of ocean voyages, and the more general use of preserved tinned provisions of various kinds, have all assisted this satisfactory result. But the comparative immunity from scurvy is observed equally in ships that keep to the old sea tracks, and the most recent official inquiries show that when the disease has appeared in a severe form, carelessness had occurred in the serving out of the lime-juice, and in some few cases it was found to have been omitted altogether.

It remains to notice the suggestions given in the report as to the prevention of scurvy, and the use of lime-juice and other (so-called) antiscorbutics.

The dietetic suggestions included the carrying of eggs—a recommendation urged by several witnesses. The sense of this suggestion is sufficiently apparent, as each egg contains an average amount of nutriment equivalent to twenty ounces of fresh beef.

Eggs can be preserved without difficulty either in milk of lime or in oil, butter, or collodion dissolved in ether, or by merely placing them for a few seconds in boiling water, or by pouring boiling vinegar over hard-boiled eggs after the shells have been removed. In Arctic service any difficulty as to preserving them would of course cease as soon as high latitudes were reached. Milk is also strongly recommended, either in the condensed or desiccated form. An instance of the antiscorbutic value of milk is quoted by Sir James Paget, who some years ago, when scurvy was being discussed at the Clinical Society (of which he was then President), said that a medical man well known to him, for some reason lived for nineteen years with only milk and

various breadstuffs for food. At the end of that period, however, he had scurvy in a severe form, but quickly recovered with a change of diet.

Fresh meat, or, as the best substitute, preserved meats, are of course strongly recommended. It appears to me that the bad quality of the animal diet taken (whether salt or fresh) may form an important factor, *i.e.*, be classed as an exciting cause, in the production of scurvy. Preserved meats are to a certain extent objectionable, because they are insipid, but this defect may be remedied by the use of pickles and other condiments.

Vegetables rank highest in antiscorbutic value. They should be as far as possible of a succulent character. The tinned vegetables analysed showed little if any change, but the compressed vegetables appeared to be not so trustworthy. There appears to be little doubt that, as far as our information extends, the preserved potato (Edward's) is the best form of vegetable diet in a preserved state. It seems to retain all the chief constituents of the fresh potato in natural proportion, and is agreeable and palatable, one part being equivalent to about three and a half parts of fresh potato.

The dietetic use of alcohol has already been referred to, and its antiscorbutic virtues, or even its powers of sustaining the system under prolonged physical exertion, may be considered as practically nil.

It is very properly stated in the report that vegetable foods contain many substances that are not peculiarly antiscorbutic, and that this increase might lead to an inconvenient addition to the quantity of the food. Hence a preference should be given to vegetable substances that are more antiscorbutic than nutritious, and it appears that in this category lime and lemon juice reach very high. In consequence, however, of the presence of a large amount of pure acid in these juices, their action may be injurious if given in too large quantities, by impairing digestion and thus diminishing appetite ; and it may be fairly considered that the navy and mercantile marine ration of an ounce is, under ordinary circumstances, sufficient for prophylactic purposes.

It was agreed, however, and with some show of reason, that lime-juice cannot be carried in sledging expeditions so as to be given systematically, according to the scale above mentioned. For not only would the weight of the juice itself be an obstacle, but the fuel required for the melting, the probable bursting of bottles, and the time occupied in the process, would still further complicate matters. It occurred, therefore, to several witnesses in the course of the inquiry, whether the abstraction of a certain proportion of water from lime-juice would impair its antiscor-

butic properties. The practical outcome of these suggestions is that lime-juice has been concentrated, and reduced to the form of lozenges, three or four of which are said to be equivalent to an ounce of the juice. We have yet to ascertain the antiscorbutic value of these concentrated preparations; but there can be no doubt that the suggestion is most important, and deserves a thorough investigation, both by the Admiralty and the Board of Trade. Unfortified lime-juice freezes at 25° Fahr., and when fortified with 10 per cent. of proof spirit, at about 15° Fahr. This fact would, of course, only affect the circumstances of voyagers in high latitudes, but the advantage of procuring a specially portable, as well as an efficacious antiscorbutic for general use, needs no argument.

This short paper professes to be nothing more than a brief abstract of the evidence given before the Arctic Committee, and taken chiefly from a paper on scurvy contributed to the report by Dr. James Donnet, H.M. Inspector-General, and Dr. Thomas R. Fraser, now Professor of Chemistry at the University of Edinburgh, medical members of the Committee, who collected and elaborated the scientific evidence with very great skill and labour. The main conclusion arrived at by the Committee, and endorsed by the Admiralty, was that "the early outbreak of scurvy in the spring sledging parties of the expedition was due to the absence of lime-juice from the sledge dietaries." Sir Alexander Armstrong, the Medical Director-General of the Navy (in a Memorandum of Recommendations sent to the commander of the expedition for his guidance) expressly enjoined the systematic serving out of lime-juice on all occasions. I think that the very great majority of those competent to form an opinion upon the subject must conclude that the verdict of the Committee was just, proper, and indeed inevitable.

APPENDIX.

No. 1.—Scale of Victualling on Board Ship and Quantity of Provisions Supplied.

Scale A.	Quantities Supplied.	Weights.	Monday.	Tuesday.	Wednesday.	Thursday.	Friday.	Saturday.	Sunday.	Monday.	Tuesday.	Wednesday.	Thursday.	Friday.	Saturday.	Sunday.	Monday.	Tuesday.	Wednesday.	Thursday.	Friday.	Saturday.	Sunday.	Monday.	Tuesday.	Wednesday.	Thursday.	Friday.	Saturday.	Sunday.
Biscuit	lbs. 21,350	lbs.	1	...	...	1	...	...	1	...	...	1	...	...	1	...	...	1	...	...	1	...	...	1	...	...	1	...	...	1
Flour (for making bread)	,, 53,750	lbs.	...	1	1	...	1	1	...	1	1	...	1	1	...	1	1	...	1	1	...	1	1	...	1	1	...	1	1	...
Preserved meats	,, 29,256	lbs.	...	¾	...	¾	...	¾	...	¾	...	¾	...	¾	...	¾	...	¾	...	¾	...	¾	...	¾	...	¾	...	¾	...	¾
Corned beef	,, 20,100	lbs.	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...
Corned pork	,, 20,500	lbs.	1	...	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...	...	...
Preserved soups	,, 8,520	lbs.	...	½	...	...	...	½	...	...	...	½	...	...	...	½	...	...	...	½	...	...	...	½	...	...	...	½	...	...
Preserved vegetables:—																														
Potatoes	,, 6,400	lbs.	...	¼	¼	½	...	¼	¼	½	...	¼	¼	½	...	¼	¼	½	...	¼	¼	½	...	¼	¼	½	...	¼	¼	½
Carrots	,, 5,460																													
Onions	,, 2,832																													
Compressed vegetables:—																														
Dried cabbage	,, 544	oz.	1	...	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...	...	...
Mixed vegetables	,, 544																													
Flour (For making puddings)	,, 1,050	oz.	...	...	9	...	...	...	9	...	...	..	9	...	...	...	9	...	...	...	9	...	...	...	9	...	...	...	9	...
Suet (For making puddings)	,, 1,058	oz.	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...
Raisins (For making puddings)		oz.	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...	...	...	1	...
Split peas	,, 4,230	lbs.	¼	...	...	...	¼	...	...	...	¼	...	...	...	¼	...	...	...	¼	...	...	...	¼	...	...	...	¼	...	..	...

Spirits	galls. 1,021	gills	½ daily.	Mustard	300	oz.	½ twice a week.
Celery seed	lbs. 25	oz.	½ to every 8 lbs. of peas.	Pepper	140	oz.	¼ twice a week.
Chocolate	,, 2,950	oz.	1 daily.	Fruit	3,634	oz.	2 twice a week.
Consolidated tea	,, 112	oz.	¼ daily.	Sugar for ditto*	Included above.	oz.	¾ twice a week.
Tea	,, 927			Vinegar	51	oz.	As requisite.
Sugar*	,, 12,250	oz.	1¾ daily.	Oatmeal	84	oz.	As ordered.
Lime-juice	,, 4,240	oz.	1 daily.	Salt	144	oz.	As requisite.
Sugar for ditto*	Included above	oz.	1 daily.	Beer	10 hhds.	pts.	As ordered.
Pickles	lbs. 4,082	oz.	1 daily.				

* 1 man for 28 days.

No. 2.

DIET LIST, &c., ARCTIC SLEDGE PARTIES.

	lb.	oz.	
Pemmican	1	0*	
Biscuit	0	14	
Bacon	0	4†	
Potato	0	2	
Rum		½	gill.
Chocolate	0	1	
Sugar for ditto	0	0½	
Tea	0	0½‡	
Sugar for ditto	0	1½	
Stearine	0	3	per man.
Spirits of Wine	0	1	per man.
Tobacco	0	0½	
Salt	0	0⅛	
Pepper	0	0 1/20	
Onion and Curry Powder	0	0¼	

* Pemmican consists of equal parts of powdered lean beef and suet, seasoned with cayenne pepper in the proportion of $\frac{1}{8}$th of an ounce of pepper to 8 lb. of the mixture. To make sweet pemmican ½ lb. of sugar is added.

† Increased in some cases to 6 ounces, at request, in lieu of pemmican.

‡ Double allowance of tea was carried in lieu of rum.

No. 3.

CARBONIC ACID ESTIMATIONS BETWEEN DECKS, H.M.S. "ALERT."

By Staff-Surgeon Edward L. Moss.

Date.	Position and Circumstances of Air Examined.	Percentage of Carbonic Acid.
23 Oct. '75 before housing	Air taken at midnight from level with the men's heads as they slept, and at a point removed from direct indraughts	·220
8 Nov. after housing	Air taken under the same circumstances, and at same time as before . .	·405
8 Dec.	Air taken as before	·308
16 Jan '76.	Air taken as before	·2882
29 Feb.	Air taken as before	·436
16 Jan.	Air of Stokehold (temperature —2°) .	·266
18 Jan.	Air of Ward Room at 11 P.M., 5 feet from floor. Room feeling close. Lamps and candles, of course, constantly burning	·482

The value of the results obtained from examination of the external air cannot be correctly estimated without a long description of the three various arrangements made to admit of the examination of a large volume of air at temperatures between —26° and —63° Fahr. The average of my observations is ·0552.

Note.—The results of three estimations of the carbonic acid in the outside air at a short distance from the ship, were :—

10th December 1875 . .	0·0641 per cent.
18th January 1876 . .	0·0483 ,,
29th February 1876 . .	0·0536 ,,
	0·166 ÷ 3 =
Average of estimations . .	0·0552 per cent.

Abstract from Dr. Moss's Special Report on this subject, dated "H.M.S. 'Alert,' Winter Quarters, North Lat. 82·27."

The mean of the five estimations given in the Table of the percentage of carbonic acid in the air in the men's sleeping quarters is 0·3314.

FURTHER HISTOLOGICAL RESEARCHES

ON THE

PATHOLOGY OF THE NERVOUS SYSTEM.

BY

W. HENRY KESTEVEN.

In the last volume of these reports is a paper on the different histological forms of degeneration which are met with in the nervous tissues of the cerebro-spinal centres. Besides the appearances therein named degenerations, are others also of a morbid nature to be now described.

Among these are the atrophic changes of the various elements of the structure—*atrophic* as distinguished from *degenerate*, the latter term having been limited, in the papers above alluded to, to those changes which were more qualitative than quantitative.

In most cases the atrophic changes are general, that is to say, are not limited either to the nerve fibres, the nerve corpuscles, or to the neuroglia, but affect all alike, causing in some cases marked distortion and confusion. The appearances presented by the different elements of the nervous structure, when undergoing or when they have undergone atrophic change, are as follows:—

Nerve Cells or Corpuscles.—The first sign of atrophy presented by these bodies is the appearance between them and the surrounding neuroglia of a small space which gradually increases in size. At the same time the nucleus of the corpuscle undergoes degenerative change, and appears to assume an abnormal increase in size.

This latter is, however, only apparent, and is due to the fact that it is the body of the cell or corpuscle which wastes, the nucleus retaining its normal size, though more or less degenerated in intimate structure.

The processes of the corpuscle shrink, and finally, loosing their axis cylinders, either disappear altogether, or are left as simple fibres. The final stage of atrophy of the nerve corpuscles is similar to that of pigmentary degeneration of these bodies. The processes disappear, the corpuscular body shrinks more and more, and there finally remains only an agglomeration of black pigment granules. This atrophic change of the nerve corpuscles is generally due to deficient nutrition. In specimens where it is met with, it is nearly always associated with vascular changes. It is therefore very common in advanced age, as has been pointed out by Dr. Herbert Major. (See fig. 1.)

Atrophy of Nerve Fibres.—Atrophy of the nerve fibres of the brain and cord is generally found associated with sclerosis of the neuroglia. Sclerosis is an increase of the neuroglia and its component parts, taking place at the expense of the nerve fibres, &c., which they are intended to support. The nerve fibres, when cut across transversely in healthy specimens, are recognisable by the appearance of the axis cylinders. These, when stained, resemble brilliant points, fully equal in size, or even larger than the nuclei of the neuroglia. They may be distinguished from them by slightly altering the focus, when the axis cylinders will be found to be spiral cords of uniform diameter, this being proved by the fact that, when they are carefully watched during the alteration of the focus, the brilliant point is never lost to sight, but the position of it is found to vary, travelling in a spiral direction. With the nuclei of the neuroglia this is never the case ; on altering the focus, these bodies generally disappear from view.

In cases of atrophy of these nerve fibres, this spiral cord gradually shrinks, and loses it brilliant hue. It also loses its regular spiral arrangement, becomes more or less crumpled, finally disappearing and leaving a round or oval-shaped hole, according as the section is more or less oblique. This hole is gradually obscured and filled up by the hypertrophied neuroglia. The same process of atrophy may be seen in cases where there is no absolute increase, but even a decrease, in the connective and other elements of the nerve substance, as in general nervous atrophy due to external pressure. Another form of atrophy of the nerve fibres is described in the paper above referred to under the name of "*Insular Atrophy.*"

Atrophy of Neuroglia.—Atrophy of the connective tissue is not seen existing by itself. It is always associated with atrophy of the other nervous elements. Disappearance of or excessive decrease in the normal number of the nuclei of the neuroglia is the distinctive sign of this condition. General atrophy may take place in consequence either of pressure from without or from

deficient nutrition. When the former of these conditions is the cause of the atrophy, it is sometimes difficult to say at first sight whether the appearance presented is sclerosis or atrophy. The reason of this is, that, when this pressure from without takes place, the first parts to become atrophied are the nerve corpuscles and nerve fibres. The pressure causes the neuroglia which is left to fill up the vacancies thus formed, and if the examination chances to be made at this stage, there is nothing but connective tissue to be seen. These conditions however, may be distinguished by the absence, in cases of atrophy, of the so-called *cellule araignèe* of Deiters, which is always present in sclerosis. There is also a marked difference in the number of the nuclei of the neuroglia.

General nervous atrophy causes marked distortion, particularly when it is limited, as in some cases, to one side of the cord. The pressure of the serous fluids causes the unaffected half of the cord to fold over and fill up the space which would otherwise be left. The contortions presented in cords thus affected are almost indescribable. The well-known letter H, which the central grey matter simulates, is twisted in all directions, and at times quite loses its characters. (See fig. 2.)

General atrophic changes are sometimes acute, taking the form of acute softening. Portions of nervous tissue undergoing this change present under the microscope intricate mixtures of broken nerve tubes, oil globules, detached portions of myeline and general debris.

Alterations in the Blood Vessels.—The changes which occur in the blood vessels of the nervous structures are, with slight exception, similar to those which are met with in other blood vessels. It is therefore unnecessary in this place to enter into descriptions which have been fully given by various pathologists. Pathological vascular changes have also had much light thrown on them by the controversy between Dr. G. Johnson on the one hand, and Sir W. Gull and Dr. Sutton on the other. It may be, however, of some interest to point out the positions at which such changes as aneurism, varix, &c., are most commonly met with.

In transverse sections of the spinal cord, the blood vessels may be seen forming loops from the margins of the section. It is at the bend of the loop that we most commonly meet with aneurismal enlargements. As might be expected, when this is the case, it is on the outer side of the curve that the vessel has given way. (See fig. 4.) Another point at which these aneurismal dilatations are met with is at the lower end of the anterior fissure of the cord. It should be borne in mind, that in sections of healthy cord, the anterior fissure passes backwards till it reaches the grey commissural matter of the cord. On the other side of this grey

matter is the posterior fissure, whilst intermediate between these two fissures, and passing through the grey commissure, is the central canal of the spinal cord. Along the anterior fissure of the cord there is sometimes seen a blood vessel passing backward, and penetrating the substance of the cord on either side of the central canal. At the lower end of the anterior fissure this vessel is sometimes seen in an aneurismal condition, and in some cases it is found to encroach upon and to contract the central canal. (See fig. 3.) Aneurisms are also to be found in the posterior fissure.

An interesting point connected with alterations of the vessels in the nervous structures is, that whenever the vessel is enlarged, either by a thickening of its coats or by a dilatation of its lumen, in order to accommodate such enlargement the nerve tissue has to give way. The result of this yielding of the nerve substance is the formation of spaces between the vessel and the nerve substance. This appearance is treated of later on. When the vessel which is enlarged does not traverse the nerve substance, that is, when it passes through a fissure as described above, there is, of course, more room for enlargement. Even here, however, when the enlargement is excessive, the neighbouring nervous tissues may be encroached upon. When this is the case, the same result is attained; there is a yielding and consequent absorption of tissue.

This may be seen very markedly when the vessels in the fissures of the cord or between the cerebral convolutions are varicose or aneurismal. In many cases this morbid appearance is so marked as to cause absolute distortion. (See fig. 5.) This fact goes to prove the morbid nature of the so-called perivascular spaces; the argument being, that because the process of absorption of the neighbouring tissue in the case of a vessel passing along a fissure is morbid, and because such absorption is the same as that seen when perivascular spaces are formed in the nerve substance, therefore the formation of perivascular spaces is also morbid.

Sections of brain and cord sometimes exhibit divided vessels filled with blood corpuscles more or less disintegrated. No doubt in many cases this is a post-mortem hypostatic condition; but before we positively conclude that the appearance is not abnormal, it is necessary to decide whether the vascular walls are in any way diseased. Where this is the case, as in aneurism, it is very possible that the blood corpuscles form part of an embolus. Again, if the corpuscles are much disintegrated, and are mixed with crystals of hæmatoidin, it may fairly be concluded that a section has been made through an embolus.

Perivascular Spaces.—There is no morbid change which is more commonly met with in the nervous structures than these

spaces, which, as their name denotes, are mostly concentric to the vessels. They vary much in size and shape, but these variations are always found to correspond with those of the vessels within them. Thus they are frequently varicose or tortuous, and in various ways irregular. Other spaces are, strictly speaking, regular, having parallel sides. The spaces may be empty, and clean cut, but they frequently contain, especially about the larger vessels, portions of the tunica adventitia or pia mater, which, in the healthy nerve structure, lies in close contact with the vessel. Sometimes these spaces contain altered blood corpuscles and crystals of hæmatin. Bodies resembling pus corpuscles are rarely found.

It has been contended that these spaces are portions of the lymphatic system, that they are constantly present in the healthy nerve structure, and in communication with the serous cavities of the encephalon. In a work published at Stockholm by Axel Key and Retzius during the course of 1875, very elaborate and exact descriptions were given of certain injections made with a view of discovering the extent and shape of these serous cavities. That they do not penetrate the nervous substance was, however, conclusively shown. At the point where the blood vessels enter the nervous substance, they are surrounded by a funnel-shaped investment of the serous membrane, which is closely attached to the vessels immediately after they enter the nervous substance. It was found by Key and Retzius that the injections used never penetrated beyond this point of attachment. In a paper published by my father in the eighth volume of these Reports, he maintained that these spaces were not normal structures, and his views are thus confirmed by these observers.

The true explanation of this appearance is as follows. Any congestion or inflammatory action will cause a dilatation of the vessels. In this condition the nervous substance is encroached upon and compressed by the vessels. The inevitable consequence of this pressure is absorption, and in some cases condensation (sclerosis) of the surrounding nerve substance. After death, the vessels, being no longer distended with blood, contract, and are found lying in the perivascular spaces. In cases where the pressure on the nerve substance has been due to a degenerated, weakened, or thickened condition of the vascular walls, these spaces are not so well seen; for in these cases, owing to the condition of the walls of the vessels, they do not contract as completely after death as when possessed of their normal tone; their dilatation has been due to a condition of simple hyperæmia. The spaces are none the less present though obscured and partially filled by the flaccid vascular walls.

It is in this condition that extravasated blood corpuscles and crystals of hæmatin are frequently found. When the irritation of the dilated vessel is excessive, or has been long maintained, we meet with inflammatory products.

From the appearance then presented by the perivascular spaces it is possible to discriminate between diseases due to old standing degenerative changes, and those of a more recent and acute character. It has been suggested that these spaces may always be found in the nervous tissues of old people, and that in these cases they are due to an atrophic condition of the nerve substance itself. It would, however, be more correct to say that they are due to altered vascular conditions.

Spaces are sometimes formed in connection with the blood vessels which are not always perivascular. Their position with regard to the vessel is necessarily dependent to a great degree on a greater or less equal distribution of the vascular pressure. In such cases of aneurism as those described above, where the dilatation of the vessel, and therefore the pressure, is limited to one side, the space formed by such pressure is similarly limited. In fig. 4 such a specimen is shown. The smaller vessels in this specimen had real *peri*vascular spaces around them, due to mere hyperæmia. In fig. 6 the perivascular space is due to alteration in the thickness of the wall of the vessel; the space is therefore not so well marked.

It was stated above, that the changes met with in the blood vessels of the nervous structures were, *with slight exception*, the same as those met with in other parts of the body. In the "Medical Record" for August 1877 there is an abstract from the work of Professor Heubner describing the syphilitic vascular changes which occur in the nervous structures. Without admitting the necessarily syphilitic origin of these appearances, inasmuch as they have certainly not been described as present in the vessels of other parts of the body, they may be said to come within the scope of this paper. Dr. Heubner asserts that the disease is one which commences in the endothelium of the vessels, being indicated by a proliferation of that structure. One result of this proliferation is that the lumen of the vessel is encroached upon, and finally becomes choked. In other cases the increase in the endothelium takes direction both inwards and outwards, not only choking the passage of the vessel, but encroaching on the middle muscular coats of the vessel.

There is no doubt that this appearance is sometimes found in the vessels of the nervous structures. There is also one reason why there may be some truth in ascribing to it a syphilitic origin. In a series of sections lately examined, in which this

proliferation of the vascular endothelium was found, there were also found vessels which were altered in shape and size by a deposit in the middle coats of a granular homogeneous substance, bearing a close resemblance to the structure of the syphilitic gummata (see fig. 6). These appearances were in this specimen closely associated, the same vessel being found attacked by Heubner's endothelial proliferation, and almost choked; and at the same time having this gummatous deposit in its walls, with the lumen, in some places, patent and clear. It is, however, notwithstanding this case, at present doubtful whether the appearance is syphilitic, and somewhat difficult to see why the syphilitic virus should single out the endothelium of the vessels of the nervous structures alone, leaving the same structure in other vessels unaffected.

The question may be asked, To what extent are these various morbid changes in the nervous elements pathognomonic of the different forms of nervous disease met with during life? Up to the present time this question can only be answered in a general manner. We cannot always assert before death that such and such symptoms are due to such and such particular forms, either of degeneration or atrophy. As was mentioned in the former paper on this subject, that particular form of morbid change which has been denominated *Miliary Degeneration*, has been met with in many and various forms of nervous disease, and the same may be said of nearly all the other morbid changes above described. It must even be regarded as doubtful, at present, whether the endothelial proliferation of Professor Heubner can be certainly said to be pathognomonic of syphilis. The only certain conclusion which can be arrived at before death regarding the changes which are taking place in the nervous tissues is, as was before remarked, of a general nature. These remarks, of course, apply to those forms of disease which are called chronic. Acute nervous affections have distinctive and recognisable causes; but in the chronic forms of paralysis and mental disorders, we can only conclude that the tissues are undergoing one or more of the various degenerative and atrophic changes described above. How far these changes are amenable to therapeutic agents, whether, in fact, they are at all so, remains a problem for future study. But it is only by such pathological investigations into the nature of these morbid changes, that we can ever hope to apply any means otherwise than empirically.

DESCRIPTION OF FIGURES.

Fig. 1.—Atrophy of nerve corpuscle, showing peri-corpuscular space with colloid bodies.

Fig. 2.—Distortion of spinal cord, due to unequal atrophy of both sides.

Fig. 3.—Aneurismal dilatation of a blood vessel in the anterior fissure of the cord, the aneurism encroaching on the central canal.

Fig. 4.—Aneurism of the loop of a vessel in the spinal cord, showing the space formed at the expense of the nervous tissue at the point where the chief pressure existed.

Fig. 5.—Absorption of nervous tissue in the anterior fissure, caused by dilatation of the blood vessels.

Fig. 6.—Blood vessel showing proliferation of the endothelium obstructing the lumen; with gummatous deposit between the outer and inner coats of the vessel. This figure also shows perivascular space.

Fig. 1

Fig. 2

Fig. 3

Fig. 4

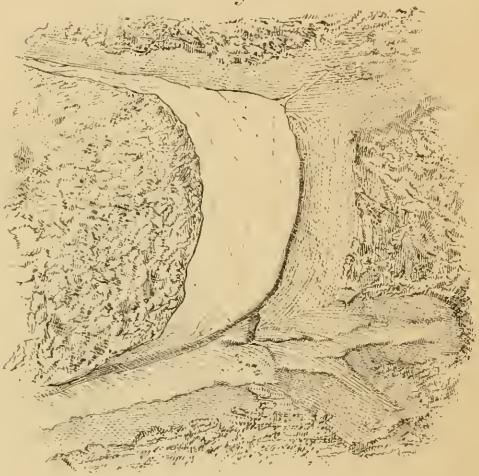


Fig. 5

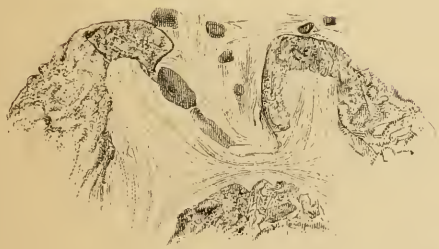
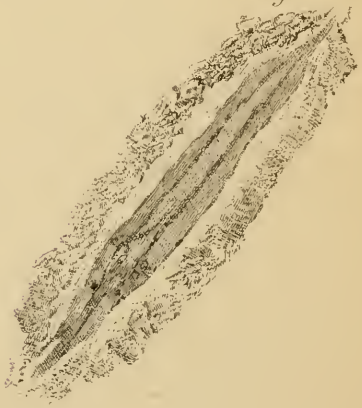


Fig. 6



THE STRUCTURE AND FUNCTIONS OF THE OLIVARY BODIES.

BY

W. HENRY KESTEVEN.

These bodies are placed on either side about one-eighth of an inch away from the anterior fissure of the medulla oblongata, just below the pons varolii. Between this portion of the brain and each of the olivary bodies there is a deep fossa. Separating the olivary bodies from each other are the anterior pyramids of the medulla. In measurement these bodies are about half an inch long and two-fifths of an inch broad. They form olive-shaped projections (hence the name) on either side of the medulla. Externally they consist of white fibrous matter, internally of grey matter arranged in convolutions, presenting on longitudinal section somewhat the appearance presented by a similar section of a kidney, the hilum of the pelvis being directed inwards and backwards. On examining a transverse section of the medulla made about the middle of these bodies, the roots of the ninth nerve are seen in their passage from their nucleus to pass forwards internal to the olivary bodies, cutting off a small portion of the grey matter of the olive at the point where the roots bend outwards to pass out of the medulla. Some fibres from these bodies pass out with the roots of the ninth nerve. Posterior to the olivary bodies is a network of fibres described by Lockhart Clarke, through which the roots of the vagus pass to leave the medulla at the posterior border of the olive, in which, about the level of the olivary bodies, may be seen the lower extremity of the nucleus of the fifth nerve.

The olivary bodies communicate with each other by means of minute fibres, which may be seen intersecting each other as they cross the central raphé of the medulla.

The grey or vesicular matter of the olivary bodies consists of nerve corpuscles, fibres, and neuroglia. The corpuscles are of the size of the larger form usually met with in the cerebral cortex, that is, rather smaller than in the cord. The fibrous matter of this portion of the olivary bodies, is formed partly by the neuroglia and partly by the processes of the nerve corpuscles, some of which serve as means of communication between the corpuscles, whilst others pass into the white cortex of the bodies in various ways to be hereinafter described.

The external white matter, or fibrous cortex of the olivary bodies, consists of two layers of fibres, which are derived from the nerve corpuscles of the grey portion of these bodies.

The fibres of the external layers are derived from some of the internal corpuscles of the convolutions. The processes of these corpuscles which form the external layer, converge and pass through the pelvis in company with other fibres from the same source. Leaving the pelvis of the olivary body, these fibres pass upwards, winding over its superior border, and then passing downwards on the outer side, are continued down the medulla, somewhat diverging as they proceed.

Starting with them from the pelvis of the body are fibres which pass directly inwards and downwards in the substance of the medulla; whilst a third portion, starting from these internal corpuscles, serve as commissural fibres between the bodies crossing the raphé as described above.

The internal layer of the white fibrous cortex is formed of fibres, which pass from the corpuscles of the external aspect of the folia of the convolution, and, passing at right angles to the fibres of the external layer, proceed in a direction from before backwards, closely investing the folia of the vesicular convolutions.

At the posterior aspect of the bodies, in the substance of the medulla, these fibres divide. Some pass inwards across the raphé to help in forming the commissures, and others pass backwards into the network which is situated behind the olivary bodies, which has been mentioned above; some of them joining the vagus previously to its exit from the medulla, and others mingling with the processes from the corpuscles of the nucleus of the fifth nerve. The blood vessels of the olivary bodies enter at the hilum of the pelvis, and penetrate into the convolutions of the grey matter and external white layers, being finally distributed by means of loops.

The function of the olivary bodies is doubtful, but there are considerations connected with their anatomical structure, which make it possible, or which afford sufficient grounds, on which

to base a theory as to the rôle they play in the nervous portion of the vital economy. They are not isolated bodies; they are closely connected with several important nerves, either at their nuclear origins, or in their course at very slight distances from the nuclei. These nerves are the fifth, the eighth, and the ninth.

The question which presents itself is, Are these nerves ever associated in the production of any particular results?

If we examine the parts which are supplied by these nerves, we find that the fifth nerve, by means of its motor root, supplies certain muscles which are chiefly concerned in moving the lower jaw and the soft palate. The ninth nerve supplies motor power to the tongue, and the eighth nerve supplies the pharynx with motor and sensitive power. We therefore find that it is these nerves which supply the power of movement to the upper portion of the alimentary canal, in other words, they are the nerves which preside over the movements of deglutition, mastication, &c. However, for the satisfactory performance of these acts, something more than mere motor power is required. In order to excite the necessary movements certain sensations are required. And here, again, we find that it is to these nerves, or rather to two of them, that the power of perceiving and transmitting these sensations is committed. The fifth nerve supplies not only common sensation to the inside of the mouth, but also the special sense of taste; while the eighth nerve, by its glosso-pharyngeal branch, supplies a great part of the tongue and pharynx with common sensation.

We thus see that when food is placed in the mouth it is tasted, felt, masticated, and swallowed by the action of these nerves. It is then evident that, for the satisfactory performance of these duties, the powers exerted by these nerves must be co-ordinated or brought into communication. Such combination evidently exists, and its very existence proves the co-existence of some special contrivance to accomplish the desired result. This contrivance, one is fairly justified in arguing, is found in the olivary bodies, which, by their position, and by the connection which it has been shown they have with the nerves chiefly concerned in the production of the movements requisite for deglutition, are well fitted to act as centres for the co-ordination of these nerves.

ON

THE CHRONIC PNEUMONIA

WHICH ATTENDS DISEASE OF THE TRACHEAL AND BRONCHIAL GLANDS.

BY

SAMUEL GEE, M.D.

The tracheal glands lie alongside the trachea down to its bifurcation; the bronchial glands lie immediately below the diverging bronchi. It has seemed to me that disease of these glands sometimes causes a chronic pneumonia. If it be so, it is a sequence of diseases not commonly known to happen. I will first narrate four cases which bear upon this topic, and will comment upon them afterwards.

Case I.

Eliza W., 3 years and 9 months old.

1. No strumous diseases known of in her kindred, except that her mother's uncle died of phthisis.

2. She was deemed to be a healthy child until she had a mild attack of scarlet-fever in June 1876. She did not recover thoroughly. In November she suffered from diarrhœa, which weakened her still more.

3. *Onset of pulmonary symptoms.*—About December 7, 1876, the cough began; breath short; a crackling sound heard in her chest by her mother; loss of appetite and flesh.

4. *Course of disease.*—She was admitted into the Hospital for Sick Children on December 22, 1876, and died there on March 8, 1877. The notes by Dr. Garlick.

(1.) She became more and more emaciated and cachectic.

(2.) Hectic fever throughout, yet the temperature, until

February 19, very seldom rose above 101° (38·4° C.); after February 19 it rose frequently to 103° (39·4° C.), and on one occasion to 104·4° (40·4° C.). The exacerbation of fever concurred with the symptoms of pulmonary gangrene.

(3.) The bowels were mostly loose, and the evacuations offensive. Abdomen swollen and doughy.

(4.) Cough, towards the end, became very troublesome, and was often followed by vomiting.

(5.) Sputa became very offensive on February 19, and continued to be so until death. On February 28 fragments of the elastic framework of the lung were detected in the sputa by the microscope.

(6.) Hæmoptysis, copious, occurred on the four days before death.

(7.) *Physical signs.*—Right side of chest natural; breathing puerile; left side of chest retracted somewhat, excepting the apex; heart's impulse, fourth interspace, nipple line; spleen just palpable below margin of ribs; marked dulness to percussion at the left base behind, reaching at first to the angle of the scapula, and afterwards to its spine; no dulness in lateral region or in front; stomach note reached as high as the nipple level; respiration weak over the whole of the left side; highly bronchial over the dull region with bronchophony; rales very variable in place and extent, sometimes none at all; when present, mucous, and especially at left base; decided dulness to percussion behind upper half of sternum.

5. *Post-mortem examination.*—Bronchial glands: below the bifurcation of the trachea, immediately behind the pericardium, which was discoloured at that spot, was a cavity, the size of a thrush's egg, containing a little slough and black liquid. The sloughs, cut into, seemed to be swollen, bluish, flabby glandular tissue; no caseous matter. The cavity opened into the front of the œsophagus by a small discoloured hole two inches above the cardia; another similar opening into the left bronchus, on its internal wall, half an inch below the bifurcation. The cavity was hard upon a large branch of the right bronchus, but did not perforate it. Many small caseous glands in roots of lungs.

Tracheal glands: alongside trachea on the right greatly enlarged and highly caseous.

Left lung: whole of lower lobe and part of upper lobe reduced to a soft, broken-up slough, enclosed in a capsule formed by the thickened pleura. The left bronchus came to an abrupt end almost as soon as it entered the slough. One large branch of the pulmonary artery entered the slough; there were no coagula in the vessel, but it was tightly contracted, especially in

the slough, where its walls were so rotten that it was hard to follow it far. The greater part of the upper lobe was in a state of slaty induration, speckled with cheesy nodules here and there.

Right lung: studded with miliary tubercles. Pericardium: some excess of fluid; turbid; a few flakes of lymph. Heart: natural; no old clots in it or large vessels. Peritoneum: some miliary tubercles over liver. Liver and spleen: tubercles beneath capsule and in substance. Kidneys: a single tubercle seen. Intestines: numerous tubercular ulcers in ileum and cæcum.

Case II.

Ellen C., 6 years old.

1. No strumous diseases in her kindred known of.

2. She had measles at four years old, hooping-cough in the beginning of 1876, scarlet fever two or three months afterwards (in the summer of 1876), followed by dropsy for a short time. She seemed to recover thoroughly. She was always thought to be a strong child.

3. *Onset of pulmonary symptoms.*—One day in September 1876 she came home from school on account of illness; headache, repeated vomiting, becoming bilious; shivering. The shivering recurred every second day (according to the mother's account); heat of skin; no sweating; these febrile symptoms lasted about two months. Cough began at the same time, and continued all along, gradually becoming much worse, so as to keep the child awake at night. Small yellow expectoration. Emaciation.

4. *Course of disease.*—She was admitted into the Hospital for Sick Children on February 1, 1877, and died there on March 12, 1877. The notes by Dr. Garlick.

(1.) Emaciation and cachexia gradually increased.

(2.) Hectic fever throughout, yet the temperature seldom rose above 102° (39° C.), and only once above 103°, namely, to 103·2° (39·8° C.).

(3.) No diarrhœa.

(4.) Cough at first dry and hard, but latterly became loose, with bright yellow sputa, in which no elastic tissue was found. More or less lividity throughout. Sometimes, at night, she would become very blue, seemed choked, had to sit up in bed, and coughed much. No hæmoptysis. Pain in the right side of the chest.

(5.) Voice became very hoarse and weak.

(6.) Lymphatic glands in neck not enlarged. No bulging above clavicles.

(7.) *Physical signs.*—Shape of chest perfectly natural until a few weeks before death, when the right side came to look full. Heart's impulse, fifth left interspace, just outside nipple line. Spleen palpable just below margin of ribs. Dulness to percussion on right side below level of angle of scapula; elsewhere tone is good. Bronchial breathing becoming more and more intense all over dull region. At first a sharp mucous rale in same place; towards the end the rale became very abundant, loose, and gurgling. No dulness behind sternum.

(8.) In order to confirm the diagnosis, and to exclude the possibility of local empyema, a subcutaneous injecting needle was passed into the chest at the angle of the scapula; nothing followed except a drop of serum.

(9.) Convulsions occurred some hours before death.

5. *Post-mortem examination.*—Bronchial glands: at bifurcation of trachea changed into a cheesy mass, nowhere softened. Perforations of air-tubes; right bronchus, half an inch below bifurcation of trachea, perforated by an ulcer ($\frac{3}{4}$ in. × $\frac{1}{3}$ in.), whose base was formed by cheesy unsoftened gland; further down a secondary bronchus perforated by a much smaller ulcer lying on a firm cheesy gland. Œsophagus not perforated. Pericardium lying on glands, natural.

Tracheal glands: changed into a hard cheesy mass (2 in. × $1\frac{1}{4}$ in.) lying between trachea and apex of right lung, and reaching as low as right bronchus; lung somewhat compressed by glands.

Trachea and bronchi: much injected.

Larynx: false chords a little ulcerated.

Right lung: whole of lower lobe solidified, and more or less cheesy (one cheesy mass size of a peeled horse-chestnut), or broken down into an offensive, but not sloughy, pulp; a few small cavities size of horse-beans; upper and middle lobes natural, excepting a few small solid nodules. Bronchia leading to lower lobe dilated, and full of puriform liquid. Adhesions of lower lobe.

Left lung: a large cheesy mass, unsoftened, at apex; rest of lung natural, excepting a few small cheesy and grey nodules. Adherent to diaphragm.

Liver and spleen: a few tubercles adherent to diaphragm. Heart and kidneys natural. A few of Peyer's glands tubercular and ulcerated.

Case III.

Sarah E., 9 years old.

1. No strumous disease in kindred known of.
2. Measles and hooping-cough at three years old; scarlet fever

at five years. At Christmas 1875 she began to cough; no sputa; nothing else noteworthy, until

3. *Onset of pneumonia.*—About March 7, 1876, took to her bed with fever, pain in right side, and vomiting. No shivering.

4. *Course of disease.*—She was admitted into the Hospital for Sick Children on March 21, 1876, and died there on April 17, 1876. The notes by Dr. Champneys.

(1.) She became quickly very lean.

(2.) Hectic fever throughout; temperature only once rose above 103° (39·4° C.), and then rose to 103·8° (40° C.).

(3.) No diarrhœa.

(4.) Cough dry at first, but soon became loose; and on April 10 it was noted that she brought up green sputa, almost nummular, and very fetid; this expectoration lasted until death; no hæmoptysis.

(5.) Pain in right hypochondrium.

(6.) No enlarged lymphatic glands.

(7.) *Physical signs.*—Right side of chest moved much less than left, and looked rather shrunken; heart's impulse natural; liver and spleen impalpable; dulness to percussion over the lower half of the right side of the chest; bronchial breathing (becoming more and more intense or cavernous) and bronchophony over same region; also rales, at first very sharp, but latterly gurgling.

5. *Post-mortem examination.*—Green colour of chest walls on right side, just outside and below nipple line and level.

Bronchial glands: at the bifurcation of the trachea, between the œsophagus and pericardium, exactly where there is a packet of lymphatic glands in the natural state, was a sloughy cavity the size of a date; no trace of cheesy matter, only a little black pigment here and there; a few glands close by were enlarged and glistening. Right bronchus opened into this sloughy cavity, the walls of the air-tube being quite destroyed, except in front; into the same cavity opened both the bronchia of the right lung; the cavity lying in the posterior part of the root of the lung, and the part of the lower lobe immediately behind; edges of ulcer smooth, thin, and flabby; what was left of mucous membrane very dark red; destruction of bronchus began close to bifurcation of trachea. Œsophagus natural.

Right lung: middle and lower lobes, and posterior part of upper lobe, solidified throughout; posterior part of lower lobe occupied by a sloughy cavity, very irregular in shape, size of a hen's egg; walls mere slough, no attempt at demarcation; around this cavity are many smaller cavities of divers sizes, with black walls; in posterior part of middle lobe a similar cavity, size of a

small peeled walnut. Solidification reddish grey, translucent, moist; very uniform in look, excepting near the sloughy cavities. Only part of lung containing air was apex, and even this had much gelatiniform induration, diversified by an appearance like lobular (catarrhal) pneumonia. No cheesy tubercle.

Left lung: everywhere contained air, excepting a few scattered nodules in the lower lobe, which, when cut into, had all the characters of recent hepatisation; no tubercle of any kind.

Pericardium contained soft lymph and puriform liquid.

Other organs natural and free from tubercle.

Case IV.

Henry Joseph R., 2 years and 6 months old.

1. No strumous diseases in his kindred known of.

2. He always was a weak child; never walked; towards the end of 1875 he had measles, hooping-cough, and bronchitis; had a cough ever since.

3. *Onset of severer pulmonary symptoms.* — At Christmas 1876 his cough became worse; general condition became very bad; no very definite symptoms.

4. *Course of disease.*—He was admitted into the Hospital for Sick Children on January 30, 1877, and died there on February 10, 1877. The notes by Dr. Garlick.

(1.) No great loss of flesh.

(2.) Fever slight; only four times was the temperature noted to be above 100° (37·4° C.), and the highest reached was 100·8° (38·2° C.).

(3.) Cough loud and barking; no sputa.

(4.) No continual or abiding dyspnœa or lividity until the day of death. He was subject (whilst in the Hospital) to nocturnal attacks of asthma,[1] during which he became very blue, kicked about, struggling for breath; great distress; much rattling in the throat; seemed choked by phlegm; after a short time these symptoms passed away.

(5.) Indistinct swelling above right clavicle.

(6.) *Physical signs.*—Chest looked distended; inspiratory recession of base; right base smaller than left, moved less also; well-marked dulness to percussion at right apex, back and front; bronchial breathing and bronchophony in right suprascapular region, but not much reliance could be placed upon these signs, because they were almost as well marked in the left suprascapular region; general mucous rales, rather more on right side than on left.

[1] Spirandi difficultas per intervalla subiens.—*Cullen.*

(7.) On the day of death, the dyspnœa and lividity were continual; much recession of base of chest; rales abundant all over lungs; voice loud, but hoarse.

5. *Post-mortem examination.* — Tracheal glands: between upper lobe of right lung and trachea was a large mass composed of lymphatic glands, closely adherent to and compressing lung; it lay along inner side of trachea, reaching from bifurcation below to within three-quarters of an inch of the cricoid cartilage above; aorta, innominate artery, and subclavian artery were in close contact with the mass, the subclavian lying above it. On section, the tumour was found full of thickish, creamy pus, occupying a cavity the size of a large walnut; walls of cavity, thin, white, and cheesy within. The middle part of this abscess compressed the side of the trachea, so as to flatten it there.

Bronchial glands: an enlarged and caseating gland below bifurcation.

Larynx: natural.

Trachea: congestion of mucous membrane began at the sixth ring (passing downwards); became greater where the trachea was flattened, and continued so to the bifurcation.

Right lung: pleura densely adherent; lung much retracted; whole lung more or less collapsed; upper lobe, where compressed by abscess, in a state of iron-grey induration; lower lobe also somewhat indurated, but much less so; a few small cheesy nodules. Bronchi of lower lobes much dilated; mucous membrane much congested throughout; contents were mucus.

Left lung: adherent; anterior parts distended with air, so as to cross over to right and cover heart; two nodules, becoming cheesy, size of hazel-nuts, in upper lobe; lower lobe, œdematous. Mucous membrane of air-tubes congested, but less than in right lung.

Other organs: nothing noteworthy; no tubercle.

The points in these cases to which I would draw attention may be summed up as follows:—

I. *Disease of bronchial glands.*

1. Accompanied by perforation of the adjoining bronchus (I., II., III.)

(1.) Perforation opened into a sloughy cavity, left by destruction of the glands (I., III.); gangrenous pneumonia of lower part of lung on same side as perforation.

(2.) Perforation simply exposed an unsoftened cheesy gland (II.); pneumonia ulcerative or phthisical.

2. Character of glandular disease.

(1.) Strumous, that is to say, tuberculous (I., II.); pneumonia

also strumous, going on to gangrene in one case (I.), and phthisis in the other (II.); general tuberculosis in both cases.

(2.) Not strumous (III.); genuine gangrenous pneumonia; no tubercle found in lungs or other organs.

II. *Disease of tracheal glands* (IV.), forming a strumous abscess; compressing trachea and upper part of lung on same side; chronic bronchitis of both lungs; chronic pneumonia of part of lung compressed.

I will now make a few remarks upon my cases, such as they appeared to us during life, so as to render them serviceable towards an empirical history of this form of disease.

1. No definite strumous ancestry could be made out, even in the strumous children.

2. Possible causes of the glandular disease. In the strumous children: hooping-cough six months before the pneumonia (II.); measles, hooping-cough, bronchitis, and chronic cough within the twelve months which preceded severe pulmonary symptoms (IV.); no such causes noted in I. The non-strumous child (III.) had cough for two months or more before the pneumonia.

3. Onset of pneumonia sudden in all the cases of bronchial perforation.

4. Hectic fever in cases of gangrenous and ulcerous pneumonia; not in the case of slaty induration.

5. Asthma in case of suppurated tracheal glands.

6. Signs of glandular disease very obscure. In I. there was decided dulness to percussion behind the upper half of the sternum, a dulness probably due to disease of the tracheal glands, and certainly not due to disease of the bronchial glands, which lay behind the pericardium. In IV. there was some fulness above the right clavicle, due to suppuration of the tracheal glands.

7. All the cases of chronic pneumonia of the base of the lung raised at first the question of diagnosis from chronic pleurisy, a diagnosis not always easy to make.

8. Pericarditis in the cases of sloughing of the bronchial glands.

ON SPASTIC PARAPLEGIA.

BY

SAMUEL GEE, M.D.

The following cases relate to a form of disease which possesses these essential characters :—Constant rigidity of the legs, or of the legs and arms, which increases when the limbs are handled, disappears under the influence of chloroform. The functions of the limbs are, as a matter of necessity, much impaired.

Other characters, accidental or negative, are these :—

The lesion is congenital, or at least begins in early infancy.

The affected muscles act well to faradism.

The nutrition of the muscles is well kept up. (In the first case, one calf did waste somewhat after the disease had lasted eleven years. These wasted muscles were not wholly relaxed by chloroform.)

No tremors.

Intellect and sensation natural.

No lesion of micturition.

General nutrition of body good.

Back apparently weak in all cases.

Choreiform movements of face in two cases.

Tendency to convulsions in two cases.

Painful cramps in the legs in one case.

Case I.

John B., aged 10 years when admitted into the Hospital for Sick Children, September 1874 : seen for last time June 1877.

Nothing found noteworthy in the accounts given of his kindred.

Earliest nervous symptoms.—1. Convulsions, lasting three days, at 14 days of age ; no more convulsions until he was 12 years old.

2. Tendency to draw legs up, noticed at 9 months of age by mother.

3. Never able to walk; when 8 years old, he began to be able to shuffle along by holding on to something.

Backward in development; only three teeth when a year old; did not talk until 4 years old; did not hold head up well until 3 years old.

His symptoms whilst he was in the Hospital were the following:—

1. General symptoms: nutrition good; well grown for his age; no fever; his temperature was taken daily night and morning, and was always natural, excepting during an attack of erysipelas of the face, which lasted 13 days.

2. Shape of head: natural.

3. Masturbation: never observed, although closely watched for.

4. Intelligence: natural; he is clean in his habits.

5. Eye symptoms: none; no strabismus, no ptosis; pupils equal; fundus of eyes natural as seen by ophthalmoscope.

6. Tongue: protruded easily and straight.

7. Speech: natural; no halting between the words.

8. Swallowing: natural.

9. Face: no paralysis; slight occasional twitching of corners of mouth.

10. Neck: natural.

11. Rigidities:

(1.) Left leg: usually lies slightly flexed at hip and knee; extended and slightly inverted at ankle; toes strongly flexed. He can voluntarily extend the hip, and flex the knee to a slight extent, but he has no power over the ankle (which only becomes more strongly extended the more he tries to flex it), nor over the toes. The knee can be completely extended by the observer without putting the boy to pain; however, the hamstrings become very tight. Sometimes, when the limb is quite quiet, the ankle is not extended nor are the toes flexed so strongly as usual; but a very little handling of the leg brings back the ordinary state.

(2.) Right leg: at first sight looks almost natural; when it is let alone it lies flat and straight; but any slight irritation of either leg is followed instantly by flexion at the hip and adduction of the thigh; the knee remains extended, and its extensors stand out in strong relief. The result of these movements is, that the right leg is brought across the left; the more the boy tries to resist this movement, the more rigid do the muscles become; he tries to push the right leg down with his hands, but without avail. He cannot flex the knee at all; forcible flexion of it causes pain in the upper part of the popliteal space. The ankle is extended; he seems to be able to put the peronæi into play a little, but he cannot flex the joint or invert the foot. He can bend and extend the toes a little; the big toe often diverges strongly from the rest. The right leg is more refractory than the left.

(3.) Left hand: the boy has some difficulty in opening it; when it is opened, the fingers diverge and look stiff; rest of arm natural.

(4.) Right hand: grasps pretty well, but not always equally with all the fingers; when opened, fingers diverge. Right hand less manageable than left; hence the boy is left-handed. Right arm natural.

(5.) Some weakness in muscles of back; he cannot sit up well in bed unless he hold on to its sides.

(6.) Rectus abdominis becomes hard when the skin of the abdomen is irritated.

12. Cramps: whilst he was in the Hospital, he passed through an attack of erysipelas of the face. On the fourth day of the erysipelas (temp. in axilla: 100·8°—103°) he began to suffer from painful cramps in his legs, lasting for a few seconds at a time, and relieved by rubbing. All night he cried out with the cramps in both his legs. On the fifth day (102·4°—103·6°) we noted these: cramps confined to left leg, which is strongly flexed at hip and knee, and extended at ankle: every now and then these conditions become more strongly marked all on a sudden; the strongest contractions last not more than for a minute at a time. Touching the leg causes slight contractions, but they are nothing like so strong as the spontaneous cramps. Right leg strongly extended, as before described. The boy dislikes his legs to be touched, but likes them to be rubbed. The erysipelas and cramps continued. Twelfth day: for last two or three days he has been very restless, always wishing to be moved; painful cramps in left calf, sometimes in right; both calves very rigid; back likewise rigid; yesterday arms noted to be very stiff; neck not so; opens mouth well. Thirteenth day; last day of the erysipelatous fever (100·4°—101°); only now and then complains of cramp. Seventeenth day: eruption quite faded to-day; cramps have almost ceased. A day or two afterwards they wholly ceased, and did not recur.

13. Muscular atrophy: the right calf gradually became wasted and flabby; all the other muscles remained firm and well nourished.

14. Micturition: he was sometimes a long time in passing his water, but not always so; no dribbling.

15. Convulsions; one day he had a fit of general convulsions, and the next day another; he lost consciousness, foamed at the mouth, and bit his tongue. No cause was discoverable. Temperature remained natural. These were the only convulsions since infancy, nor did he have another whilst under observation.

16. Tremors: none.

17. Sensibility and other senses: natural.

18. Effect of chloroform: on September 25, 1874, he was

narcotised by chloroform; the rigidities completely disappeared. On February 26, 1876, we found that the rigidity of the right calf (the atrophied calf) was not wholly relaxed, even under the deepest narcotism from chloroform.

19. Effect of hemlock: whilst he suffered from the painful cramps, and afterwards, we tried what relief hemlock could give. At 3·12 P.M., we gave him half an ounce of succus conii, the best that could be procured: p. 80, r. 37. 3·32 P.M., complains much of pain referred to left ham; muscles stiff and hard as ever; no relaxation of orbicularis palpebræ: p. 100. 4·40 P.M., soon after last note began to cry with pains referred to ham, this lasted nearly an hour; now, p. 106, very irregular, that is, sometimes very frequent, sometimes relatively infrequent; no relaxation of rigidity. Oct. 13, to take half an ounce of succus conii every morning. Oct. 20, increase daily dose to one ounce. Oct. 23, give an ounce and a half every morning. Oct. 27, let him take two ounces of succus conii every morning. On Nov. 3, we could not discover that the hemlock had any effect whatever, and so we left it off.

20. Faradism: in September 1874, all his muscles acted well to very slight faradic currents. February 1876, right tibialis anticus and peronæi contract well, also the other muscles.

A drawing of this boy is preserved in the portfolio of the Children's Hospital.

Case II.

Alice B., aged 8 years and 7 months when admitted into the Hospital for Sick Children, June 26, 1876.

Nothing found noteworthy in the accounts given of her kindred.

She was brought up by hand; was late in teething; but is said to have begun to talk when she was a year old.

Earliest nervous symptoms.—1. When a baby she did not move her legs about, but kept them stiff, so that her mother had some difficulty in washing her on this account. She made no attempt to stand until twelve months ago (when seven years and a half old); when she was put on her feet, her legs went stiff; of late has been able to stand with help; stands on toes. 2. When two years old she began to suffer from fits of convulsions; the first lasted for an hour; she cried out, became stiff, and then twitched all over, but more on the left side than the right. During the succeeding three years she had four such fits; the last was when she was five years old; it lasted seven hours; she screamed, then became stiff; then twitched; left side of mouth drawn a good deal; the left side of her body was left weak for a day or two.

She moves her arms well. No difficulty in micturition or defæcation. Seems well in general health.

Her symptoms while she was in the Hospital were the following:—

1. General nutrition: good; no fever.
2. Shape of head: natural.
3. Intelligence: not great; but can speak, and knows a few letters.
4. Back: seems weak; sits leaning forward, with back much arched, as if no strength in it; this symptom had existed from infancy.
5. Rigidities: all the muscles of both her legs are rigid. When she is told to bend her legs, she flexes the hips and knees a little, but very little. These joints can be bent by force, but so soon as the force is removed, the leg straightens again like a spring. To handle the legs, or move or excite them in any way, increases the stiffness; the tendon of Achilles becomes very prominent. Faradism of any part of the leg makes the whole limb very stiff, it becomes flexed at the hip, and adducted so as to cross the other limb. She cannot oppose these movements. When put on her feet she cannot support herself at all; she cannot move her legs; they become very stiff; a highly marked talipes equinus shows itself in both legs; and if she be moved along, the limbs have a great tendency to cross. The rigidity does not disappear during sleep. Indeed there is no reason to believe that it ever intermits. When she was deeply narcotised by chloroform, the rigidity wholly disappeared. All the muscles act well, and not excessively, both to faradism and to galvanism. No paralysis of arms, face, or other parts.
6. Tremors: none.
7. Nutrition of rigid muscles: good; no hypertrophy; no atrophy.
8. Sensibility of legs: natural.
9. Effect of belladonna: the extract of belladonna, in doses of a grain and a half night and morning, brought no relief.

Case III.

Margaret B., aged 8 years when admitted into the Hospital for Sick Children, October 11, 1876; readmitted, June 17, 1877.

Her mother was dead, but nothing was found noteworthy in the accounts given of her kindred.

The child was put out to nurse; when she returned home at twelve months of age, her condition is said to have been much the same as it was when she was admitted into the Hospital; in

respect, that is to say, of the paralysis and rigidity. She never walked; she never was able to feed herself.

Her symptoms whilst she was in the Hospital were the following:—

1. General symptoms: nutrition good; no fever, excepting during an attack of measles.

2. Intelligence: probably natural; can speak; is clean in her habits.

3. Seems weak in the back; sits up badly, bent double; tendency to throwing back of the head.

4. Face: articulation imperfect; when she speaks, her mouth moves like a case of chorea; occasional meaningless contraction of muscles of face.

5. Rigidities: when the arms and legs are observed, they become stiff.

(1.) Arms tend to be brought forward at the shoulder, and rotated outward; elbows strongly extended; wrist pronated; hands extended strongly, and thrown back at wrist; fingers flexed. She can move the opposing muscles, but with difficulty; and the arms soon relapse into the state described. The left arm seems rather worse than the right.

(2.) Legs strongly extended at all joints; great tendency to cross; when held up, she touches the ground with her toes only.

The constant strife between the rigidities and voluntary movements causes a sort of chorea.

(3.) Chloroform, given to unconsciousness, removes all the rigidities.

6. Faradism causes all the muscles to contract well.

7. No muscular atrophy.

Case IV.

George W., aged $3\frac{1}{2}$ years when admitted into the Hospital for Sick Children, January 23, 1877; readmitted June 26, 1877.

Nothing noteworthy in the accounts given of his kindred.

He had been very rickety in infancy; was backward in teething and in mental development. He had squinted all his life.

He never could walk, and indeed never tried to do so.

His symptoms whilst in the Hospital were these:—

1. General symptoms: nutrition good; temperature never above 99°; all teeth cut; signs of past rickets.

2. Intelligence rather low, but he could not be called idiotic.

3. He seems rather weak in the back.

4. All the paralytic symptoms relate to his legs; arms natural.

(1.) When lying in bed he can move his legs well; to handle them much brings on the rigid state.

(2.) When raised up and put on his feet, his toes touch the ground; the legs become rigid, crossed, rotated inwards; knees extended, ankles extended, feet inverted; rigidity greater in left leg than in right.

(3.) Chloroform removes the rigidity.

5. All muscles act well to gentle faradic currents.

6. No muscular atrophy.

A CONTRIBUTION TO THE HISTORY OF POLYDIPSIA.

BY

SAMUEL GEE.

In November 1876, I saw, with Mr. John Dee Shapland of Thornton Heath, two boys who suffered from excessive thirst and urination; in other words, from polydipsia and polyuria (or diabetes insipidus). There was nothing special in the symptoms; but there were two points in the etiology which seem worthy of record.

I. First, the disease was inherited, an inheritance which was known to have afflicted the family for four generations. In relating the instances of the disease, I will begin with the remotest generation of which there is any tradition.

Generation I. — Patient 1. — The great-grandfather of the children I saw suffered in the same manner all his life. Story says that in his youth he became faint when unable to quench his thirst. In adult life, his customary draught was two or three quarts of water at a time. Two gallons were regularly provided for his supply through the night; nor did the supply need to be renewed before the morning. He became paralysed at twenty-eight years of age, and died at forty-four.

Patient 2.—One of his brothers was afflicted in like manner.

Generation II.—Patient No. 1 had two daughters; one inherited her father's disease, and the other did not.

Patient 3.—The former daughter, still alive, is the grandmother of the children I saw. Her excessive thirst has abated in her old age.

Generation III.—Patient 4.—The other daughter (sister of

No. 3), who escaped the family disorder, had many children, who also escaped, excepting one son, an heir of his grandfather's disease.

Patients 5 and 6.—Patient No. 3 had nine children, of whom two, a son and a daughter, suffered from polydipsia. Two other daughters, themselves unaffected, transmitted the tendency to their children; thus :—

Generation IV.—Patient 7.—One of these unaffected daughters of patient No. 3 had five children, of whom one, a son, inherited his great-grandfather's complaint. This patient is now eighteen years old, and is thought to be less thirsty than formerly.

Patients 8, 9, 10 and 11.—Another unaffected daughter of No. 3 had four male children, all sufferers from their great-grandfather's disease. Two of these children died—two are living, namely, the patients I saw. The mother, spared by the polydipsia, died of phthisis soon after the last child was born.

II. Secondly, the disease was congenital. Patient No. 8 cried for many hours after his birth, and could not be comforted. His mother soon suspected that he might perchance be tormented with the family thirst, and she begged her nurse to give him some water. At first the nurse refused; afterwards she gave water to the child, and straightway his cries were stopped for the time. He died at six months of age, and of suffering from thirst. Patient No. 9 was like his brother, excepting that he lived four months only. The living children are aged nine years and eight years respectively; their thirst has been unquenchable since birth.

A CASE

OF

EXTROVERSION OF THE BLADDER IN A FEMALE CHILD, WITH DISSECTION.

BY

FRANCIS HENRY CHAMPNEYS, M.B.

At the latter end of February I was called by one of the midwives employed in my out-patient department of Queen Charlotte's Hospital to see a child, born February 14, 1877, which she said was afflicted with a tumour such as she had never seen or read of. She added that the parents were very anxious to have it cut off.

On my arrival, I found what I had expected from her description—an extroverted bladder—and then learnt the following facts:—

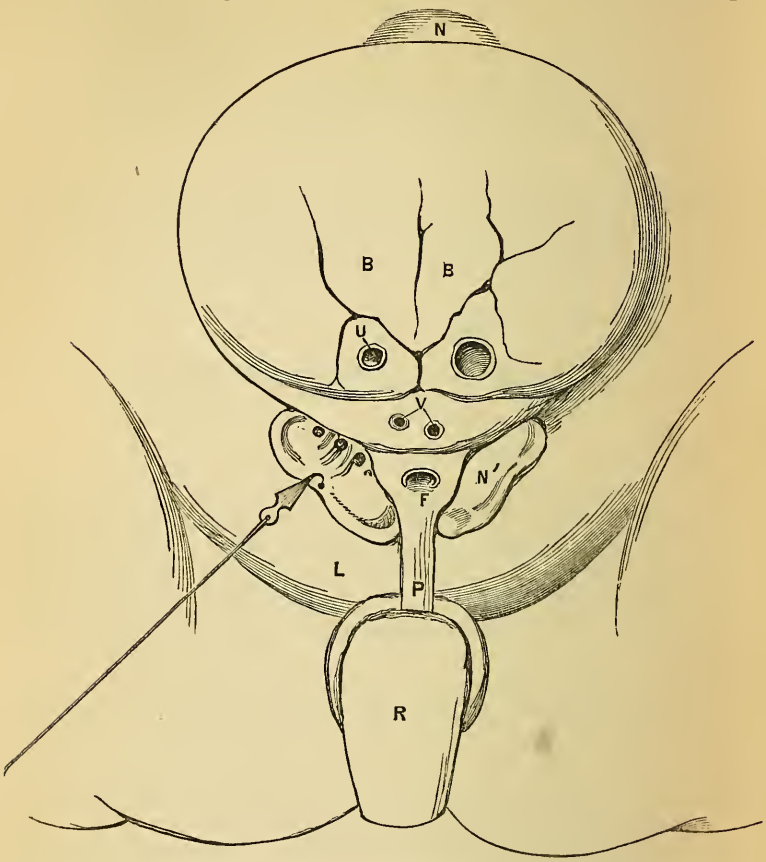
The mother was aged eighteen, the father twenty, a labourer; both were healthy as far as could be ascertained; there was no history of congenital deformities on either side, nor of syphilis. This was the first child, born at full time. There was no history of maternal impressions.

The labour was an easy and natural one, only lasting three and a half hours. Repeated inquiries elicited the fact that the navel-string was split, just before reaching the child's body, into two cords; it separated naturally on the fifth day. There was no adhesion of the membranes to the belly or any part of the body.

The child had always strained since birth, and on the seventeenth day a prolapsus ani occurred.

Three weeks after birth a rash appeared round the chin, somewhat scaly; the palms of the hands were rough, and were said to have been so since birth. No snuffles. Nothing distinctive in the way of a rash round the anus, though all the skin on the buttocks and thighs was dusky red, probably from urine.

There was congenital talipes equino-varus of the left leg,



The parts after soaking in spirit, which had the effect of rendering them more diagrammatic.

N. Navel.

B. Bladder, showing two lateral and one mesial fissures, the latter indicating its occasional separation into two halves.

U. The right ureter, natural size; the left shows the size to which they could be dilated.

V. Two openings into vagina.

F. Blind fossa, entirely posterior to vagina.

N′. Left nympha, the right drawn down by a hook to show the crypts and frænа.

L. Labium majus.

P. Perineum.

R. Rectum prolapsed.

and the whole leg from the trunk downwards was wasted, the left patella even being slightly smaller than the right.

The child had been considered a female, and named accordingly.

The lower part of the trunk presented the following appearances. In the normal situation of the navel none was to be seen; the lower third of the abdomen was occupied by a large, round, convex, purple-red, moist, easily bleeding body, towards the lower end of which were seen two papilliform prominences about one inch apart, into which a probe could be passed for some inches, and out of which clear fluid was seen to flow, at times by drops, but at other times (especially when, after continued suckling in the recumbent position, the child was raised up and began to cry) in fine jets an inch or two long. The mother said as much as a tablespoonful was sometimes passed at once. The surface of the large red body was not smooth, but thrown into numerous wrinkles and prominences, and at its lower end there was one mesial wrinkle, making that part seem bilobed. From the lower end of this wrinkle two others ran upwards and outwards; and from these, again, two others ran downwards, insulating the papilliform prominences. The margin of the large body passed into the normal skin by a boundary-line of smooth bluish tissue, such as lines a hare-lip. This large body was smaller in the recumbent position, and when the child was quiet it could even be inverted and the two edges approximated, but became larger in the erect posture, and when the child strained or cried. At the top of the large body was seen a brown mark of semicircular form, quite flat, and presenting none of the usual button-form of a navel. The summit of this body had a glazed appearance, as if epidermis were beginning to grow on it; the margins of this glazed part were irregular.

The anus seemed situated unusually far forwards; there was a projection of some two inches of bowel, which could be returned, but was immediately protruded again by a combination of straining and hiccough, which was very persistent. Fæces passed freely. From the anus a mesial raphé ran upwards or forwards.

Generative organs of ordinary form there were none, male or female. Below the papillæ above mentioned there was on each side a fold of wrinkled integument like a little lappet turning upwards; it was somewhat semilunar, the convexity upwards; on drawing the lappet down, the furrow which it formed with the surface was seen bridged over by several little frænа, by the side of which were little blind pits or depressions. The little lappets contained no trace of a body harder than themselves. Between them there was felt to be an absence of any pelvic bone, but each of them was situated on a hard body feeling like a tuberosity, continuous with the pelvis.

Beyond and external to these lappets was seen on either side

a fold or redundancy of smooth skin, larger than the above, but, unlike them, not lappet-like, but with a broad base. These folds of skin contained nothing.

There was no appearance of any penis, urethra, vulva, or vagina, perforate or imperforate.

Very close examination, however, detected below the papillæ and above the lappets, nearer the middle line than either, two small round holes with slightly raised edges, their diameter being about that of an ordinary pinhole. They were equally distant from the middle line, but the right was slightly higher than the left. A probe could be inserted into each about three-quarters of an inch.

There was no rupture, inguinal or other.

These appearances made the sex very doubtful, admitting of more than one interpretation. The large red body was of course the extroverted bladder, the pigmented mark at its summit was the umbilicus, the papillæ were the openings of the ureters; but with regard to the folds of integument and the small holes, there was nothing distinctive of either sex, for the larger, broader folds of skin, which I took for the outer integumental folds of the cloaca of the embryo of undetermined sex, might be either[1] labia majora or the two halves of a scrotum with undescended testicles. The smaller lappets, which I took for the inner integumental folds, might be either the ununited nymphæ or the integument of the lower surface of an ununited penis, the corpora cavernosa of penis rclitoris being absent. The two smaller openings might be either the vasa deferentia, no sinus pocularis intervening, or the openings of Müller's ducts, which had not fused to form a single vagina. The direction in which a probe went into these, namely, upwards, was also no help, for this might be the direction either of the vasa deferentia with retained testicles, or of the ducts of Müller (double vagina).

There yet remained one mode of investigation; if the child were male, nothing should intervene between a finger in the rectum and the base of the bladder. The finger in the rectum detected a body about the size of a hazel-nut in this position, and this I believed to be the uterus.

I showed the child at the Abernethian Society on March 8, and gave it as my opinion that it was a female for the above reasons; the external appearances were also less unlike those of a female than a male child, but the more one thought of embryonic equivalents, the less weight this argument seemed to have. I believed this to be a case of extroversion of the bladder in a female child. I believed the larger and smaller integumental folds to represent

[1] Quain's Anatomy, ed. 1867, vol. ii. p. 1001.

the labia majora and minora respectively, divaricated in correspondence with the divarication of the pubic bones, and I believed the two smaller openings to be the openings of Müller's ducts or of a double vagina, leading possibly to a double uterus.

The child continued to take the breast well till March 17, after which it began to grow weaker. On the night of March 12 it had convulsions, and died at noon on March 13. I was thus prevented from showing the child at another Society, as I had intended, but had fortunately been able to get a drawing made by Mr. Godart the day before its death. The drawing is in St. Bartholomew's Museum. (Series 31.)

The day after its death, Dr. Barlow, of the Children's Hospital, very kindly made a careful post-mortem examination with me, of which the following are the notes :—

Talipes equino-varus, left leg wasted from trunk, left patella somewhat smaller than right.

Abdominal swelling pale, moist, scarcely prominent, width $1\frac{7}{8}$ inch, length $1\frac{3}{4}$ inch.

Measurements.—From ensiform cartilage to navel = $4\frac{1}{2}$ inches. From navel to anus = $2\frac{1}{2}$ inches.

Pelvic measurements, interspinous (least) = 3 inches.

Interpubic = $1\frac{1}{2}$ inch.

Conjugata vera (to a line joining pubes) = $1\frac{1}{2}$ inch.

Interacetabular (internal) = 2 inches.

Intertuberous (between tuberosities of ishia) = $1\frac{1}{2}$ inch.

Diameter obliqua (to end of opposite pubic bone) = 2 inches.

The ilia are so far back that the anterior superior spines are almost on the same plane with the anterior surfaces of the bodies of the vertebræ.

Heart.—The foramen ovale widely open.

Umbilical vein patent in nearly its whole length, which was greater than normal.

The arrangement of the abdominal viscera is far from normal.

The duodenum descends vertically to the upper border of the third lumbar vertebra, where it has a long peritoneal attachment to the lower end of the inner border of the right kidney and the brim of the pelvis, at this point making a sudden bend to the left. The small intestine natural.

A common mesentery suspends the whole of the intestines, large and small, except the duodenum and descending colon, the cæcum can be drawn away five inches from the right iliac fossa, the whole attachment exceedingly loose, no diverticulum, the rectum suspended by a long and very loose mesentery, rendering prolapse very easy.

In the pelvis a well-developed uterus, with all appendages complete; two well-developed ovaries; at the apex of the left a small additional acinus, apparently identical in structure. Round ligament plainly seen on each side going to the internal abdominal ring, which is opposite the labium majus. Recto-uterine folds well developed.

The kidneys, ureters, and pelvis, with all its contents, were removed and carefully dissected by me, an injury to my hand, which prevented obstetrics, giving me this opportunity.

The notes of this subsequent dissection are as follows:—

Arteries.—The *common iliacs* bifurcate normally, the external having a normal course and distribution, but the distribution of the internal iliacs, which are much the larger, is altogether abnormal, for there is no division into anterior and posterior trunk, but the arteries are continued as the hypogastrics, which give off along their course all the branches of the internal iliacs separately. The vesical arteries are given off last of all; their trunks are large, the largest of them being about half as large as the external iliac. The uterine and vaginal arteries are given off just before the vesical, and are of a fair size. The vesical are three in number, and run to that part of the abdominal wall which corresponds with the back of the extroverted bladder. The hypogastric arteries terminate in the back of the umbilical scar, about half an inch apart, and are obliterated before their termination. From the upper part of the umbilical scar rises the umbilical vein, patent in nearly all its course.

Renal arteries normal.

The internal iliac veins are distributed in a manner corresponding with their arteries, and likewise possess no primary bifurcation; their distal branches are the vesical veins.

The ovarian vessels normal.

Nerves.—Principal nerve trunks of pelvis normal.

Recti abdominis muscles inserted normally, insertion $1\frac{7}{8}$ inch apart. From the inner borders of the insertions of the recti muscles a tough strong aponeurosis stretches across behind the posterior wall of the bladder.

Generative organs.—The two small openings described above, below the openings of the ureters, are found to open into a single well-developed vagina, and are therefore perhaps in their nature two perforations of a hymen. On the other hand, they look so much like the openings of ducts, that, considering the absence of any external appearance like the opening of a vagina, it is not unlikely that they may be the openings of Müller's ducts, remnants, in fact, of a double vagina, of which the septum has disappeared.

The thickness of the tissue through which they run is considerable, and very unlike a hymen. Length of vagina 1 inch.

The cavity of the uterus is normal. A probe can be inserted three-eighths of an inch into each Fallopian tube, when the calibre narrows so as not to admit even a bristle. It measures from os externum to fundus five-eighths of an inch. Extreme width of cavity at fundus three-eighths of an inch.

Both ureters are much dilated at various points, especially at their lower end. The diameter of their largest parts being more than a quarter of an inch. No obstruction anywhere. Right ureter = $6\frac{1}{2}$ inches long, left = 5 inches long. Both ureters run down into the pelvis, and curve up again to their orifices.

Kidneys.—Both pelves markedly dilated, but tissue healthy; surface lobulated.

Spina bifida.—Last lumbar vertebra well developed; spinous processes of three upper sacral vertebræ feebly developed; the last two do not meet, and the spinal canal is patent. A drawing of the internal parts is in the Hospital Museum. (Series 31.)

Remarks.—It is not my intention to write a complete treatise on extroversion of the bladder, for there are many good accounts of the malformation; nor is it possible to make a complete table of the cases even in one sex, for the literature is far too copious. It will, on the whole, be most convenient to use the principal features of the case just described as the text on which to comment.

Sex.—With regard to the relative frequency of this deformity in the two sexes, there is no real doubt that it is comparatively uncommon in females. Schneider, Meckel, and Velpeau, indeed, doubt this; Meckel because he was able to find many female instances; Schneider believes the relative infrequency of recorded female cases is due to female modesty; and Velpeau, who expresses the same opinion, adds that the deformity is more easily concealed in a female, and that in many cases the sex is not stated. Earle collected sixty-eight cases, of which sixty were in males, eight in females. M'Whinnie had himself seen nine cases, two being females. Mr. Holmes states that he has never seen a female case. Sir H. Thompson (Holmes' System of Surgery, 1870, vol. iv. p. 882) had seen eight cases, of which two were in females. Dr. Braxton Hicks, in his Croonian Lectures for 1877, gives a table furnished from Mr. Bryant's notebook as follows:—

	No. of Cases.	Male.	Female.	Doubtful Sex.
1. Spina bifida	30	13	17	...
2. Extroversion of bladder . . .	20	14	2	4
3. Epi- or hypo-spadias . . .	26	22	2	2
4. Hare lip	64	44	20	...
5. Do. with cleft palate . .	21	17	4	...
6. Malformation of bowel . . .	8	2	6	...
7. Do of extremities (excluding talipes)	46	27	19	...
8. Malformed ears	4	3	1	...

On which he remarks, that in deficiencies of the anterior median line males far exceed ; in those of the posterior median line, females rather exceed. In anomalies of the heart, cyanosis is much commoner in males (comp. St. Hilaire, "Anom. de l'Organisation," Schüler, Nasse, Meckel).

Benjamin Phillips collected twenty-one cases in females. Duncan attributes the comparative frequency in males to greater complexity of their generative organs.

Nomenclature.—The deformity has been known by various names, but principally by two, "Extroversion of the bladder" and "Ectopia vesicæ." With regard to the latter, Vrolik has confined it to a certain class of less severe cases, in which the abdominal walls only are cleft, the bladder projecting between them, but being itself perfect. It would seem advisable to use it for the future in this restricted sense. Mr. Holmes suggests the name "Congenital hiatus of the bladder;" this represents the truth, but since it does not represent the whole truth, there is no sufficient reason for discarding the well-known name "extroversion."

Degrees of deformity.—The lowest or slightest degree of deformity tending to extroversion is that observed by Mr. Mayo, in which the pubic bones were five inches apart, but there was no fissure of the abdominal walls, but only a weak linea alba forming a hernial pouch containing a bladder, which was perfect and held a large calculus.

The next degree is that described by W. Vrolik under the name "Ectopia vesicæ," the bladder being perfect, but protuding through a fissure in the abdominal walls (Taf. 30, fig. 1). In this condition inversion or prolapse of the bladder is possible, either through the urethra or through the urachus if that be patent (Froriep), though Rudolphi states this to be inconceivable in animals possessing an allantois.

The next degree is that commonly observed, in which both bladder and abdominal walls are cleft, as in my case.

The highest or greatest degree of deformity is that in which the

extroverted bladder is separated into two halves by the opening of the intestine (Bartels, Berlin Museum, No. 3077 and 9482; Retzius, p. 532; Friedländer, Taf. 22, figs. 8 and 9; Rose; Fränckel; Meckel, vol. i. p. 734). The reason of this division will be seen when we consider the causes of the deformity.

Double insertion of umbilical cord.—About this fact there is some doubt in my case, but abnormalities of the umbilical vessels have been noticed. Dietrich describes the umbilical vessels running separately to the placenta.

Umbilicus.—This is usually a flat pigmented spot, as in my case; it was formerly described as *absent*, not being recognised under its changed appearance. Schneider mentions a case in which the extroversion did not reach up to the navel, some normal skin intervening. Benjamin Phillips mentions a case in which the tumour reached *above* the navel; this seems hard to represent to one's mind.

In connection with this, the *urachus* is usually said to be *absent*, but this is an incorrect expression; it would be more correct to describe it as not differentiated from the bladder. Ayres describes a case in which the urachus was prolonged into the umbilical cord, and being included in the ligature, fatal peritonitis resulted. This connection of events is not easily intelligible. It may be well to notice here that the word "urachus" is used by Allen Thomson (p. 134) to signify the proximal end of the allantois, that is to say, the part abutting on the Wolffian bodies; this is, however, incorrect, though the allantois is described by some embryologists under the name "urachus."

The low position of the umbilicus is a point universally observed; it is due to arrest of development (Chaussier, Meckel).

Position of anus.—This is generally placed more anteriorly than is usual.

Prolapsus ani.—This is mentioned in a case described by Vrolik. In the cases presently to be alluded to by Bartels and others, in which there was a protrusion of intestine through an opening between the two separated halves of the bladder, it was not the rectum but lower end of the ileum which opened there (the rectum being generally absent); there was therefore no sphincter ani, and the conditions would be different.

Hernia.—In my case there was none; in some cases herniæ have been noticed. Thus in a case mentioned by Schneider (Case 10) there was a congenital umbilical hernia; in Handyside's case a double inguinal hernia occasionally happened on exertion.

Absence of clitoris.—This is often but not always the case. In Sir Astley Cooper's case, the clitoris and its muscles were present, but in two separate halves; the corpora cavernosa passed

towards the glans, not straight, but in a semicircular direction; the two halves of the glans were two inches apart. Both penis and clitoris are often absent (Schneider); the nymphæ may also be absent (Schneider, Case 10); all the external genitals may be absent (Meckel). In the male the testicles are often in the abdomen, and are in that case often undeveloped (Breschet, Ayres); but they may be in the cleft scrotum and well developed (Handyside).

Opening of vagina.—In my case this was double; and for the reasons above given I think it not unlikely that this was a remnant from the period when the ducts of Müller, which form the uterus and vagina as well as the Fallopian tubes, were still separate, not having fused. This fusion takes place from below upwards, the Fallopian tubes marking the point of termination of the process above (Virchow). From various degrees of arrest of this process of fusion arise those varieties of double uterus, or double uterus and vagina, of which many cases are on record, some of them in cases of extroversion of the bladder in females (Saviard, Retzius, Foerster, Rose, Fränckel); in these cases the vagina may be imperforate (Bartels, Meckel, Coates, Schneider). In one very remarkable case (Meckel), the double uterus ended in a double vagina, which opened into the sheath of the umbilical cord. In a case related by Meckel an opening from the bladder led into a uterus-like cavity (gebärmutterähnliche Höhle.)

Prolapse of uterus has been observed in several cases (Ayres). It is attributed to deficient support in connection with divarication of the pubic bones by Isodore St. Hilaire. In some cases it has thrown doubts on the sex by simulating a penis; such cases are related by Breschet, and (though not in connection with extroversion of the bladder) by Sir Everard Home, and by Mahon, in whose case (that of Margaret Malaure, 1693) the possessor of the deformity pretended to be a hermaphrodite. After having deceived many physicians and surgeons, Saviard reduced the prolapsus, and with it the number of sexes.

Round ligament of uterus in Bartels' case, as in mine, was plainly traceable to the internal abdominal ring.

Divarication of pubic bones (*cleft symphysis*) is commonly said to be an invariable point in the deformity, but this is not the case (Coates, Draman, Roose, Walther, Quatrefages).

When present, it necessitates an awkward gait, the thighs being too far apart (M'Whinnie); the *diameters of the pelvis* are, of course, necessarily affected by it, the transverse bearing too large a proportion to the conjugate; and this disproportion increases with age, a fact utilised by Schroeder (pp. 12–14) in illustration of the operation of the different factors which combine to

render the adult's different from the child's pelvis. The posterior edges of the ilia are pulled forwards by the weight of the body, acting through the sacro-iliac ligaments, and therefore the anterior extremities of the ossa innominata (the symphysis) tend to be drawn apart. In the normal pelvis, divarication is prevented by the symphysis, and the result is curving of the bones and increase of the transverse diameter; but when the symphysis is cleft, this force can act unopposed, and separates the pubic bones more and more.

Isodore Geoffroy St. Hilaire remarks on the solidarity existing between the bones and their superjacent soft parts (clitoris or penis), which are generally cleft in correspondence. But as extroversion of the bladder may exist without divarication of the pubic bones, so may divarication of the pubic bones exist without extroversion of the bladder (M'Whinnie, Meckel, Vrolik). In Mayo's case the pubes were five inches apart, yet the bladder was perfect, only projecting through a weak point in the linea alba, forming a hernia; it even contained a large calculus. Yet here the external generative organs were defective. Mr. Mayo remarks well that this state of pelvis bears a resemblance to that of female guinea-pigs near the end of pregnancy, and M'Whinnie adds, after Vrolik, that in birds, who have no urinary bladder, there is no symphysis (the struthionidæ, however, have a symphysis pubis); that in the two-toed sloth the pubic bones are permanently separated, and that in the mole, whose pelvis will scarcely admit a small probe, the ossa pubis unite to enclose the caudal vessels only, forming a simple hæmal arch. Lastly, divarication of the pubic bones does not necessitate abnormality of the generative organs (Vrolik, Walther).

Bladder.—This was formerly described merely as cleft. To Tenon is due the credit of pointing out in 1761 the fact that all the anterior part is *wanting*, that little, in fact, beyond the Trigone remains.

Behind the extroverted bladder an exceedingly strong aponeurosis is continued from the abdominal muscles and fasciæ. This has many times been described, and was very well marked in my case. The whole of the bladder is thus completely extra-abdominal. The protruding bladder becomes larger and more prominent as age advances, which is attributed by Tenon to distension by the intestines and abdominal pressure.

The bilobed appearance which I have described has been noticed by Chaussier and Breschet. It is of extreme value in explaining the origin of the deformity, and forms a step in the direction of that remarkable set of cases related by Bartels, Retzius, Friedländer, Dietrich, Vrolik, Foerster, Rose, and Fränckel, in which

the bladder was actually divided into two separate halves, the intestine opening between them. In a case mentioned by Palfyn the bladder and vagina were fused into a common cavity, the anterior half presenting the appearances of a bladder, the posterior half those of a vagina.

The *ureters* are nearly always dilated, and with them the pelves of the kidneys ; they are also lengthened, and instead of running straight to the bladder, descend into the pelvis and ascend again towards the bladder. In one case they were two inches wide (Petit) ; in Sir Astley Cooper's they were larger than the rectum ; in another case the right measured nine and a half Paris inches in length, the left fourteen (Mörgelin) ; in Schneider's Case 10, one kidney had two ureters. In Bartels' case the right ureter (the left kidney and ureter being absent) was convoluted, its calibre varying from that of a fine bristle to that of a pencil, and opened into the right half of a double vagina. In cases in which the ureters open abnormally, *e.g.*, into the rectum, vagina, or urethra, they are sometimes dilated, sometimes not. For instance, in Saviard's Obs. 94, the comon ureter which opened into the rectum was not dilated ; in Bousquet's case (p. 128), in which both ureters opened at the orifice of a cloaca, they were not dilated ; but in Thilow's case (p. 17), in which both ureters ended in the urethra, they were much dilated ; in this case, however, they were not specially examined as to the existence of an obstruction ; in Blasius' case (p. 52), where they ended in a similar manner, they were much dilated.

The dilatation, therefore, seems to be in some way connected with the general state of disordered development rather than with any proven obstruction ; and on this point Meckel (p. 650) remarks, that the ureters are sometimes found much dilated in other sorts of congenital deformities, as, for instance, in a case quoted by Klein of an acephalous fœtus (p. 30).

From the mouths of the ureters the urine distils by drops, or in occasional spurts (Mörgelin), as in mine ; the quantity discharged at one time will vary. In one case already alluded to, the urine was retained in the dilated ureters till a prolapsed uterus was reduced, when it flowed again, gushing out at least a foot. This was considered a proof of the existence of a bladder (Huxham) ; in one case it could be voided by a voluntary straining effort (Handyside).

The ureters may open in many abnormal situations ; into the vagina (Bartels, Haller, Van Horne) ; into the urethra (Thilow, Lieutaud, Binningen) ; in a case related by Saviard, in which there was a double uterus and vagina, the urethra opened into the left vagina, but a common ureter from both kidneys into a cloaca ; they may also open into the rectum (Oberteuffer, Meckel).

The *urethra* may end blindly behind (Coates, Nebel), though the external genitals are malformed (Vrolik); it may open into the vagina (Saviard, quoted above).

The *kidneys* have often dilated pelves. In Bartels' case the left kidney was absent, though both suprarenal bodies were in their natural position. Specimen No. 3077 in the Berlin Museum, described by Bartels, shows the right kidney so low down as to be almost in the pelvis (the suprarenal bodies not being displaced), or quite in the pelvis (Rose).

Intestines.—Among the anomalies which have been noticed in cases of extroversion may be mentioned the following:—

Diverticulum (Bartels); the small intestine opening through an unnatural orifice between two separate halves of a double extroverted bladder, in some cases prolapsed, the colon in some cases partly or entirely absent, the rectum absent or imperforate (Bartels, Berlin Museum, Nos. 3077 and 9482; Retzius, Friedländer, Dietrich, Vrolik, Foerster, Rose, Fränckel).

"Fistula ani vaginalis congenita" (Papendorp), rectum opening into vagina (Meckel; Burns, related by Duncan; Schneider, Case 12).

In one case, related by Huxham and Oliver, the rectum, which opened by a perforate anus, communicated with the vagina, which was situated so far forwards as to be in the normal position of the symphysis pubis, and through which the subject was impregnated; labour setting in, the two had to be laid into one by an incision.

"Fistula ani vesicalis congenita" (Papendorp), rectum opening into bladder in the male.

"Atresia ani urethralis" (Papendorp), rectum opening into urethra.

The *anus* is often imperforate (Retzius, Friedländer, Dietrich, Foerster, Rose, Vrolik, Fränckel, Bartels, in the cases above alluded to, with small intestine opening between the two separate halves of a double extroverted bladder). In one extraordinary case related by Bartholin, the anus was imperforate; the subject lived forty years, all the ingesta returning through the mouth. It is usual for the anus to be placed abnormally far forwards.

Omental and peritoneal adhesions have been noticed by Bartels. The mesentery was very abnormal in my case.

Arteries.—In my case all the branches of the internal iliac were given off separately from the hypogastric, which was continued as the trunk of the internal iliac, and the veins followed a similar course. In Schneider's Case 14, the branches of the posterior division of the internal iliac were given off from the hypogastric; the spermatic artery on the right side absent (Schneider); the left renal and the left kidney absent, the left spermatic being

given off from the left suprarenal (Bartels); the inferior mesenteric absent, the right renal arising just above the bifurcation of the aorta, the sacra media being larger than the renal, and bifurcating opposite the coccyx (Bartels); two left renal arteries (Schneider); absence of the left hypogastric artery (Fränckel); the umbilical vessels running separate to the placenta (Dietrich); one umbilical artery absent (Marin, Dupuytren);—such are some of the recorded abnormalities of arteries in these cases. It will be observed that, in my case, the fœtal state was maintained, the hypogastric artery continuing the main aortic current, as in the early embryo.

Sexual potentiality in the male is generally entirely abolished, for not only are the testicles (and with them the sexual appetite) often undeveloped (Breschet, Ayres), sometimes intra-abdominal, but, when they are well developed, and the sexual appetite strong, as in Handyside's case, there is generally an extreme degree of epispadias, the penis being absent or in two halves, and the vasa deferentia opening nakedly below the ureters. Besides the sterility which, apart from the question of possibility, such a repulsive deformity might well be expected to entail, nothing but accident or artifice could ever effect a junction between sperm cell and germ cell. Thus males are practically sterile.

It is different with females. In them the essential generative organs need not be malformed, and many cases of pregnancy and parturition are on record. One related by Huxham and Oliver has already been quoted; in another the delivery took place through the perineum (Thiebault), though it may be perfectly normal. It must be remembered that the mechanism of parturition may be altered by the alterations in the pelvis. In Phil. Trans., vol. xxxii., will be found a letter from Oliver to Mead with regard to a female with extroversion, in which this passage occurs: "Hunc in modum conformata, valetudine satis bona fruebatur virgo, et æterna virginitate ex necessitate laboraturam concluserant omnes quibus res innotuerat. Advenit tandem nauta quidam, cui æs triplex circa pectus erat: illam vidit, amavit, duxit, et non multo post impregnavit."

Viability.—Contrary to former preconceived ideas, there is no prejudice to life in extroversion of the bladder. Bartholin records a case aged forty, Quatrefages two aged forty-six and forty-nine respectively, and Flajain one aged seventy.

Other co-existing deformities.—Spina bifida (Berlin Museum, No. 9482, and Fränckel); non-closure of the cranial vault (Is. G. St. Hilaire); patent foramen ovale (in my case); pes varus dexter (Fränckel); pes varus sinister (my case); adhesion of fœtal membranes to edges of bladder (Wood).

Comparative teratology.—The only instance of extroversion of the bladder in animals which I have been able to find is one of a young male cat dissected by Is. Geoff. St. Hilaire ; he says the appearances were the same as in man.

Maternal impressions.—It would be indeed strange if there were nothing to record under this head. We are indebted to Schneider for the following instances :—A woman having been largely occupied during her pregnancy in eviscerating geese, brought forth a child with extroversion of the bladder. A woman fell from a pear-tree on her coccyx ; she felt as if her bowels were coming out at her navel, and her child at its birth was found to have an extroverted bladder. A pregnant woman during the French Revolution saw many people killed, fainted away, and on recovering her senses imagined she had been ripped up ; she bore a child with an extroverted bladder. A woman mentioned by Saviard attributed a smilar result to her longing for a fowl's tail.

Paternal impressions.—Schneider (Case 1) mentions that a man dreamt that his eldest son was standing by his bed with genitals just torn from his body, which was red and bleeding ; he was much alarmed. Next afternoon his wife bore a son who had extroversion of the bladder.

Theories.—The numerous theories which have been put forth in explanation of this deformity seem to fall under the following heads : —*A.* Mechanical ; *B.* Pathological ; *C.* Developmental, or combinations of them.

1. *Breschet* believes it is probably due to (1) the bladder bursting through the abdominal walls by distension or fœtal movements ; (2) the posterior wall of the bladder breaks through the anterior wall, which leads (?) to the (3) obliteration of the urethra. This is a mechanical explanation, and will be seen to be the converse of Duncan's.

2. *Duncan* believes the order of events to be (*a*) impervious urethra ; (*b*) distension of the bladder from retention of urine, not fatal because he says fœtal urine contains very little excrementitious matter, and children have been even born alive with a bladder having no outlet, some of the contents being reabsorbed ; (*c*) divarication of the pubic bones ; (*d*) rupture of the bladder. Thus the first term is developmental, the others mechanical, the force acting from within outwards.

3. *Mörgelin* adopts Duncan's view, and says that obstruction of the urethra is a sufficient cause ; but adds that the urachus is generally open in the fœtus, and instances cases in which it has again become pervious after birth in consequence of retention of urine. He modifies Duncan's view so far as to say that the

obstruction begins before the abdominal walls have closed over the bladder. Lastly, he gives out that he believes the adhesion of the bladder to the abdominal walls yet ununited has more to do with their failure to close than rupture of the bladder from obstruction of the urethra. This is therefore a mixed view, savouring of mechanical and pathological or developmental explanations.

4. *Foerster* attributes it to accumulation of fluid in the allantois, which prevents the abdominal walls from closing over it. This is a modification of Duncan's view, but is, strictly speaking, dependent on arrested development.

5. *Velpeau* believes it to be due to pathological causes, and not to arrest of development. The sequence of events is, according to him, (*a*) extreme thinness of the lower abdominal walls; (*b*) laceration and ulceration; and he cites cases of intra-uterine laceration observed by Desalle and himself. He believes that the ulceration may begin at the hypogastric region or the root of the penis, and that the bladder being destroyed, in the second place unites with the abdominal walls; the navel may or may not be included in the destructive process, and the pubes are not separated, as usually described, but partly destroyed.

6. *Benjamin Phillips* believes it to be due to ulceration, and states that fœtuses have been seen in which the abdominal parietes only were destroyed. "In one of three months the bladder was already comprised in such a perforation, and the borders of the whole were so ragged, thin, and unequal, that it could be referred to nothing else than a laceration. The pubes are commonly destroyed, and not simply separated."

7. *Chaussier* attributes it to (*a*) failure of the anterior abdominal walls to meet; (*b*) destruction of the anterior wall of the bladder "par une cause quelconque" (!) This is an explanation based partly on arrested development, partly on a "cause quelconque."

8. *Creve* believes the deformity to be due to separation of the pubic bones.

9. *Roose*, giving the same explanation, says he believes it to be due to mechanical injury to the mother or to the fœtus in utero. Both these explanations depend on arrested development, one of them attributing this to injury.

10. *Meckel* believes it to be due to failure of the bladder to unite in front, and says that the bladder is originally a flat surface open in front, the edges subsequently uniting.

11. *Schneider* gives a similar explanation, attributing it to imperfection of the urethra. Both these depend on arrested development.

12. *Ayres* believes the order of events is as follows: (*a*) deficient development of the "urachus" (? = allantois); (*b*) failure

of the anterior abdominal walls to unite; (c) extroversion of the bladder by abdominal pressure. This depends, as will be seen, on arrested development.

13. *Rose* (in whose case the right kidney was in the pelvis) attributes the extroversion to the abnormal position of the kidney. This is presumably a theory of arrested development.

14. *Bartels* (in whose case an abnormal opening of the intestine separated the two halves of a double extroverted bladder) believes this form of defect to be due to abnormal development of the intestine, beginning before the two halves of the allantois have joined, and as Scanzoni describes a human embryo of three weeks with a perfect allantois, the deformity must have been established before these.

15. *Isodore Geoffrey St. Hilaire* believes it to be due to arrest of development because of the co-existence of complications of (1) generative organs, (2) fissure of the head, (3) umbilical hernia, (4) abnormalities of the intestine, and imperforate anus, (5) spina bifida in various regions, all showing arrest of development, and also the bilobed appearance of the bladder in many cases.

16. *W. Vrolik* agrees with the above opinion because of the co-existence of (1) occasional absence of an umbilical artery (G. Vrolik), (2) abnormalities of kidneys and ureters (Pinel, Astley Cooper, Isenflamm), (3) fissured dorsal vertebræ (Littré, Revolat, Delfin, G. Vrolik), (4) hare-lip (Dupuytren, Meckel), (5) confluent toes (Saxtorph); and he adds that the urinary bladder is probably developed in two halves, which may be separated by the rectum (probably alluding to Bartels' case and others, in which, however, the unnatural anus was *not* the opening of the rectum but of the small intestine in most cases).

17. *Wood* explains that the allantois is composed of two layers, (*a*) the outer or "vascular" layer of Von Baer, containing the umbilical vessels, (*b*) the inner or "mucous" layer of Von Baer, which connects the allantois with the intestine. "If about this period (the period of the differentiation of the sinus urogenitalis), by inflammatory change or adhesion, or some degenerative process arising from one of those specific diseases, such as syphilis, to which we know the fœtus in utero to be liable, the normal progress is arrested in the outer abdominal or amniotic layer of the allantois, without affecting the inner or mucous layer, the result at the time of birth would be the formation of the deformity termed by Vrolik 'ectopia vesicæ,' *i.e.*, a fissure of the hypogastric wall only, with a completed condition of the walls of the bladder itself. But if the morbid process extends also to the subjacent portions of the internal cylinder or mucous layer of the allantois, then, according to the position or extent of the abnormal change,

the result would be a more or less extreme degree of epispadias, an open or imperfect urachus, or a completely fissured and extroverted bladder, with separation of the pubic bones, and the other changes associated with that deformity. An important observation has been made in these cases, that, at the time of birth, the hypogastric surface of the fœtus, from the umbilicus to the genital organs, is usually found to be adherent to the placenta or its membranes, the adhesions becoming separated by the process of parturition at the parts in which the cicatricial appearance is afterwards found" (Müller's Physiology, translated by Baly, vol. ii. figs. 220 and 222). This, he thinks, explains the deficiency of the anterior or superficial portions only, and also by cicatricial contraction, the low position of the navel. The above changes occur at the second month. This explanation belongs to the heading of arrested development.

Objections.—The following objections have been raised to the above explanations:—

1. Against *Duncan's* theory.

(*a*) In animals having an allantois, stoppage of the urethra cannot cause extroversion of the bladder (Schneider).

(*b*) Duncan himself saw extroversion in a boy in whom the urethra, situated at the root of the penis, curved towards the anus and admitted of an easy flow (Benjamin Phillips).

(*c*) Duncan's theory does not account for rupture of the abdominal walls (Meckel).

(*d*) It does not account for the malformation of the sexual organs (Mörgelin).

(*e*) (1) If the bladder bursts, it need not burst the abdominal walls. (2) It does not account for the epispadias, which is an essential part of extroversion, but often exists without extroversion. (3) The penis may be cleft, though the posterior part of the urethra is closed. (4) Vrolik has found atresia urethræ in the fœtus with great distension of the bladder, but without any symptom of extroversion. (5) "Ectopia vesicæ" (Vrolik) shows that the abdominal walls only may be affected (W. Vrolik).

(*f*) (1) The fœtus makes no urine during intra-uterine life; and Duncan quotes a case of extroversion where there was a free passage through the urethra. (2) Duncan states that divarication of the pubes is the second step, and, according to his theory, this must be the case; whereas in some cases of extroversion the symphysis is complete (Velpeau).

(*g*) Co-existing deformities are certainly due to arrested development, *e.g.* (1) malformations of genital organs, (2) cleft skull, (3) umbilical hernia, (4) imperforate anus and abnormalities of intestine, (5) spina bifida in various regions, (6) bilobed appear-

ance of bladder (Is. Geoff. St. Hilaire). This is also an objection to Bonn, Roose, and Chaussier, whose theories St. Hilaire calls "destructive."

2. Against *Roose and Creve's* theory.

It implies early ossification of the pubes, which is really a late process, and occurs after the deformity is already a *fait accompli;* nor does it account for fissure of the bladder or abdominal walls (B. Phillips).

3. Against *Meckel's* theory.

The bladder is never a flat open surface (B. Phillips).

The above objections to Duncan's theory may be arranged as follows:—

1. In atresia urethræ, the allantois (urachus) should prevent extroversion, as Rudolphi says it prevents inversion of the bladder (Schneider).

2. The urethra is not always impervious (Duncan, Phillips, Velpeau).

3. The theory does not account for rupture of the abdominal walls (Meckel, Vrolik).

4. It does not account for co-existing abnormalities, which are signs of arrested development, and some of which, *e.g.*, epispadias, though essential parts of extroversion, are often found apart from extroversion (Mörgelin, Vrolik, Is. Geoff. St. Hilaire).

5. The divarication may extend *beyond* the obstruction, *e.g.*, epispadias with impervious urethra (Vrolik).

6. When atresia urethræ exists in the fœtus, there may be great distension of the bladder, but there need be no extroversion (Vrolik).

7. "Ectopia vesicæ" (Vrolik) is quite unexplained, for the rupture of the bladder should *precede* rupture of the abdominal walls (Vrolik).

8. Extroversion of the bladder without divarication of the pubes is quite unexplained, for divarication of the pubes should *precede* extroversion of the bladder (Velpeau).

9. The fœtus secretes no urine (Velpeau).

Before proceeding further, it will be well to review shortly these objections, for some of them appear to be not well founded, while those which are valid will not need to be repeated.

1. Schneider's objection that the allantois should prevent extroversion cannot be maintained. Extroversion is a totally different thing from inversion; and were this not so, cases of inversion are on record either through the open urachus or meatus (Froriep).

2. The objection that the urethra is not always impervious is certainly true, but the statement that the urethra is or is not impervious is most misleading. The cases in which an urethra

exists as such at all are exceptional, the usual state being one of complete patency, in the form of at most a demi-canal in the male; and in the female even this does not usually exist, for the whole bladder is cleft, and the urethra likewise. Therefore it is correct to describe the urethra as "absent," but not as "impervious," the usual condition being one of only too great patency.

9. Velpeau's statement that the fœtus secretes no urine is untrue. Cases of congenital distension of the bladder, such as that mentioned by Vrolik, would be quite enough to disprove it. Moreover, urea is found (though not constantly) in the liquor amnii; and when it is absent, it has probably been reabsorbed, and excreted by the mother. At any rate, its presence in the liquor amnii, even if not constant, is enough for our purpose. Again, Claude Bernard has found sugar in the liquor amnii during the first six months of fœtal life, during which period it is also present in the fœtal urine, the quantity diminishing as birth approaches. Many other urinary salts have also been found in the liquor amnii. An interesting paper on congenital hydronephrosis, by Mr. Morris, will be found in the "Lancet" for May 13, 1876, p. 708.

We will now proceed to discuss the various theories for ourselves; and, first, it will be well to state—with reference to our analysis of the theories, and our division of them according as they appeal to *A.* Mechanics, *B.* Pathology, *C.* Development—that theories appealing to more than one of these influences must be *a priori* regarded with distrust. It is surely very unlikely, even on the arithmetical doctrine of chances, that a combination of such factors should result in producing a deformity which, though not uniform, is capable of such satisfactory classification. If, therefore, we should find that of two theories which equally well explained a phenomena, one depended on two causes, and the other on one, it would so far be wise to prefer the latter.

1. *Breschet* believes the course of events to be that the rupture of the abdominal walls by means of the distended bladder precedes rupture of the bladder itself. This is to attribute to the bladder not only far more strength, but more capacity for distension, than the abdominal walls. But what other instance is there in which the abdominal walls have ever been ruptured by distension of the bladder? Again, if the bladder ruptures, why should it always (on this hypothesis) rupture along its anterior wall and in a lineal direction; and why should it rupture from top to bottom? This objection applies to all mechanical explanations which include rupture of the bladder. When rupture of the bladder occurs after birth, an excessively rare accident except as the result of direct violence, the rupture in the majority of in-

stances extends "from the attachment of the urachus through the *posterior* wall of the organ" (Birkett, in Holmes' System of Surgery, vol. ii. p. 715, 1876). Cases of rupture from over-distension are quoted there in the fœtus (Wilkinson King, Guy's Hosp. Rep., vol. ii. 1837, p. 508; also Dr. R. Lee, Med. Chir. Trans., vol. xix. p. 238); and in the adult (Mus. of Roy. Coll. of Surg., No. 1967), it is needless to say, without rupture of the abdominal walls. The rent need not be large, for a very small hole gives immediate relief to the pressure. It would be very difficult, if not impossible, to rupture a bladder from top to bottom by liquid pressure outside of the body.

Lastly, the obliteration of the urethra as a consequence of this is inconceivable, besides our former objection that in most cases the urethra is not obliterated, but only too patent.

2. *Duncan's* theory is sufficiently combated by the authors cited above, and other objections have just been mentioned. It is, however, undeniable that this is the only hypothesis which explains satisfactorily the very constant distension of the ureters.

3. *Mörgelin's* plea that the urachus is generally patent in the fœtus would imply that obstruction to the urethra more commonly resulted in patent urachus than in extroversion of the bladder. But if obstruction of the urethra is the cause of extroversion of the bladder as well as of patent urachus, patent urachus should be a commoner malformation than extroversion of the bladder. But the reverse is the fact. His idea that the obstruction to the urethra begins before closure of the abdominal walls amounts to a theory of arrested developement, but is open to the objection already named, that the urethra is not, as a rule, "impervious."

4. *Foerster's* view is much the same. It is presumed that the "allantois" means that part of the structure included in the abdomen, *i.e.*, the bladder, and that the "fluid" is the urine.

5 and 6. *Velpeau and Benjamin Phillips.*—Without denying the possibility of ulceration during intra-uterine life, no stress can be laid on the cicatricial appearance of the edges of the extroverted bladder, unless it is intended to apply the explanation very widely; for many other failures of union in the fœtus, hare-lip for instance, present a similar aspect.

Of the statement that the ossa pubis are not usually separated, but partly destroyed, I can find no confirmation.

It is hard to imagine why, if the process be one of ulceration, the bladder should so accurately apply itself to the hole in the abdominal walls in all cases. We should expect a proportion of cases in which other organs, such as the intestines, filled the gap.

7. *Chaussier's* explanation is a mixed one, and therefore objectionable; and to refer the destruction of the bladder to a "cause

quelconque," is to "darken counsel by words without knowledge." The "cause quelconque" is the whole object of search.

8 and 9. *Roose and Creve's* theory does not attempt to explain *how* extroversion of the bladder depends on divarication of the pubes. Besides, as we have seen, divarication of the pubes is not a necessary feature of such cases.

10. *Meckel's* explanation that the bladder is originally a flat surface open in front is the first attempt in our list to attribute the state of the bladder to arrested development of the bladder. The explanation, however, is not in accordance with the facts of embryology. Still it shows an appreciation of the real point to be ascertained.

11. *Schneider's* explanation again recurs to the "impervious" state of the urethra.

12. *Ayres* is so laconic, that it is hard to understand how much he means by "deficient development of the urachus" (? = allantois). This, however, includes the whole question.

13. *Rose* attributes the phenomena to a casual and most unusual feature in a single case.

14. *Bartels* goes to the root of the matter, and his explanation sufficiently accounts for the phenomena of his extreme case, and for those of the cases which he cites.

15 and 16. *Is. G. St. Hilaire and W. Vrolik* lay stress on the co-existence of other abnormalities which are certainly due to arrested development.

17. *Wood* gives an exhaustive account of the embryology concerned in the production of the deformity, and there are few points to criticise. He attributes the arrest of development to pathological causes, "inflammatory change or adhesion, or some degenerative process arising from one of those specific diseases, such as syphilis, to which we know the fœtus in utero to be liable;" and says, that "at the time of birth the hypogastric surface of the fœtus, from the umbilicus to the genital organs, is *usually* found to be adherent to the placenta or its membranes, becoming separated by the process of parturition at the parts in which the cicatricial appearance is afterwards found."

I have no doubt Mr. Wood is speaking from facts when he writes of adhesion of the membranes, but I have been unable to find a single recorded instance. The literature, however, is too large for me to claim to have read all recorded cases. Still I think that he must be speaking beyond the mark when he says this usually exists.

Again, as I remarked above, the cicatricial appearance of which he speaks cannot be quoted as a proof of pathological change, being shared by so many other kinds of deformities, unless it is

intended that it should apply to all. There is, as a rule, nothing which marks pathological change to be found in the subjects of arrested development, nor are syphilitic children specially liable to deformities. Still I believe my case to have been the subject of congenital syphilis. It is, however, a very interesting fact that, till lately, nearly all the lions, tigers, and other large felidæ born in the Zoological Gardens had cleft palates. For some years back, probably in connection with improved hygiene, this defect has been much less prevalent (Mr. W. Sedgwick, Brit. Med. Jour., July 21, 1877, p. 96).

It would, I think, have made Mr. Wood's explanation more complete to have added instances of the process of arrested union in the middle line, going beyond and above ordinary complete extroversion, or, in other words, affecting the posterior as well as the anterior wall of the bladder. These, I think, we have in those remarkable cases mentioned by Bartels and others, in which the extroverted bladder was divided into two separate halves.

There can be little doubt that the theory which attributes the deformity to arrest of development is the correct one, because the other theories do not bear inspection, some of them being even unimaginable; because of the frequency of accompanying proofs of arrested development; and lastly, because of the known development of the allantois.

The first point has been already disposed of. As regards the second, the following instances of results of arrested development coinciding with extroversion of the bladder have been mentioned above:—1. Malformations of the generative organs, too numerous to recapitulate, most of them only to be explained by reference to the development of the cloaca. 2. Cleft spine. 3. Cleft skull. 4. Hare-lip. 5. Open foramen ovale of heart. 6. Abnormalities of kidneys and ureters. 7. Abnormalities of intestines. 8. Abnormalities of peritoneum. 9. Hernia. 10. Abnormalities of blood vessels. 11. Talipes. 12. Confluent toes. 13. Low position of navel. 14. Forward position of anus.

Now, with regard to the development of the malformation, it is well known that the abdominal walls are developed from two lateral halves which approach one another, and this part is plain; the pelvis is developed in a similar way, the bones growing from behind forwards, and their anterior extremities meeting in front to form the symphysis pubis (Meckel).

With regard to the bladder, this is developed from the allantois, and the question remains as to the development of the allantois. It is often described as originating in the form of a hollow vesicle, but that would not explain our point; it is obvious that what we want is a history of bilateral development.

In Baly's supplement to his translation of Müller's Physiology, p. 86, we read: "According to Bischoff, the allantois of the mammalian embryo is developed neither from the intestinal tube, as stated by Von Baer, Rathke, Valentin, and others, nor from the Wolffian bodies, as was said by Reichert to be the case in the chick; for at the time of its first appearance, no trace either of the intestinal canal or of the Wolffian bodies can be perceived. At its earliest appearance, the allantois in the rabbit consists of a solid mass of cells projecting from the visceral plate of the tail. But in the dog this mass is at first double (fig. 13 and 14), though the two halves soon fuse together, and are converted into a single vesicle. The allantois is abundantly vascular, for it contains the ramifying extremities of the two arteries which run along the sides of the vertebral arches, and of the two veins which are situated within the walls of the visceral laminæ. These vessels subsequently become the umbilical arteries and veins. When the allantois has assumed the form of a vesicle, it then communicates both with the intestinal tube and the corpora Wolffiana, though the mode in which this communication is effected is not quite clear. The allantois now rapidly increases in size, and the two umbilical arteries in connection with it are recognised as branches of the iliac, while the umbilical veins unite into either one or two trunks, which empty themselves in the liver and the inferior vena cava. As the visceral laminæ close in the abdominal cavity, the allantois is thereby divided at the umbilicus into two portions, the smaller of which is retained in the abdomen, and is converted into the urinary bladder, while the external and larger portion, accompanied by the umbilical vessels, proceeds to the chorion, where its vessels are brought into connection with those developed within the villi of this structure. The middle portion of the allantois, namely, that which traverses the umbilicus, at first contracts into a canal, and subsequently is converted into a fibrous cord, the urachus."

Here we have a distinct account of the bilateral development of the allantois, and of its close connection with that of the hypogastric vessels. Its intimate association with the formation and differentiation of the cloaca, and its near proximity to the lower end of the intestine, are also facts well ascertained.

Reichert (Müller's Handb. der Phys., Buch viii. p. 688) also describes the allantois as beginning by two solid heaps of cells at the posterior end of the Wolffian bodies, which approach one another by degrees, and which afterwards unite and form a hollow body.

Scanzoni (Lehrbuch der Geburtshülfe, i. 86) describes a human embryo of three weeks with a perfect allantois, so that the malformation must occur before then.

Professor Wood refers to drawings of two human embryos in Baly's translation of Müller's Physiology (pp. 1585–1587, figs. 220, 222), fig. 220, after Coste, of about 16 to 20 days, the other, fig. 222, after Wagner, of about the 21st day, and to them may be added fig. 221, after Allen Thomson (Edinb. Med. and Surg. Jour., vol. cxl. 1839, p. 135, fig. 3) of about six weeks. He thus describes them: "The allantois sac is found just as it begins to be attached to the chorion to form the umbilical cord. Its outer or amniotic layer (the vascular layer of Von Baer, in which are afterwards developed the umbilical arteries, and which forms the sheath of the funis) is seen in both these specimens to be continuous with the edges of the fissure in the yet imperfectly closed abdominal wall. In the middle of this is an inner cylindrical portion (constituting the mucous layer of Von Baer), which is prolonged down to the pelvic cavity, where it is continuous with the end of the intestine, and is connected with the lower part of the Wolffian body, from which it seems to be originally developed. This union with the intestine afterwards forms the common cloacal cavity, persistent in some of the lower animals, which, according to Tiedemann, opens externally about the fifth or sixth week. By arrest of development at this stage originate those extreme cases of the deformity in which the bowel opens upon the exposed bladder or urethra, or the ureters open into the rectum. In the normal course of development the rectum and anus become separated from the ducts of the urinary and genital organs, and a common sinus urogenitalis is then found, in which open the Wolffian ducts, the ureters, and the efferent ducts of the generative apparatus. From this is afterwards differentiated the bladder, urachus, and urethra, on the one hand, and the prostate and its appendages, or the uterus and vagina, on the other." The remainder has been quoted above. Professor Wood explains "ectopia vesicæ" as arrest of union in the superficial or "vascular" layer, and complete extroversion as arrest of union in both layers of the allantois.

The points just mentioned will appear plain after reference to Coste's Pl. ii. figs. 3 and 5 (Human Ovum, 15 to 18 days), which shows the early allantois (*a*), with its two arteries and its abutment on the rudiment of the future large intestine (*i*); Pl. iii. fig. 3 (Human Ovum, 25 to 28 days), which shows the continuation of part of the allantois from the unclosed abdominal walls; Pl. iii. *a*, figs. *A* and *B*, which show a similar continuation, and also the two umbilical arteries and two umbilical veins (*n*, *n*, *u*, *u*) in the external or "vascular" layer of Von Baer, the inner or "mucous" layer being visible (*q*, *q'*) as a sac ending blindly at its distal end.

We may add, that when the process affects the posterior as well as the anterior part of the allantois, or when the two halves of the allantois fail to join behind as well as in front, we get a condition such as that described by Bartels and others, in which the extroverted bladder is divided into two entirely separate halves.

As we never get congenital fissure of the bladder without fissure of the abdominal walls, we may conclude that the process of arrest operates from the surface towards the deep aspect.

Here, then, we have an explanation accounting not only for all the various degrees of extroversion of the bladder, but for some of the best-marked co-existing malformations, namely, those of the pubic bones, genitals, intestines, and umbilical vessels.

The other frequently co-existing defects are similarly explained, namely, by arrest of development, though the cause of this arrest is at present quite unknown.

The period at which the malformation begins is fixed by the date of development of the allantois, that is, according to Scanzoni, before three weeks of embryonic life.

The degrees are seen to be principally four in number, as we stated at first, namely :—

1. Weakness of the anterior abdominal walls, constituting a hernia of the bladder (Mayo).
2. Fissure of the abdomen, but not of the bladder, constituting "ectopia vesicæ" (Vrolik).
3. Extroversion of the bladder, generally with more or less bilobed appearance.
4. Complete separation of the extroverted bladder into two distinct halves (Bartels and others).

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Retzius.—Oppenheim, Zeitschr. f. d. ges. Med., Bd. xxxii.
Rose.—Monatschr. f. Geburtskunde, Bd. xxvi.
Scanzoni.—Lehrbuch der Geburtshülfe.
Thilow.—Obs. Med., cent. ii. obs. 34.
Virchow.—Vorles. ueber. Path. Anat. der Gener. Org., 1866.

A CASE

OF

INTERSTITIAL FIBROMYOMA (FIBROID) OF THE UTERUS.

BY

FRANCIS HENRY CHAMPNEYS, M.B.

The absence of the Physician-Accoucheur and Assistant-Physician-Accoucheur at the beginning of this year left me in charge of the gynæcological and obstetrical departments of St. Bartholomew's Hospital, and brought the following case into my hands.

Emma D., always thin and spare, aged 25, married some two years, had miscarried shortly after her marriage, but had never borne a child. Her catamenial periods having, with this exception, been regular, she menstruated for the last time during the last week in March. There had never been at any time flooding or other symptom of uterine fibroid, and she observed nothing remarkable during her pregnancy till the sixth month, when she found her abdomen irregular in shape, a round hard mass being felt above the pubes.

On January 9, at 5 A.M., she believed herself to be in labour, the pains, however, were hypogastric and not sacral, and were very irregular in rhythm. Mr. Husband, the clerk in charge of the case, to whom I am indebted for some of the following notes, believing the pains to be spurious, gave a dose of castor-oil and opium, with directions that he was to be sent for when anything happened. The pains soon ceased, but returned the next day for a short time, and in the afternoon she "felt as if something had come down." At the same time she had a dull constant pain in the back, and the abdomen felt smaller; a dark brownish

discharge began to flow from the vagina, probably meconium-stained liquor amnii.

The next day Mr. Husband was sent for, and found a constriction below the navel, dividing the abdomen into two masses. There were no fœtal heart sounds to be heard. The vagina was hot but moist, the os uteri the size of a shilling, and the membranes had ruptured, though there was no history thereof; a hard tumour could also be felt. The temperature 101° Fahr.

The state of the patient seeming critical, Dr. Maberly (resident midwifery assistant) was sent for; he believed the tumour to be a fibroid, and determined to dilate the os uteri and attempt delivery. This was done under chloroform, when the tumour was found to prevent the descent of the child, which lay entirely above the pelvis. Ineffectual attempts were made at delivery, first by the forceps (which could not be introduced), and then by the crochet (which would not hold).

The temperature was now 103° Fahr.

Dr. Maberly, believing that craniotomy, or even cæsarian section, might be required, sent for me.

On my arrival I found the patient with a temperature of over 103° Fahr, a pulse of some 120°, restless and anxious; skin dry, yellow, shiny. There were no labour pains.

The abdomen presented a singular appearance; it was distended nearly up to the ribs, but a deep constriction ran across it below the navel, from the right below to the left above, dividing it into two portions. The upper portion was fairly uniform, but the lower portion was irregularly prominent, the most prominent point being nearly half-way between the navel and the pubes to the right of the middle line. The upper portion was felt to contain moveable parts; the lower portion was hard, but not very hard, and gave distinct fluctuation. Both were absolutely dull on percussion. No fœtal heart sounds. The lower portion could be moved laterally within certain limits, the hand receiving the impression that it was attached to the uterus by its lower and posterior aspect. Was it a twin, a co-existing extra-uterine fœtus, or a fibroid, or ovarian tumour? With regard to the first two, no moveable parts could be felt.

The vagina was hot, but not dry, the bed-clothes showed signs of a dark-brownish discharge, not fetid; the finger could hardly reach the os uteri, which was very high up posteriorly, and two fingers had to be introduced before any fœtal parts could be felt.

The head was found to be presenting, in the second position (occiput to right), the bones of the cranium were loose, and glided over one another, confirming the diagnosis of death of the fœtus. The os uteri was three-quarters dilated, but the inlet of

the pelvis was much narrowed by a round hard mass in front of the os uteri, felt by bimanual examination to be the same as the hypogastric swelling. Not more than two fingers could be introduced in the practical conjugata vera, and the question turned on the possibility of diminishing the size of the tumour, or of moving it out of the way. I introduced my left hand into the vagina, and with some difficulty through the os uteri, and thoroughly explored. The second tumour was clearly not within the uterine cavity, and no small moveable parts could be felt in it. It could not be an intra-uterine twin, and was very unlikely to be an extra-uterine fœtus. It was found that steady pressure from the vagina upwards and forwards tilted it somewhat over the pubes, and increased the available space, but not much. The fluctuation was so distinct in the hypogastrium that I emptied the bladder, ascertained its relations by means of a catheter, and punctured the hypogastric swelling, first with a small, then with a large trochar, employing suction, but no fluid followed. It could not, therefore, be an ordinary ovarian tumour, and was probably fibroid. I preferred the hypogastrium to the vagina for puncture, as I could see better any fluid that might come, and could also the better deal with bleeding should that occur. Since the tumour could not be diminished, I considered that turning would give the best chance of delivery, hoping that by bringing the thin end of the wedge first, I might exert enough force to tilt the tumour forwards sufficiently to allow the passage of the child. Chloroform was therefore given, and introducing my left hand, as being the smaller, I seized the left foot, and turned. The skin peeled, again confirming the death of the fœtus. The hand in the uterus ascertained that the mucous membrane opposite the tumour felt quite natural, and that there were no nodules projecting into the uterine cavity. Not without considerable difficulty and the expenditure of force greater than I should have felt justified in using had not the child certainly been dead, I succeeded in effecting delivery, meconium escaping. I afterwards ascertained that the hip was not dislocated. The placenta was situated at the fundus. I had no difficulty in detaching and expelling it by the method recommended by Professor Credeé, and no after-bleeding followed. The upper swelling was found to have completely disappeared, having sunk behind the hypogastric swelling.

A binder was tightly applied, the lower swelling being utilised as a pad to compress the uterus, which lay behind and below it.

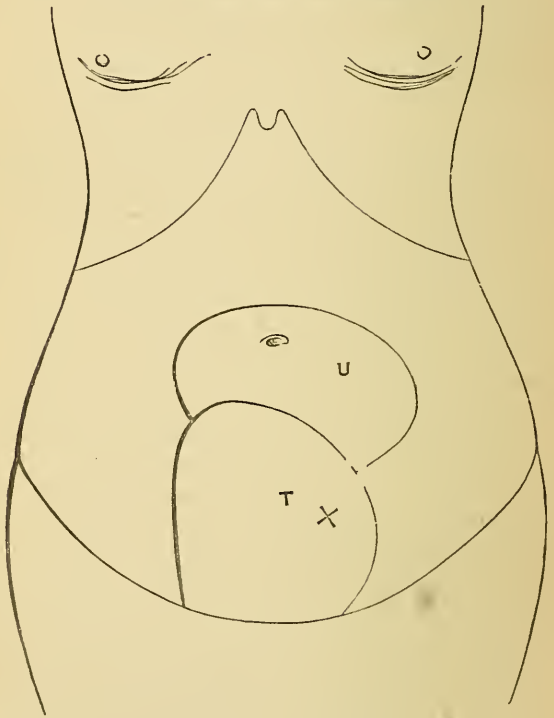
Nothing of interest followed the delivery at once.

The child was 21¾ inches long, and weighed 5¾ lb. after a few days in methylated spirits.

On January 13, the third day, I found the uterus had risen above the navel, behind the tumour, which still kept it higher than natural. Its appearance will be seen by the diagram. It was not uniformly round, but there was a projection to the right of the middle line above mentioned, and the summit of the tumour, which lay to the left of the middle line, was markedly protuberant.

DIAGRAM OF THE PARTS ON THE THIRD DAY AFTER DELIVERY.

(*N.B.*—The sides are reversed.)



U Uterus. T Tumour.
× Most prominent part, referred to.

Careful measurements were taken, which will be found in the table. The state was fairly comfortable, no bleeding, discharge free, no pain. The pulse was, however, somewhat rapid, the temperature 101°.

A few days later the discharge became offensive, and injections

of carbolic acid lotion $\frac{1}{30}$ were ordered. The temperature and pulse remained high.

The tumour steadily diminished in size (see table). On January 27 full doses of ergot were ordered three times a day. The rest of the treatment consisted in nourishing food and quinine.

As the conditions remained unaltered, and as the patient did not gain strength, she was taken into St. Bartholomew's Hospital on February 7.

On admission, her aspect was suggestive of exhaustion from some continued drain; her skin was hot, sallow, and sweating, her tongue dry, brownish-red; she was very thirsty; there was slight hypogastric tenderness, an offensive muco-purulent discharge, and a bad cough, which was due to bronchial catarrh, probably septic in origin. Temperature 102°. Pulse 120. She never had any rigors. In this condition she remained. The tumour gradually changed not only its size but its shape, the protuberance to the left of its summit becoming less and less marked, and finally disappearing.

On February 19, exactly five weeks, or thirty-five days, after her delivery, the patient felt something coming down in the vagina, and on getting up, a body protruded through the vulva. On examination Dr. Maberly found a long thick, irregular ragged mass of tissue, very fetid, occupying the vagina, protruding through the os uteri, and adherent to the left lateral wall of the uterus near the fundus. It was gently twisted off and extracted. No hemorrhage followed. The mass was about as large as a fist; there were no labour-like pains before its expulsion.

The os uteri, after its removal, was found patulous, deeply fissured up to the vaginal reflection on the right, the uterus enlarged, and felt through the vagina more plainly than natural anteriorly, and to the right and left. The part of the uterine wall from which the mass had grown was very rough. The mass weighed four ounces avoirdupois, and under the microscope consisted of fibrous and plain muscular elements in a state of fatty degeneration. No capsule. Almost at once after its removal her temperature went down to 99·6° F., but the pulse remained high. The discharge became less and less offensive, the thirst disappeared, and the cough became better.

On February 26, or six and a half weeks after delivery, the cervix was still patulous, a few nodules were felt in the uterine cavity, mostly soft, apparently disintegrating and offensive; the fundus still above the pubes.

On March 2, seven weeks after delivery, the discharge had

become watery, scanty, and hardly at all unpleasant, and the general health was much improved. Dr. Maberly noticed that the fundus could no longer be felt in the hypogastrium.

On March 10, eight weeks after delivery, the general health was good; in the abdomen, however, a hard lump, the size of a walnut, could be felt in the *right* groin. The os uteri still admitted the finger, and at the fundus a polyp-like growth, the size of a large green pea, was felt. The discharge was not offensive.

She left the Hospital a day or two later.

MEASUREMENTS.

	Jan. 13.	Jan. 27.	Feb. 15.
TAPE—			
Circumference round navel . .	31 in.	28½ in.	27½ in.
Do. do. tumour at ×	35 „	29½ „	28 „
From pubes to navel . . .	9 „	8 „	8 „
From navel to ensiform cartilage .	6 „	6 „	6 „
CALLIPERS—			
Across the widest part of tumour .	7 „	5½ „	Too low in pelvis to measure.
Height of tumour above pubes .	6½ „	5⅞ „	About 3 in.
Conjugata diagonalis from spine of the last lumbar vertebra to front of tumour at × . .	10½ „	8½ „	8 „
Height of uterus above pubes .	9½ „	Gone.	

Fibroid tumours of the uterus are of such frequent occurrence that it often happens they complicate labour, and medical literature is well supplied with records of such cases. Their influence on parturition is very various. Among the recorded cases the following bear most resemblance to the case just related.

Barnetche, in the "Journal de Bordeaux," September 1844, describes a case in which the os uteri could hardly be reached; there were no heart sounds, the meconium flowed, and the pains were weak; delivery was effected by turning, and death resulted on the third day from metro-peritonitis. Post-mortem several interstitial fibroids were found, and some subperitoneal at the fundus, one the size of a six months' fœtus. They contained sero-purulent fluid in cavities in their substance.

Späth, in the Wien. Zeitschr., N. F. iii., 1860, records a case in which a subperitoneal fibroid in Douglas's pouch, existing before conception, and producing a retroversion, grew rapidly during pregnancy, and reached the size of a child's head

at the time of labour. This became incarcerated, but was pushed up, and delivery was effected by turning. Death followed on the third day from puerperal peritonitis.

Schneider, "Schweizer Correspondenz-Blatt," 16, 1872, records a case of a woman, aged 36, who aborted in the third month, after which a tumour was felt. Two years later she again became pregnant, but her general health was so bad, with constipation, albuminuria, œdema of legs, dyspnœa, palpitations, and severe asthmatic attacks, that Breisky induced premature labour. The child weighed only 3¼ lb., but lived six weeks. After delivery Breisky discovered a large tumour of the size of a child's head to the left side of the uterus, and below it, pressing the uterus into the right hypochondrium. The placenta had to be detached by introducing the whole hand into the uterus, and the tumour was felt to grow from the uterus, but was considered subperitoneal. In the second week the patient had a severe attack of metro-peritonitis; in the fourth week severe bleeding; a few days later several attacks of pain; and great fleshy masses came away, one as large as a fist. These were found to be myomata. Fourteen days after their removal she was able to get up, and a tumour could no longer be felt. The next year she aborted in the second month, but her general health continued good.

Michauck, Inaug. diss., Leipzig, 1866. (The case was one of Küchenmeister's at Dresden.)

The patient was aged 29; she had suffered for many years from painful and copious menstruation. The doctors diagnosed anteflexion of the uterus. Fearing for her life in case of pregnancy, she for some time refused to be married, but at length consented. Three months after marriage she became pregnant. At term the child presented in the first cranial position, and was delivered by forceps. After detachment of the placenta copious bleeding ensued, the uterus remained abnormally large, but not hard or uneven; it would not contract on external manipulation. A large flat fibroid was found to be sessile on the right and posterior wall, quite immoveable, and preventing the contraction of the uterus. In the puerperal state the lochia were very copious, and there was fever. Forty-three days after delivery gangrenous masses began to come away, and the day after, the whole of the tumour followed. It was as large as a plate, oval, weighing 515 grammes 24 c.m. × 18 c.m. × 4 to 1½ c.m., and consisted of a collection of fibroid kernels. The uterus recovered itself, and the woman got well.

Retzius (Hygiea, Bd. 23, p. 190) gives an account of a case in which there was a tumour the size of a hen's egg, which was

not observed until the abdomen had become much enlarged. Delivery was natural; there was no after-bleeding. After delivery the patient had pain in the belly, and offensive lochia, with sweating and diarrhœa. Meanwhile the tumour shrank, and twenty-two days after delivery could be no longer felt.

A myoma, whether polypoid or not, need not prevent impregnation. (Schroeder, however [Geburtshülfe, p. 463], says that submucous and large interstitial fibroids very seldom permit impregnation, and that abortion in that case very frequently follows.) If pregnancy occurs, the patient becomes liable to a variety of accidents. She may abort; in relation with which it is asserted by *Forget*, Bull. de Thérap., April 1846, that the nearer to the fundus the growth is situated the earlier the abortion is likely to occur. This is confirmed by Toloczinow, Wien. Med. Presse, x. 30, 1869. Bleeding may occur during pregnancy (*e.g.*, Heiss, Bayer. ärztl. Intell.-Blatt. 33, 1861), or metritis (Schmiedt, Beiträge zur Lehre von den fibr. Geschw. des. Ut.). If pregnancy advances, it may be complicated with placenta prævia. In a case of this kind mentioned by Pajot (Gazette des Hôpitaux, 16, 1862), the placenta was situated over the new growth in the posterior wall, from which it could not be detached; it was bored through by the hand, the child was turned and extracted, the placenta followed of itself, but the patient died of after-bleeding. Post-mortem a myoma was found, interstitial and submucous, twice the size of a hen's egg. In one case recorded by Yeld (British Med. Journ., June 3, 1871), the left foot and funis presented; the child was hydrocephalic; it had been dead three days; the funis gave way at the navel (how is not stated), and could not serve as a guide to the placenta. A tumour, which was broad and sessile, 9 inches in circumference, weighing 4½ lb., was mistaken for the placenta, and removed with the blunt hook by the help of three doctors; it is related that there was no after-bleeding, but death resulted from shock two hours later. There are no notes of a post-mortem examination.

If the growth is polypoid, it need not produce abortion, even if the size of a child's head (Robertson, London Med. Gazette, February 1842); twins may even be contained in the uterus with the tumour (Meissner, Summarium, 1842); the polypus may be removed without producing abortion (Gooch, Merriman, Delaware), or it may be driven before the child's head and torn off, which may occasion serious bleeding (Lehmann, Die fibr. Geschw. des Ut. besonders als Hinderniss der Geburt, Amsterdam, 1854).

Fibroids may be the cause of severe after-bleeding, as in a case mentioned by Fiedler (Summarium, 1842, No. 1023); the

lower uterine segment contained many polypi; after delivery the uterus still reached above the navel; the fundus was hard and well contracted, the lower segment was soft. The bleeding continued until the polypi were removed, and then ceased. There were rigors and a high temperature for a few days, and then the patient recovered. This danger arises principally from inability of the uterus, and especially the placental site, to contract, owing to the presence of the myoma (Schroeder, *loc. cit.*).

The uterus may become inverted, or may rupture (Eiselt, Œsterr. Med. Jahrb., Bd. 21, p. 4; Fabricius von Hilden, Obss. Chir., cent. 1. obs. 68, p. 189), especially when many fibroids are present (Forget, Bull. de Thérap., April 1846). In the case of polypi, this is due to the obstruction to the parturient canal; in the case of the interstitial variety, to the weakness (in spite of thickness) at their site (Schroeder, *loc. cit.*, p. 463). The obstruction may be so great as to necessitate Cæsarian section (Schmiedt, Beiträge zur Lehre von den fibr. Geschw. des Uterus).

Under these circumstances the operation is very fatal. Lambert (Spiegelberg. Arch. fur. Gyn., Bd. 5, p. 110) collects fifteen cases, with only two recoveries. Storer (Obstet. Trans., xi. p. 99) first performed Cæsarian section, and then removed the whole uterus, tumour and all. The patient lived three days.

Horwitz (Petersburg. Med. Zeitschr., 145, p. 249, 1868) has recorded several cases, mostly ending fatally, of which the following are some:—

Case 1.—Normal delivery; death on eleventh day from septicæmia, from destruction of a fibrous polypus growing from the fundus.

Case 2.—Normal delivery; death on eighth day from destruction of a fibrous polypus in the anterior uterine wall.

Case 3.—Rickety pelvis, craniotomy; death from destruction of a polypus on the posterior wall.

Case 4.—Premature birth in seventh month; removal of a large fibrous polypus six days after birth; recovery.

In three out of these four cases death resulted from disintegration of the tumour during the puerperal state.

Toloczinow (Wien. Med. Presse, x. 30, 1869) gives a collection of one hundred and nineteen cases of uterine fibroids complicating pregnancy.

In ninety-eight cases birth followed at the proper time.

In twenty-one cases the patients aborted.

In forty-eight cases the presentation was mentioned.

In twenty-five the head presented.

In thirteen the pelvic pole of the body.

In ten the presentation was transverse.

Thus the presence of a fibroid affects the presentation materially.

In fourteen cases of abortion :—

In three this occurred in the third month.

In six in the fourth.

In five in the fifth or sixth.

In seven there was premature birth, *i.e.*, between seventh and ninth months.

He confirms the remark of Forget, mentioned above, the nearer the fundus the earlier the abortion.

Of the cases which arrived at maturity, delivery in thirty cases was normal.

In twenty-one cases difficult, but ended without interference.

Thirty-nine cases required help.

Death occurred in eight cases.

Five were cases of rupture of the uterus.

Three were cases of exhaustion, intra-partum.

Lambert (Des Grossesses compliq. de Myomes Uterus, Thèse, Paris, 1870) gives a collection of one hundred and sixty-five cases in an elaborate treatise.

Out of forty-four fibroids, fourteen were interstitial, thirty pedunculated.

In twelve cases of interstitial fibroids eight died.

In eighteen cases of fibrous polypi three died (one of rupture of uterus, one of septicæmia, one of convulsions).

In seven cases in which the placenta was inserted over the tumour there were three deaths.

In six cases of inversion of the uterus from fibroid tumours there were two deaths, two reductions, one uterus was unreducible, and two were removed by ligature.

Even large fibroids may be quite undiscovered during pregnancy, or the only thing observed may be the disproportionate height of uterus (Ramsey, Med. Times, August 8, 1857). Of the cases recorded which ended fatally, most seem to have died from destruction of the tumour ending in septic infection, described variously as pyæmia, septicæmia, puerperal peritonitis, metroperitonitis, &c. (Barnetche, *loc. cit.*, Späth, *loc. cit.*, Heiss, *loc. cit.*, Schmiedt., *loc. cit.*, Horwitz, *loc. cit.*, Alling, Gaz. des Hôp., 92, 1869); and in the cases ending in recovery, nearly all seem to have suffered from fever, sweats, offensive lochia, diarrhœa, &c. (Fiedler, *loc. cit.*, Rankin's Monthly Journal, July 1850, Retzius, *loc. cit.*, Michaeck, *loc. cit.*, Schneider, *loc. cit.*).

The question of increase and decrease of these growths before and after delivery will best follow a short statement of the pathology of the subject.

Scanzoni (Lehrbuch der Krankh. der weibl. Sexualorgane, 1875) states (p. 270) that fibromyomata are commonest at the fundus of the uterus, and very rare in the portio vaginalis (p. 273). Spontaneous extrusion is very rare; when it takes place the process is as follows:—The tumour existing within a capsule, the capsule is attacked by destructive inflammation, which starts from the mucous membrane; the growth is set free, and then expelled.

They may also be reabsorbed undergoing retrograde metamorphosis as the uterus does in involution. Scanzoni saw one of eleven years' growth, the size of a man's head, entirely reabsorbed six weeks after confinement.

Virchow (Die krankhaften Geschwülste, III. Band, 1 Hälfte, 1867) gives a masterly account of their pathological history.

The commonest part of the uterus to be affected by them is the part richest in muscular fibres, that is, the corpus uteri; the neck is very seldom affected; interparietal myomata are most apt to form from the thickest part of the uterine walls, *i.e.*, the posterior wall, next from the fundus, least often from the anterior wall.

As lipoma : polysarcia : : myoma : hypertrophy of the uterus, *i.e.*, myoma is a *partial* hypertrophy.

Myomata are broadly divided into soft and hard.

The *soft* myomata resemble uterine tissue in a state of *physiological* hypertrophy, *e.g.*, in pregnancy, and are rich in plain muscular fibres and vessels, the connective tissue being comparatively scanty and loose.

The *hard* myomata are more like the chronic and indurated forms of *pathological* hypertrophy, the connective tissue being more tough and abundant, the muscular fibres fewer, the vessels fewer and smaller.

The myomata which grow very large during pregnancy do not probably originate during pregnancy, but being very small before, then grow rapidly. These are always soft myomata, rich in muscular cells, and are of the kind which appear comparatively early in life.

Myomata are often cavernous in structure, containing large sinuses like the pregnant uterus, especially at the placental site. This form Virchow calls "Myoma telangiectodes s. cavernosum." This structure is seen principally in interstitial varieties. This is the kind of myoma which is often observed to vary in size, rapidly swelling up, and then shrinking; the swelling depends on a sort of erection from increased fulness of vessels, the shrinking on muscular contraction.

The softening of myomata may be of various kinds. It may depend on a state of the interstitial tissue, called by Cruveilhier "œdema." This œdema may be caused by infiltration with fluid of the connective tissue, between the nuclei or kernels of which large myoma are composed, and also between single fibres; but the connective tissue may also undergo mucoid changes, a growth of real mucous tissue, containing mucin, taking place. A tumour thus is formed which is really a myxomyoma. The round cells of this mucoid tissue may undergo fatty degeneration, and the growth soften in a second way.

In another form of softening there is no new growth, but the muscular fibres atrophy, and the connective tissue undergoes softening and disintegration, forming vacuoles without a cyst wall.

Both these forms can give rise to distinct fluctuation, and present all the physical signs of a cyst filled with fluid; but on puncture, at most a drop or two exudes, except in cases where the process of softening may occasionally have produced large cavities.

With regard to the above, the case of Barnetche, mentioned before, gives us an example; in this, the tumours, some of them subperitoneal, most of them interstitial, one of the former being as large as a six months' fœtus, contained sero-purulent fluid in cavities in their substance.

As instances of distinct fluctuation giving the impression of a cyst, in addition to my case, may be cited the following:—

Kilian (Etlinger. Obss. Obstet., Bonn, 1844) met with one so soft and elastic that he punctured it, believing it to be a cyst.

Ramsey (Med. Times, August 8, 1857) did the same.

Virchow also describes the processes which result in expulsion or apparent reabsorption. Spontaneous expulsion is caused (p. 183) by a sort of dissecting suppuration round the myoma, which may then be shelled out by uterine contractions, or may gradually work its way through the uterine walls by the help of uterine contractions, but without any suppuration, causing the absorption of the tissues in front of it by pressure.

It may also undergo retrograde changes, leading to partial or complete (?) disappearance. This takes place generally, if not always, by means of fatty degeneration, as in the uterus after delivery. Virchow thinks it unlikely that *complete* disappearance by this means ever takes place, and it has never been proved by dissection. The muscular fibre cells do degenerate, but the connective tissue is unlikely to be reabsorbed. As the retrograde changes take place in the muscular cells, it is in the soft rather than the hard myomata that striking diminution of size is observed. As also it is in the muscular cells

that the power of growth resides, it is again the soft myomata which grow rapidly.

Retzius (Hygiea, Bd. 23, p. 190) claims to have seen many cases of uterine myoma in which the size of the tumour was unaffected by delivery; these would be presumably cases of the *hard* variety.

Schmiedt (*loc. cit.*) asserts that variations in size in connection with pregnancy and parturition are more marked in sessile than in pedunculated fibroids.

Horwitz (*loc. cit.*) states the opposite with regard to diminution, and says that this depends on the pressure to which the pedunculated forms are subjected; and, in connection with this, that there is far more danger from the pedunculated than from the interstitial varieties.

Blot (Gaz. des Hôp., 38–51, 1869) has observed rapid increase and disappearance apart from pregnancy.

The involution of the uterus which takes place at the change of life, and sometimes before, generally affects the myomata also.

It is often said that the certainty of fibroid excludes the possibility of malignant growth; but Virchow (p. 121) says that fibromyomata can undergo such changes, especially into carcinoma or cancroid: and he has several times seen them changed into sarcoma, spindle or round celled, with fibroid or mucoid intercellular substance: the commonest seat of such a change is the stomach. A most interesting case of this kind is recorded in Virchow's Archiv., vol. lxviii. p. 227, by Brodowski, in which a myosarcoma in the stomach produced myosarcomatous deposits in the liver. An abstract will be found in the "Lancet" for August 5, 1876, p. 195. The metastasis of muscular fibre is almost unique, but has been observed in a case of myosarcoma of the kidney, which produced similar deposits in the diaphragm (Eberth., Virchow's Archiv., vol. lv.).

The period at which fibromyomata have been expelled is of some interest.

One was expelled in thirty-eight days (Rankin's Abst. *cit.*), another in forty-four days (Michauck, *loc. cit.*), another in about thirty-five days (a few days after four weeks, Schneider, *loc. cit.*), mine in thirty-five days.

Thus five to six weeks seems the average.

The period of rapid diminution of size:—

In one case the tumour could no longer be felt in twenty-two days (Retzius, *loc. cit.*).

In another case, where the tumour was the size of a child's head, after four weeks there was only slight want of mobility

and unevenness of the uterus (Gussmann, Würtemb. Correspondenz-Blatt, 38, 19, 1868).

In another, a fibroid, as large as a child's head at eight months, was found by dissection twenty-two days after birth to have shrunk to the size of a nut (Edmond Alling, Gaz. des Hôp., 92, 1869). The woman had observed a similar increase and diminution in all her former confinements.

In another, already quoted, a tumour of eleven years' growth, the size of a child's head, disappeared six weeks after birth (Scanzoni, *loc. cit.*).

Virchow has collected the literature of the comparative pathology of the subject.

Instances in animals are recorded by—

Gurlt, Lehrbuch der path. anat. der Haus-Säugethiere; Nachträge zum Lehrb. der path. anat. der Haus-Säugethiere.

Röll, Lehrb. der Path. u. Ther. der nutzbaren Hausthiere, Wien. 1856.

Foerster, Handb. der Path. Anat., 1863, Bd. 3.

Leisering, Bericht über das Veterinärwesen in Königr. Sachsen für das Jahr. 1859 und 1860, Dresden.

Fibroids (sessile) of the uterus, having much the same characters as in man, have been observed in the mare, sow, and bitch by Gurlt, and in the cow by Leisering. Large polypi have been seen in the uterus of the cow and mare by Gurlt, Röll, and Foerster.

Submucous fibroids have been seen in the vagina of the bitch by Leisering.

The case which I have described seemed to me specially instructive and picturesque from its completeness, rapid growth during pregnancy, influence on labour, involution during the puerperal state, with accompanying symptoms of absorption of septic matters, spontaneous enucleation (in spite of its being placed nearer the peritoneal than the uterine aspect, as shown by its subperitoneal and not intra-uterine bulging), with relief of the symptoms, and the recommencement of growth in other nodules exactly two months after delivery, when the uterine involution was completed.

The subject of uterine fibroids as affecting labour, &c., will be found treated with various completeness in all the text-books, which I have not quoted, as being well known. Also by Oldham (Guy's Hosp. Rep., April 1844), by Routh in his Lettsomian Lectures, and lately by Playfair before the Obstetrical Society, "Lancet," April 21, 1877, p. 575.

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ON

FIBRINOUS EXUDATION INTO THE BRONCHIAL TUBES.

BY

F. DE HAVILLAND HALL, M.D.

The disease which has been variously designated plastic, croupous, or fibrinous bronchitis is so rare, that, as Riegel says, "even in large hospitals years and decades may pass before a single case of the kind comes under observation."[1] I have carefully searched through the tables of statistics of St. Bartholomew's Hospital for the last eleven years without being able to find the record of a single case, consequently, as I was fortunate enough to have the opportunity of carefully observing a patient suffering from this peculiar malady during the year I held office as Medical Registrar at the Westminster Hospital, I was induced to look up the cases that have been described in England since Dr. Peacock, in the fifth volume of the Transactions of the Pathological Society, drew up a careful list of "Published Cases of the so-called Bronchial Polypus, or Plastic Exudation from the Bronchial Mucous Membrane," and this paper is the outcome of my researches into the literature of the subject, based on the knowledge I gained from carefully watching the case, which Dr. Basham has been good enough to allow me to publish. A plastic exudation into the bronchial tubes may take place under three conditions—

1. As a primary and independent disease, constituting what is known at the present day by the title of plastic or croupous bronchitis. The casts which are expectorated during the course of this disease have been described and figured by the older writers, such as Baillie[2] and Cheselden,[3] under the title of "Bronchial Polypi."

[1] Ziemssen's Cyclopædia of Medicine, vol. iv. p. 433.
[2] Baillie's Works, vol. ii. pp. 87 and 109.
[3] Cheselden's Anatomy of the Human Body, 4th edit. 1730, Table xxi.

2. Where it is associated with croupous pneumonia, in which case it seems to be due to continuity of the same process as that on which the pneumonic consolidation depends.

3. Where the disease is met with in association with true croup and diphtheria.

As I have already stated, I have had one case of the first class under observation, and by the kind permission of Dr. Fincham I am enabled to give some brief notes of a case belonging to the second group, which also came under my notice at the Westminster Hospital. Cases in which the exudation into the bronchial tubes is the result of croup and diphtheria are tolerably frequently met with, and though I am disposed to think that the process which in the lungs constitutes plastic bronchitis is analogous to that occurring in true croup, I cannot yet give in my adhesion to those who maintain that croup and diphtheria are merely modifications of one and the same disease. In the case of plastic bronchitis which I had under observation, albuminuria occurred, and this symptom would appear to point to some general blood disturbance, such as occurs in diphtheria, rather than to the more local disease croup, with which, however, plastic bronchitis has greater affinities than it has with diphtheria.

Though hospital statistics seem to prove that plastic bronchitis is a very rare disease, I am disposed to believe that it is more common than is generally supposed, as it is very likely to be overlooked if the sputa are not carefully examined, and even then the same mistake might be made, unless the sputa were shaken up with water. In my case I was nearly passing by the characteristic sputa. What struck my attention, however, was the peculiar appearance of the sputa, very much resembling milk curdled by the action of the gastric juice; so much so, that I at first thought that the patient had vomited up some of his milk.

I. Plastic Bronchitis.

1. *Etiology.*

The words of Niemeyer still hold good, when, speaking of the etiology of this disease, he says, "We have not as yet any accurate knowledge as to either its predisposing or exciting cause."[1] Even as regards sex, there seems to be some difference of opinion. Dr. Peacock, in his paper on plastic bronchitis in the fifth volume of the Pathological Transactions, says, that out of the thirty-four fully reported cases he collected, the males affected were twenty-five as against nine females. Dr. Walshe, on the contrary, says that this

[1] Text-Book of Practical Medicine, vol. i. p. 85.

disease is more common in females, "a slight excess."[1] Out of thirteen cases that I have found recorded in the periodical literature of the day, including the one I have reported in full, nine were males and four females, which bears out Dr. Peacock's statement.

Though plastic bronchitis may occur at any age, all statistics go to prove that from twenty to fifty is the period of the greatest liability to it.

Plastic bronchitis is essentially a chronic disease, for though some few cases be acute, the patient having no return of the expectoration after having brought up portions of casts in quick succession, yet in the majority of instances the patients suffer from a series of attacks lasting weeks and months; nay, Niemeyer mentions the case of a girl "who for years has almost daily coughed up a complete cast of the left bronchial tree."[2] In my case, the patient had had a similar attack three years previously, and in all probability he will continue to have a recurrence of this troublesome complaint. I cannot find that the season of the year has any influence on the occurrence of the disease, nor do territorial conditions affect it in any way.

Plastic bronchitis cannot be said to be caused by any particular pulmonary disorder, as it may be met with in persons who are otherwise healthy; and even in those who die straight off from this complaint, the lungs may be found but little affected, with the exception of the membranous deposit. Still plastic bronchitis is more frequently met with in delicate persons, who have had some previous lung mischief, or possibly have suffered from hæmoptysis. When we come to analyse the cause of death in cases of the disease under consideration, we cannot fail to be struck by its apparent association with phthisis. Out of the ten cases of death recorded by Dr. Peacock,[3] three were directly due to phthisis, one to laryngeal phthisis and three to hæmoptysis; and when we consider the close relation existing between phthisis and hæmoptysis, I do not think we should be far from the mark in saying that had the three patients recovered from the hemorrhage, they would have died from phthisis, even if they were not already affected by it.

2. *Course and Result.*

As I have stated above, the disease is usually very chronic, and is not attended with much risk to life. Andral, however, records two cases in both of which the patient died from suffocation, owing to the blocking up of a large branch of one of the bronchi;

[1] Disease of the Lung, 3d edit., p. 222.
[2] Op. cit., p. 85.
[3] Path. Trans., vol. v.

and Dr. Hilton Fagge[1] has given the particulars of another interesting case in which a like result happened. The patient was a girl of seven, who, after suffering for ten days from a spasmodic cough, expectorated a cast of the bronchial tubes. For the next three days one of these casts was ejected at least daily. On the morning of the third day she had a most distressing fit of coughing, in which her friends thought she would die; but after getting rid of a larger cast than any before, she was much better afterwards. About twelve hours later she became much distressed, coughed violently, fought for her breath, became dark in the face, and died. The post-mortem examination showed that a large fibrinous cast, similar to the one which had been expectorated in the morning, occupied the lower part of the trachea. The ramification of this cast extended into the right bronchus and its branches, but its larger end lay across the bifurcation of the trachea, occluding the entrance of the left bronchus so completely that it appeared at first as if it would occupy the branches of that tube also. This case is interesting as affording a typical instance of a somewhat rare form of death in plastic bronchitis, and it also proves the rapidity with which this exudation can be poured out, as the second cast, which caused the fatal termination, must have been formed during the last twelve hours of life.

As a rule, however, the course of the disease is determined by the conditions with which it is associated. It is frequently a complication of phthisis, but does not appear to affect the duration of that or any other disease with which it is combined materially.

3. *Pathology.*

When we come to consider the anatomical changes which take place in plastic bronchitis, we find a considerable discrepancy as to their origin. Dr. Peacock[2] says: "The precise cause which leads to the effusion of fibrine from an inflamed mucous membrane is not clear. It cannot depend on the intensity of the inflammatory action, for there is no proof either in diptherite, croup, or plastic exudation from the bronchial mucous membrane that the inflammation is particularly intense. Neither does there seem any evidence that the extension of the inflammation to the submucous tissue is the cause of the fibrinous effusion. It would rather appear due to some peculiarity in the constitution of the individual." Still, as Mr. North[3] says, "It must, at all events, be admitted that these polypi are not the result of common inflammation of the bronchial membrane, either active or chronic;

[1] Path. Trans., vol. xvi. p. 48.

[2] Op. cit., p. 51.

[3] London Medical Gazette, 1837, p. 332.

for if they were, they must be of much more frequent occurrence in various diseases that are accompanied by bronchitis."

It has been suggested that these concretions are due to the inspissated condition of the bronchial mucus, the air during expiration having carried out the thinner and more watery portion, and left the solid part behind to form a mould of the tube. But Dr. Baillie [1] completely upset this theory by observing that the dried mucus of the trachea is very different from the appearance presented by these casts, even if it were possible for the mucus in the bronchi to become dry; and his opinion is quite in accord with the view at present entertained. In his description of a polypus of a trachea he stated, "The mucous membrane of the trachea seemed to be perfectly natural, and the layer of adventitious membrane exactly resembled the coagulable lymph which is formed in other parts of the body."

Dr. Walsh [2] looks upon plastic bronchitis as "probably, though not demonstrably, dependent on a peculiar diathesis." He very pertinently says, "The immediately exciting causes are those of simple bronchitis; why, then, is the excreted product different?" And though many attempts have been made to settle this point, no adequate explanation has yet been given; and this is less to be wondered at when we consider the rarity of the disease as compared with croup and diphtheria, about the pathology of which there is still so much in dispute.

Sir Thomas Watson [3] assigns plastic bronchitis to "a chronic and limited inflammation of certain of the bronchial tubes," which leads to the formation of tubular membranes, and that, when these membranes become detached, hæmorrhage results. He speaks, however, from the experience of only two cases. In both of these the expulsion of the so-called polypi was preceded by hæmoptysis, which subsided when the casts began to be expelled. Hæmorrhage is, however, by no means a necessary factor in this disease; on the contrary, in the majority of cases it is absent.

I am disposed to agree with the view entertained by Niemeyer, who, in speaking of this disease, describes it as a "croupous inflammation of the tracheal and bronchial mucous membrane;" [4] and accordingly I would regard it as analogous to laryngeal croup. Dr. Fuller adopts a different view. He says, "The disease bears no affinity to croup, but is essentially a form of bronchial inflammation, connected probably with some peculiar diathesis which leads to the outpouring of concrete albuminous and fibrinous matter." [5]

[1] Works by Wardrop, vol. ii. p. 88.
[2] Diseases of the Lungs, 3d edit., p. 222.
[3] Principles and Practice of Medicine, 5th edit., vol. ii. p. 62.
[4] Op. cit., p. 84.
[5] Diseases of the Lungs, p. 353.

In spite of the many points of resemblance between the two diseases, the fact of the frequency of croup and the rarity of plastic bronchitis would seem to point to some essential difference between them.

4. *Morbid Anatomy.*

The post-mortem appearances observable in plastic bronchitis necessarily vary considerably according to the disease with which it is complicated, plastic bronchitis itself being rarely a cause of death, except in those cases where a fatal result is brought about by the impaction of one of the membranous casts in the trachea or larynx. As phthisis is the most usual accompaniment of this disease, it stands to reason that evidences of its presence will be found after death. Emphysema, collapse, and cirrhosis of the lung are the other changes most frequently met with. In those cases in which casts have been found in the bronchial tubes on making the autopsy, the mucous membrane has sometimes been found healthy, at other times injected, and the casts have not been adherent. A full description of this and other features of the disease is given in Ziemssen's "System of Medicine."

5. *Symptoms.*

The first symptom, which draws the attention of the physician to the presence of this disease, is the expectoration of the characteristic branching casts of the bronchial tubes. In most of the recorded cases this has been preceded or accompanied by symptoms indicative of slight bronchitis. The cough is often stated to be paroxysmal and dry before the casts are expectorated; the terms hard, ringing, rough, teasing have been applied to it, and the patients often complain of soreness about the sternum in consequence. Constriction and oppression in the chest is also a frequent complaint. In most cases, during some period of the attack, shortness of breath is experienced; in many the dyspnœa is marked. In my case orthopnœa was present at the height of the attack; and, as we shall see, it is not very unusual for the patient to succumb, to apnœa in consequence of the obstruction to aeration of the blood, offered by a large cast in the bronchi. While the obstruction persists, the patient is often cyanotic, but this and the other unpleasant symptoms of plastic bronchitis disappear almost immediately after the expectoration of the casts. Hæmoptysis is sometimes met with, either occurring before, during, or after the characteristic sputa have been expelled; usually, however, the membranous casts are brought up either

alone, or mixed with the ordinary muco-purulent sputa of bronchitis, as occurred in my case. "Dr. Peacock supposed that the escape of blood, when it occurred in cases of this kind, was due either to the intensity of the inflammatory action in the part, by which not only the plasma, but also the blood globules were effused, or to the rupture of blood vessels in the separation of the cast from the mucous membrane of the moulding bronchial tubes, or to congestion of the lung tissue in the seat of the disease. This view was in opposition to that of Michaelis, adopted by Dr. Cheyne, that the masses were of two distinct descriptions, one a fibrinous inflammatory cast, the other a moulded blood coagulum."[1] In some instances epistaxis has been met with.[2] In cases of true plastic bronchitis, in which the casts are confined to the bronchi, the voice is not altered; if it is, the alteration is generally due to laryngitis. In those cases, however, in which the casts extend up the larynx, as in croup and diphtheria, there may of course be hoarseness.

The amount of febrile disturbance varies. In my patient the temperature reached as high as 103·4° F. on one occasion, which is somewhat higher than is usually the case.

6. *Expectoration.*

The membranous casts, when first expectorated, are usually in the form of pellets, and it is not till they are floated out in water that their peculiar tree-like form is detected; as I have before stated, they are liable to be overlooked, if they are only in small quantity, or mistaken for masses of coagulated milk. Austin Flint compares them to boiled maccaroni, and the comparison is further borne out by the fact that the larger branches are perforated. The casts expectorated in my case bear out fully the appearance described at length by Riegel;[3] Dr. Peacock[4] has also given a very good and at the same time concise account of these casts. The exudation appears to be poured out in bronchial tubes of the third or fourth diameter, and may extend to their ultimate ramifications, and when expelled entire, they demonstrate beautifully the manner in which the bronchial tubes divide. They are usually of a white colour, but may be blood-stained when first expelled, and they appear to be formed of superimposed laminæ. The centre of the larger cast is perforated, in the smaller ones it is occupied by a softer material. According to Riegel,[5] "Micro-

[1] Path. Trans., vol. vii. p. 54.
[2] See cases recorded by Dr. Peacock, Path. Trans., vol. ix. p. 53; and Medical Times and Gazette, December 30, 1854.
[3] Op. cit., p. 451.
[4] Medical Times and Gazette, December 30, 1854.
[5] Op. cit., p. 454.

scopic examination of the cast reveals, as a rule, a structureless, hyaline, basement substance, between which cellular elements are thickly pressed together, principally composed of pus corpuscles." Unfortunately, in my case I was compelled to defer the microscopic examination of the casts expectorated till it was too late to get any satisfactory result. The casts, which are deposited in the Museum of the Westminster Hospital, show very clearly slight bulgings at intervals, said to be caused by the exudation being more abundantly thrown out at some parts than at others. As I have already stated, these casts may be formed with great rapidity, twelve hours being sufficient for the formation of a complete cast, extending from the bifurcation of the trachea into the right bronchus and its branches. As regards the quantity expectorated, a case is recorded of a man, aged 36, suffering from recurrent attacks of sub-acute bronchitis, who would spit up from two or three to sixteen or eighteen of these bodies in twenty-four hours, and a basinful of them might have been collected in the course of a few weeks.[1]

7. *Physical Signs.*

All authorities are agreed in saying that there are no physical signs peculiar to plastic bronchitis, so that, in the absence of the characteristic casts, the diagnosis of this disease cannot be ventured upon. The physical signs vary according to the amount of obstruction to the entrance of air offered by the casts. If this be complete, there will be dulness on percussion, absence of vocal fremitus, of breathing sounds, and of vocal resonance quite as marked as in cases of pleural effusion ; and, as I pointed out in last year's Reports, the presence of casts in the bronchi is quite sufficient to mask the special physical signs of pneumonia; these peculiar signs are accounted for by the collapsed condition of the lungs. The dulness due to the presence of an exudation in the bronchi is to be distinguished from pleuritic effusion, by the absence of any displacement of viscera and of ægophony. In a case which Dr. Hyde Salter[2] has recorded, the only physical sign during life was one spot, very small in size, which gave constantly a fine crepitation like that of pneumonia. Inasmuch as the disease is usually accompanied by chronic bronchitis, the most frequent auscultatory phænomena are sibilant and sonorous bronchi. In cases where there has been much obstruction in the bronchial tubes, the breathing has been described as puerile over the rest of the lung. The situation of the disease may at times be deter-

[1] British Medical Journal, May 28, 1864.
[2] Path. Trans., vol. xi. p. 36.

mined by the normal respiratory sounds suddenly becoming audible at a place where, previous to the expectoration of the cast, there had been symptoms of bronchial obstruction.

8. *Diagnosis.*

As I have before stated, plastic bronchitis usually occurs as a complication of some other chest disease, phthisis or sub-acute bronchitis being the most frequent forms, and till the characteristic sputa are expelled there are no symptoms which would suggest the idea of this disease. The acute form of plastic bronchitis might be mistaken for lobar pneumonia, or for circumscribed empyema, "and when, as in cases mentioned by Dr. Gordon, in addition to dulness on percussion and absence of respiration, the heart also is dislocated, it would be difficult, if not impossible, to distinguish it from the latter disease." [1]

In the patient I had under observation, till the expectoration commenced the difficulty was to distinguish the attack from pleurisy with effusion. What militated against the latter, however, was that the heart was not displaced, and that the dulness had come on much more rapidly than is usually the case in pleuritic effusion; still I cannot help thinking that the dulness, which persisted at the left base, after the rest of the chest had become resonant, was due to effusion into the pleura, unless we can believe that one of the casts broke off short, leaving the peripheral extremity impacted in the bronchus, and thus keeping up a state of pulmonary collapse.

9. *Treatment.*

During an attack of plastic bronchitis, the primary object of treatment is to facilitate the expulsion of the membranous casts, and with this idea emetics have been advised. If I had occasion to order one, I would prescribe twenty grains of ipecacuanha with ten of carbonate of ammonia. Riegel suggests the subcutaneous injection of the muriate of apomorphia; he says it is "an emetic which acts very promptly, is always certain, and is free from unpleasant attendant effects." [2] In some cases it may be necessary to assist in the removal of the casts by traction. With the idea of softening and thereby hastening the detachment of the casts from the mucous membrane, various inhalations have been suggested, such as limewater, the carbonates of the alkalies, permanganate of potash, lactic acid, or steam by itself. Dr. John Ogle [3]

[1] Dr. Peacock, Medical Times and Gazette, December 30, 1854.
[2] Op. cit., p. 467.
[3] Path. Trans., vol. xi.

reports a case in which great "benefit was derived from the use of inhalation of the vapour of water containing liquid pitch in the proportion of three drachms to the pint."

Various forms of counter-irritation to the chest have been resorted to, the chief ones being turpentine stupes, sinapisms and flying blisters; dry cupping may also be tried; but past experience does not hold out the hope of obtaining much good from these measures. As regards internal remedies, mercury was extensively employed, and in two cases recorded by Drs. Cane and Corrigan,[1] the disease yielded in a short time to a brisk course (the gums being rendered tender) after other remedies had failed; and inasmuch as mercury is of use in true croup, I should feel inclined to give it a trial in an obstinate case of plastic bronchitis. At the present time most authorities advise the prolonged administration of the alkalies, with the idea thereby of rendering the blood less plastic and liable to the formation of these membranous deposits. Niemeyer states that "in one case iodide of potassium afforded very marked and brilliant service (ʒss. daily), so that this treatment should be imitated."[2] If the croup is very troublesome and paroxysmal, sedatives may be cautiously employed, or a few whiffs of chloroform tried. In the intervals between the attacks, all that can be done is to get the patient into as good a condition of general health as possible, and to avoid anything likely to cause bronchial catarrh. For the former purpose, quinine, the mineral acids, syrup of the iodide of iron, but especially cod-liver oil, are indicated; for the latter, residence in a dry climate and the avoidance of exposure to cold and damp.

II. Pneumonia with Fibrinous Exudation into the Bronchial Tubes.

As regards cases belonging to the second group, I may mention the trouble they occasionally give in distinguishing them from pleuritic effusion, but I need not enter into this question here, as I fully discussed it in these Reports last year,[3] and gave brief notes of a case which came under my observation at the Westminster Hospital. According to Juergersen,[4] Remak was the first to point out that casts of the smaller bronchi may be found in pneumonic sputa at the height of the disease. "Remak supposes that these casts are formed from the fibrine set free by the inflammation. Grisolle, Gubler and Küss, maintain that the fibrine of the blood plays the most important part in their formation. The last two of these writers have found ciliated epithelium still covered with

[1] Dublin Journal, vol. xvii. 1840, p. 116. [2] Op. cit., p. 86.
[3] Vol. xii. p. 67. [4] Ziemssen's Cyclopædia of Medicine, vol. v. p. 89.

cilia on the coagula. This is their chief reason for opposing the theory of Remak." [1]

In the Pathological Transactions, vol. vi., Dr. Bristowe gives four cases of pneumonia attended with fibrinous casts in the bronchial tubes. He regards these as formed by the moulding of the exudation poured out into the air cells, which, instead of escaping, as is usual, by expectoration, is retained in the bronchi. In two of these cases no physical examination of the chest was made; in one the ordinary physical signs of pneumonia do not appear to have been observed; but in the remaining case, on admission, the right side was dull with minute crepitation, next day there was bronchial breathing. Death took place three days after admission, but before that event all sounds had disappeared from the right side. On making the post-mortem examination, the upper and middle lobes, and upper two-thirds of the lower lobe on the right side were found to be solid. Most of the bronchial tubes connected with the diseased portions of the lung were filled with casts.

In none of these cases were the casts adherent, in all the mucous membrane was healthy.

Dr. Wilks [2] reports a case almost precisely similar. The right side was universally dull on percussion, and there was a total absence of all sound during respiration. Post-mortem the whole of the right lung, except at its very lowest part, was in a state of grey hepatisation, and the bronchial tubes filled with solid casts of lymph. Mucous membrane healthy; casts not adherent.

In my case, the lower and middle lobe of the right lung were found to be in a state of red hepatisation passing into grey, the main bronchus occupied by a fibrinous deposit, which extended throughout the bronchi down to those of the fourth or fifth magnitude, not adherent to the walls of the bronchi.

What the predisposing causes of this form of pneumonia are I am unable to say, but I agree with Dr. Wilks that it is generally met with in cases of an asthenic character. Epidemic influence has been suggested as a cause, and there certainly was a great run of cases of pneumonia, when the one I placed on record occurred. In three instances of which he had notes, where solid casts had existed, renal disease with other complications existed in two. In my case, there was kidney affection, but the true type of the pneumonia was clearly shown by the symptoms and the manner in which death occurred. One would have imagined that the formation of plastic casts in the bronchial tubes during the course of pneumonia would not have been very uncommon, but experience

[1] Op. cit. [2] Path. Trans., vol. vi. p. 68.

in the post-mortem room shows that it is very rarely met with, and it is not likely to be overlooked.

Diminished amount or absence of expectoration is a symptom of pretty constant occurrence in this form of pneumonia, clearly explicable on physical grounds, the exudation becoming consolidated and decolorised in the tubes, instead of being expectorated, as is usually the case, in the form of the so-called rusty sputa.

III. Croup and Diphtheria with Fibrinous Exudation into the Bronchial Tubes.

The third division of cases, in which casts may be formed in the bronchi, comprises croup and diphtheria. In these two diseases the casts found in the bronchi are directly continuous with the exudation in the larynx, but, as far as microscopic examination goes, they are not to be distinguished from the casts formed in a typical instance of plastic bronchitis.

These cases are interesting as showing the connection between true croup and croupous bronchitis, and it seems to me that all speculation as to the cause of the exudation in the bronchitic form must be suspended, till an explanation has been given of the etiology of the more common disease of croup, and up to the present time no satisfactory answer has been given to the inquiry, What particular modification of inflammation is the cause of the exudation in croup?

Any one who would take the trouble to examine the periodical literature of the day, could collect any number of cases of this form, while looking up the subject of plastic bronchitis. I came across several instances, of which the following may be taken as a fair example:—

A child of four was admitted, on the third day of croup, into the Westminster Hospital under Dr. Fincham's care, and tracheotomy was performed on the night of admission. The child went on well up to the third day, when the tube of the canula became blocked up by portions of loose fibrine, which nearly suffocated him. The canula was removed, and a complete fibrinous cast of the entire trachea and larger bronchial tubes extracted. The child eventually died from pneumonia on the twelfth day. The fibrinous exudation was cylindrical throughout, excepting at its smaller terminations where the bronchial tubes seemed to have been wholly blocked up.[1]

In diphtheria occasionally complete moulds of the air passages are expelled. Dr. Murchison[2] has placed on record a marked instance. In this case the patient died from suffocation, within twelve hours after expectorating a membranous cast of the larynx, trachea, and

[1] Path. Trans., vol. xvii. p. 29. [2] Ibid., vol. x. p. 320.

larger bronchi extending to the third subdivision. A post-mortem examination was not allowed, but in all probability the cause of death was due to the rapid formation of a cast, similar to the one expelled.

IV. Case of Plastic Bronchitis.

W. L., aged 19, a packer. Admitted into Westminster Hospital under the care of Dr. Basham, 28th September 1875.

The patient stated that he had always been delicate. He has not had syphilis. Three years ago he was laid up in bed for five weeks, and at this time he coughed up branched pieces of soft matter like the white of an egg, and for a year afterwards he was not well enough to work. The present attack began on 24th September, with pain in the left hypochondrium, which came on suddenly, and coughing, which increased the pain.

Soon after admission he was found propped up in bed unable to lie down.

Pulse, 132. Artery, rigid; visible pulsation in radial. Heart sounds, clear. Respiration, 36; jerky. A suspicion of pleural friction in left infra-axillary region. Not much cough. Temperature, 101·2°. Tongue moist, furred. Bowels constipated. Ordered Cat. lini. Ammon. carb., gr. iv.; liq. ammon. acet., ℥ss.; aquæ ad. ℥j.: quartis horis.

September 29.—Morning temperature, 101·8°. Evening temperature, 103·4°.

September 30.—Pulse, 116.° Morning temperature, 100·6°. Evening temperature, 102·6°. Breathing easier than it has been. Good resonance over the right lung. Some rhonchus over the back. Left base dull. No vocal fremitus. Feeble breathing. Rhonchus above dull area. Last night he began to expectorate, and by mid-day he had brought up about a quarter of a pint of expectoration; a small portion of it is muco-purulent, but by far the greater bulk consists of branching masses of fibrine, very firm, apparently from tubes of the third and fourth calibre down to the smallest bronchi; the sputa comes up without any difficulty. Urine, sp. gr., 1019; neutral; a trace of albumen.

October 1.—Morning temperature, 100·6°. Evening temperature, 102·8°. Pulse, 120. Respiration, 36. Right side good resonance and vesicular breathing; left side impaired resonance up to angle of scapula, and not good resonance above; feeble bronchial breathing over the dull region, but no abnormal sound; vocal resonance decreased; vocal fremitus absent. No more plastic sputa since yesterday. Poultices to be discontinued. Ammon. carb., gr. x.; aquæ, ℥j.; cum acidi citrici, gr. xii.; aquæ, ℥j.: quartis horis.

October 2.—Respiration, 28. Pulse, 104. Morning temperature, 100·8°. Evening temperature, 101·6°. Able to lie down lower in bed. Not much cough. No return of the plastic sputa; a little viscid expectoration. Right lung good resonance and vesicular breathing all over; left front dull up to second rib; no air entering; no vocal resonance or fremitus; above second rib a tympanitic note and feeble breathing; posteriorly dull all the way up, though the dulness is not quite absolute in supra-spinous fossa, where there is ægophony and a little creaking; bronchial breathing along spine; a little feeble respiratory sound over dull region; no vocal resonance or fremitus.

October 3.—Morning temperature, 99·4°. Evening temperature, 100·2. Urine, sp. gr., 1020; acid; no albumen.

October 4.—Morning temperature, 98·8°. Evening temperature, 102°. Pulse, 116; dicrotous. Respiration, 32.

On October 2, in the evening, he again commenced expectorating the plastic sputa, and this has continued up till now (11·30 A.M.), in quantity about a quarter of a pint, the same as on September 30. It comes up quite easily. Had a good night. No pain. Good resonance all over left front. Breathing coarse, a little rhonchus at the base. Left back good resonance and vesicular breathing down to spine of scapula; from spine to angle of scapula, impaired, and dull below; over the dull region there are mucous rales. In reference to the mention made of ægophony on the 2d, it may be stated that the vocal resonance has still a twang about it. Heart's apex, about an inch internal, and about three below nipple.

October 5.—Morning temperature, 100°. Evening temperature, 99·6° Pulse, 96; soft. Since this attack he has not been able to lie on the left side. Has never had any hæmoptysis. Left base still dull. No plastic expectoration to-day. Urine acid; no albumen.

October 6.—Morning temperature, 99·2°. Evening temperature, 100·4°. Pulse, 92; soft. Breathing easily. Hardly any cough. No more plastic sputa. No abnormal sounds to be heard on either side. Left base, resonance much impaired, with absence of breathing sounds and vocal fremitus. General condition much improved. Urine, sp. gr., 1016; acid; no albumen. Ammon. carb., gr. x.; potass bicarb., gr. xxx.; aquæ, ℥j.; e. succo limonis: quartis horis.

October 7.—Morning temperature, 98·2°. Evening temperature, 99·5°. Pulse, 96; fair volume. No cough.

October 8.—Evening temperature, 98°. Pulse, 100; fair volume. No alteration in the physical signs.

October 16.—Has been improving up to this date; free from

cough, and no return of the plastic expectoration. Pulse, 96; soft, dicrotous. Left base, resonance greatly impaired up to angle of scapula, with feeble breathing and no vocal fremitus. Anteriorly below left nipple, there is good resonance with crackling crepitation almost like a friction sound.

October 18.—Urine, alkaline; sp. gr., 1012; blood-tinged; a few wrinkled blood corpuscles visible microscopically. A copious precipitate when acidulated with dilute acetic acid and boiled; almost cleared up on the addition of nitric acid. Pulse, 112. While I was examining the patient's chest on the 16th, he said he felt as if there were a piece more of the plastic expectoration coming up, and about half an hour afterwards he coughed up some pieces, and continued to do so till the afternoon of yesterday, bringing up about twenty pellets of expectoration. All over the left front there is now good vesicular breathing, with an entire absence of the crackling sounds heard on the 16th. Left base as before. Liq. potassæ, ℥x; inf. cascarillæ ℥j.: ter die.

October 19.—Urine, faintly acid; sp. gr., 1015; blood-tinged; albuminous. Pulse, 108. Physical signs unaltered. No cough.

October 21.—Urine, sp. gr., 1014; faintly acid; blood-tinged; only a few corpuscles visible microscopically, and a few finely granular casts; the faintest trace of albumen.

October 23.—Had a return of the plastic expectoration this morning. Has brought up two long pieces, one piece three or four inches long. Pulse, 80. No abnormal sound to be heard over the lungs. Left base remains dull as before.

October 25.—Urine not albuminous. A little more expectoration yesterday; none to-day. No cough. To return to medicine ordered on October 6.

October 27.—Urine, sp. gr., 1013; faintly alkaline. No return of plastic expectoration.

October 30.—Pulse, 68; good volume. Up yesterday for an hour. Progressing favourably. Improved resonance left base, but hardly any air entering.

November 3.—Going on well.

November 11.—Quite convalescent. Resonance posteriorly greatly improved; in fact, there is now only impaired resonance at the base. Air entering down to the base, a little creaking.

As I proceeded with my paper, I have several times referred to particular points in this case, but I think it well to draw attention to the most striking features more in detail. This case illustrates the usual indefinite etiology of plastic bronchitis. The patient was delicate, but no family predisposition could be traced; then the liability of this disease to return is shown, inasmuch as this was the second attack. I have endeavoured, but without

success, to find the patient since he left the hospital, in order to see whether he has been again attacked.

His symptoms were such as are ordinarily met with in this disease, except that the sputa came up much more easily than is usually the case. As regards the physical signs, they were very characteristic, showing that the main bronchus on the left side, close to the bifurcation of the trachea, must have been obstructed, as there was dulness and loss of vocal fremitus and resonance, and only a little feeble respiratory sound all over the left side; and that this was the case is shown by examining the sputa, the largest of the casts being considerably greater in diameter than a quill, and by the fact that as soon as the expectoration had taken place, the normal physical signs returned, except that there remained dulness, with absence of breathing-sound and vocal fremitus at the left base. Whether this was due to a little pleuritic effusion, or to collapse of the lung caused by the plugging of a bronchus by a cast, I am unable to state. One curious feature in the case is that his urine was slightly albuminous the second day after his admission; it then became free from albumen till October 18, when it was noticed to be faintly blood-tinged, and on the same day he had a return of the membranous expectoration after an interval of thirteen days, and when the expectoration ceased the albumen disappeared; so it would seem as though there were some connection between the two. I have not come across a case of plastic bronchitis in which this coincidence, to say the least, has been noticed. The only other remark that I have to make is with reference to the treatment. Dr. Basham ordered alkalies with the idea of rendering the blood less disposed to throw out the plastic exudation, but I do not think that the treatment effected much, one way or the other.

CASES

BEARING ON

DISEASES OF THE LIVER.

BY

J. WICKHAM LEGG, M.D.

The following four cases have been met with in the post-mortem room of the Hospital during the past twelve months. Three of them owe their interest chiefly to the clinical phænomena; the last to the appearances found after death. My best thanks are due to Dr. Black, Dr. Andrew, and Dr. Church for their kindness in letting me publish this medical history of patients lately under their care.

Moveable or Displaced Liver.

Some eleven years ago, Cantani, an Italian physician practising in Pavia, according to E. A. Meissner, described an appearance hitherto unknown: a complete absence of the liver dulness in the right hypochondrium, together with the presence of a tumour in the right inguinal region, corresponding in size and shape to the liver. This combination of physical appearances was called by Cantani *Fegato ambulante*, as he believed it to be due to a displacement of the liver. The Germans named a like appearance *Wandernde Leber;* the French, in the only description which I can find, *Foie mobile.* Cantani's account seems to have drawn very little attention in England, for, as far as I know, no description of a similar case has yet been published in this country. I therefore feel some hesitation in using a name for this supposed disorder; but as the appearance must be spoken of, I trust the term moveable or displaced liver may be found free from objection.

The circumstances under which Cantani first described the *fegato ambulante* are as follows:—A married woman, fifty-four years old, came to him for a tumour in the belly. She had always enjoyed good health, excepting an attack of puerperal peritonitis. She had had three pregnancies, the last at the age of forty-three. Her mother died with some swelling of the belly, doubtful ovarian or carcinoma. One sister died of cancer of the breast, lasting for twenty years. The patient dated her disorder from the last delivery, soon after which she felt an unusual feeling in the belly, and in a few months this became a distinct sense of weight in the hypogastrium. She then felt a solid tumour below the umbilicus, which changed its position with every movement of the body. She felt as if still pregnant. Many physicians thought that it was a disease of the ovary, and diagnosticated a cysto-carcinoma. Others thought it an uterine fibroid. Of late years the patient had become distinctly hysterical.

On examining the chest, the lungs and heart were found quite sound; but no hepatic dulness could be made out in the right hypochondrium, axillary line, nor towards the vertebral column. The spleen was found in its usual place.

Examining the belly further, a tumour was found below the umbilicus and towards the right groin. It was rather large and smooth; of small resistance; dull to percussion. The upper border was convex, the lower could not be so clearly defined, as it passed under the pubic bone, and towards the groin. The tumour was freely moveable, and could be pushed up towards the right hypochondrium.[1]

As the patient continued in much the same state of health, no opportunity was afforded for an examination after death, and if the tumour were simply a displaced liver, life was not likely to be shortened by its presence. A vaginal examination does not seem to have been recorded, yet surely such must have been made in order to exclude ovarian or uterine tumours.

Within two years after Cantani, Romolo Piatelli published another case of supposed displaced liver. The patient, a woman fifty-six years old, was married at nineteen, and became pregnant twice within a short time. Her married life, however, scarcely lasted two years. There was found in the two lower thirds of the right side of the belly a tumour with a convex, smooth, and regular surface, of rounded irregular form, and somewhat firm consistence. The upper wall of the tumour was convex, rounded, and thick. The sides felt thinner. The tumour gave

[1] Arnaldo Cantani, Annali Universali di Medicina, 1866, vol. clxxxviii. p. 373.

no signs of fluctuation. It was freely moveable, and moved with inspiration and expiration; the right upper border could be pushed close under the ribs. The tumour was dull to percussion.

At first it was thought to be ovarian, but its free mobility and the presence of a tympanitic percussion note between the tumour and pubes seemed to forbid this diagnosis. The spleen lay in the left hypochondrium, and the tumour could not be a cystic kidney, for there was no fluctuation. Further, percussion of the right hypochondrium gave a tympanitic percussion note. Piatelli therefore concluded that there was a displacement of the liver from its natural position.[1]

The causes of this displacement are thought by Cantani to be the weight of the liver itself, which might drag upon the ligaments, tight lacing, a chronic inflammation of the peritoneum, and repeated pregnancies, which last might push back the liver. Piatelli notes that his patient had been accustomed to tight lacing, had been twice pregnant, and had ascites from a previous peritonitis. Thus many of the ætiological conditions pointed out by Cantani were present. Emil Apollo Meissner criticises Cantani's statements. He thinks that pregnancy can have but little to do with the begetting of a moveable liver, as the organ would be pressed upwards towards the diaphragm, and not brought down into the belly. Further, inflammatory processes in the ligaments are likely to end in contraction rather than in relaxation.[2]

Winkler thinks that the moveable liver may be caused by events having the following sequence. First, pregnancies, especially when they last to the full time; then a pendulous and relaxed belly; from this a descent of the liver and a *hepar pendens*, followed by a stretching of the hepatic ligaments and a falling of the liver towards the front; and lastly, a turning of the liver upon its axis, and consequent twisting of the structures entering the porta.[3]

Meissner was the first German to describe a moveable liver. His case differs little from those of Cantani and Piatelli, but it seems to have drawn a good deal of attention, for a great number of cases were soon after published in Germany, as well as in Italy; four come from Russia.[4] Last year a thesis for the degree of doctor of medicine was published at Paris, in which two new cases of moveable liver were recorded. P. Müller in 1874

[1] Piatelli, Schmidt's Jahrbb., 1869, Bd. cxli. p. 112.

[2] E. A. Meissner, ibid., p. 107.

[3] Winkler, Arch. f. Gynäkologie, 1872, Bd. iv. p. 150.

[4] It is perhaps worthy of note that a great number of cases have been published by teachers of midwifery, or in publications devoted to the diseases of women.

published a case in which an examination after death was made. I cannot, for my own part, see any great difference in the clinical phænomena between his case and the other cases which were believed to be displaced liver. I am, however, aware that it has been said that the diagnosis was not to be justified. The result was that the liver was found very small, but in its natural place, while the tumour felt during life was discovered to be the omentum, closely resembling the liver in shape, and even in colour.[1] I will now speak of a case which came under my own notice a few months ago, which, according to Cantani and others, should have been an instance of displaced liver. After death, however, it proved to be an enlarged kidney, and the liver, larger than natural, was in the right hypochondrium.

James Benton, aged 36, a French polisher, formerly a soldier, was admitted into Mark's Ward on October 5, 1876, under the care of Dr. Andrew. He said he had noticed a swelling in his belly for fourteen years, and believed it to be due to a fall from his horse two years before the swelling was noticed. He has several times passed bloody water, the attacks lasting several days. He said that the tumour was only small at first, but grew larger. He came to the Hospital on account of his cough.

In the belly there was found a prominent tumour in the umbilical region. The veins of the skin of the belly were slightly enlarged. The tumour was dull to percussion, singularly elastic to the feel, very like the sensation given by handling a hydatid cyst of the liver. It was irregularly quadrate, and very like the shape of the liver, lobed, measuring fifteen inches across, and seven and a half in the middle line from above downwards. The tumour was singularly well defined, and could be made out with greatest ease by palpation. It was not painful, and the man complained of no pain about it. It was freely moveable.

There was complete absence of dulness to percussion in the right hypochondrium, axillary line, and behind.

The chest was barrel-shaped, shoulders high, angle of Ludovicus prominent, costal angle enlarged. There was general hyper-resonance to percussion, except at the right apex, where it was impaired.

The man died somewhat suddenly of his phthisis during a dense fog on October 24.

I wish to express my thanks to my friend Dr. Wharry, the House Physician, who had charge of this case, for his kindness in letting me complete my own very imperfect clinical notes from his fuller observations.

[1] P. Müller, Deutsches Arch. f. klin. Med., 1874, Bd. xiv. p. 146.

Examination thirty-one hours after death.—Body somewhat wasted; rigor mortis persistent. An elastic-feeling tumour fills the umbilical region. On opening the belly, the liver is seen to be in its usual place. The colon is displaced, and descends on the left side of the tumour to the iliac fossa. The omentum is adherent over the tumour. The tumour fills the space between the liver and the cæcum, and extends far back into the loins. The edge of the lung comes down to the sixth rib on the right side. In the middle line the liver extends 55 mm. below the tip of the xiphoid cartilage.

Nothing worthy of note in the heart or pericardium.

At the apices of both lungs were largish cavities, and the lungs generally sown with solid yellow bodies of varying sizes. The tissue generally was emphysematous.

The liver itself quite natural in shape and place; weight, 2100 grm.; not amyloid.

Spleen, 130 mm. long; adherent to diaphragm on left side; pale; somewhat tough; not amyloid.

Stomach natural; intestines not amyloid, but about the middle of the ileum they begin to show tubercular ulceration. The two largest are the size of half-a-crown and are just above the ileo-cæcal valve.

Left kidney natural. The right is the tumour felt during life, and is the seat of a new growth of pulpy consistence.

Meninges of brain free from tubercle.

I must own that I feel very little confidence in the correctness of the diagnosis of the cases which have been hitherto recorded. There are about twenty of these published, and in only two has an examination after death been made, and in these two the liver was found in its natural place, and some other organ had been mistaken for the liver. A large number of the cases have been seen in women, only four in men—that is, the second of Blet's, about which nothing more is stated than that he died of a tuberculosis,[1] two of Wassiljew's,[2] and the one just detailed. Then as to age, in four this is not given. In the remaining sixteen, eleven had passed the fortieth year; two were between twenty and thirty; three between thirty and forty; three between forty and fifty; five between fifty and sixty; two sixty years old, and one seventy. Repeated pregnancies seem also common; but whether in excess of what is usual in women above the age of forty remains to be seen. Thus in only one is it expressly stated that the patient had never been pregnant; in six the pregnancies were seven or more in number; in one case

[1] Blet, Étude sur le Foie mobile, Thèse de Paris, 1876, p. 6.

[2] Wassiljew, St. Petersburger med. Wochenschrift, 1876, No. 30.

being sixteen; in a second twelve; and in five other cases only two or three.

In four the practice of tight-lacing is mentioned; in one it is denied that it was followed.

The tumour was freely moveable in all; in some it is said that it could with trouble be pushed into the right hypochondrium. In Chvostek's case it was very painful;[1] in most of the others, either tender at first or painless; in another it is stated that it caused much inconvenience from childhood.

It is described as lobed, smooth, elastic, with well-defined borders, and dull to percussion. Its usual place is the umbilical or hypogastric regions, in some cases tending to the right. In one case it was said to be associated with transposition of the spleen; in another a band could be felt from the right hypochondrium to the middle of the tumour, supposed to be the suspensory ligament.

In Leopold's case the tumour at first was only as big as a walnut, and then grew to the size of a child's head.[2]

In only one case, the last of Wassiljew's, has jaundice been noted.

It will be seen that very little information is gained by a comparison of these cases. The points that they seem to have in common, setting aside the tumour and absence of liver dulness, are that the diagnosis has been made chiefly in women, and in women who have borne children.

There are two points, and two only, upon which the diagnosis of moveable liver rests: absence of dulness to percussion in the right hypochondrium, and the presence in the belly of a moveable tumour having some of the physical characters of the liver.

In all the cases known, the liver could not be felt in its proper place on palpation. Nor could any dulness on percussion be found. The lung sound passed immediately into the tympanitic intestinal note, the lung sound either ending at its proper place or being continued lower down than it should. Now, does an absence of hepatic dulness on percussion always indicate a small liver, still more an absent one? By no means. The liver may be of natural size and weight, and yet the dulness be masked by emphysematous lungs or by tympanitic bowels. The case given above is an instance.

I have lately seen another case bearing on this point. A boy aged 10 was admitted into Luke's Ward, under the care of Dr. Southey, for simple jaundice. His liver could be felt two fingers'-breadth below the ribs. He suddenly became dull and

[1] Chvostek, Virchow und Hirsch's Jahresbericht f. 1875, Bd. ii. p. 238.
[2] Leopold, Arch. f. Gynäk., 1875, Bd. vii. p. 152.

hard to rouse, and his state approached that of coma; pupils dilated, and retention of urine; and at the same time the liver dulness almost completely disappeared. Some of the bystanders, myself among the number, thought we had to do with a case of acute yellow atrophy.[1] Yet on smart purging, abundant scybala were passed, the coma went off, and in less than a week the liver could again be felt precisely of the same size as before. The patient soon after left the hospital cured. It cannot be thought that the liver decreased in size during the decrease of percussion, and grew to its right size again after purgation, but rather that the liver dulness was masked by some cause not perfectly made out.

The insufficiency of mere decrease in the liver dulness to determine decrease in the liver itself has been long known; in cases, for example, of acute yellow atrophy, in which the liver dulness altogether disappeared, and yet after death the weight of the liver was quite natural. Sander has remarked a case of simple jaundice in which the liver dulness suddenly completely disappeared without corresponding tympanitis.[2]

Then as to the second point in the diagnosis, the presence of a moveable tumour in the belly, having some of the physical characters of the liver. That this may be highly deceptive, Müller's case proves. Blet seems to consider that the feeling of the notch between the right and left lobes is the keystone of the diagnosis.[3] Yet one would think that this may easily be simulated by a lobed tumour, such as an ovarian cyst. I must own that it seems to me very imprudent from any clinical physical characters whatever to assert that a given tumour is formed by the liver, in view of the only two examinations that have taken place after death. Further, displacement of the liver from relaxation of the ligaments is, as far as I can find out, an altogether unknown anatomical appearance. Sappey, a philosophical anatomist, says: "Il n'est pas sans interêt de remarquer que dans ses déplacements morbides . . . le foie ne se déplace jamais seul: il se déplace avec toute la masse des viscères abdominaux."[4]

In conclusion, I may state my belief that in none of the cases hitherto published has the evidence given been of such a nature as to command the belief that the tumour felt in the belly was the displaced liver. I am quite ready, however, to acknowledge

[1] It is only right to mention that Dr. Southey did not share in this opinion, and the correctness of his view was verified by the event.

[2] Sander, Deutsche Klinik, 1869, p. 295.

[3] Blet, *op. cit.*, p. 18.

[4] Sappey, Traité d'Anatomie descriptive, Paris, 1874, 2d ed., t. iv. p. 298.

my error should anatomical proof be given to the contrary in any of the cases.

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Cirrhosis of the Liver in Children.

I think it well to place on record two cases of cirrhosis seen at an early age ; for in neither case was much attention given to the liver during life. In the infant of seventeen months, the prominent symptom during the last few weeks of life was purging, until a few days before death when the phænomena of tubercular meningitis came on, a disease which was duly diagnosticated, and to which the fatal ending may be safely set down. No symptom to suggest a special attention to the liver was present. And in the boy, the clinical appearances were certainly such as would lead a most wary observer altogether away from the liver ; and yet no marked disease save that of the liver was found after a long and careful examination. Certainly neither tubercle nor ulceration of Peyer's patches were found ; and yet the symptoms early in the disease closely resembled typhoid fever, while later on a

[1] The references in this paper marked with an asterisk are those which I have been unable myself to verify.

suspicion of acute tuberculosis was entertained. In either case an almost superhuman sagacity would have been needed for the diagnosis to have been made aright.

Case I.

Cirrhosis of the liver in a boy of twelve, with symptoms closely resembling those of typhoid fever or tuberculosis.—William Alfred Hale, aged 12, admitted into John's Ward, under the care of Dr. Church, on December 28, 1876.

For the clinical notes I am indebted to Dr. Verco and Mr. Garstang.

His father is a hairdresser, and says that the boy has been ailing for about a fortnight with general pains; that he has been confined to his bed since December 25; since that time, that he has been light-headed at night and talking; that his bowels had been very loose for several days. No one has been ill in that neighbourhood to father's knowledge.

Present state, December 28.—Lips dry and slightly peeling. Tongue, thick white dorsal fur, clean at tip and edges. The bowels not open to-day. The chest is rather rounded and resonance good. There is no cardiac dulness. Liver dulness depressed a space.

Some moist variable crepitant sounds over right and front chest; behind sounds natural. Heart's apex beats in natural place, but with a feeble impulse; sounds are clear, the first is weak. The belly rather full. One very doubtful rose spot. Some gurgling in the ascending colon, but no tenderness in the right iliac fossa. The legs normal.

Ps. 96. R. 32. Temp. 102·2°. Milk diet: pint of milk, H. acid nit. hydrochlor. t. d. To take also 10 grains of chloral hydrate immediately, and to be repeated if necessary.

In the evening he was delirious, and in and out of bed; made water in bed. Ps. 110. R. 34. Temp. 102·8°.

December 29.—He had a bad night, in spite of 20 grains of chloral. Tongue like yesterday. Bowels open once; belly generally tender. There are no spots. The crepitant sounds on front of the right lung are gone. Ps. 130. R. 40. Temp. 104·2°.

At 2 P.M., during the visit, the presence of loud and crepitant rales over both lungs before and behind was made out.

At night he was restless and talkative in his sleep. Face flushed. Bowels open once; stool rather dark and powdery; a few shreds. Urine about three ounces. A linseed poultice to chest. Ps. 122. R. 34. Temp. 103·9°.

December 30.—A better night; some delirium; bowels open. Ps. 122. R. 30. Temp. 102·6°.

Evening.—Not delirious; bowels opened once; rales in chest as yesterday. Ps. 136. R. 44. Temp. 104·3°.

December 31.—Very bad night. Cheeks flushed. Pulse small and weak; no dicrotism. Tongue white fur. Bowels open once. Ps. 126. R. 40. Temp. 103·4°.

Evening.—Pulse somewhat improved in volume. Urine ℥v. Ps. 124. R. 48. Temp. 103·3°.

January 1, 1877.—Very restless at night, and is still delirious. Sordes on lips and teeth.

Respirations more abdominal than yesterday, still chiefly thoracic. Pulse as last night. Brandy ℥ij. Eggs ij. Five grains of chloral and thirty of bromide of potassium immediately. Ps. 128. R. 44. Temp. 102·9°.

Evening.—Bowels open twice, but no spots.

January 2.—A very bad night. Constant muttering; slight subsultus. This morning a little convergent strabismus. Bowels open; motion passed in bed described as greenish. Pulse no weaker; not dicrotous. Lies lower in bed. Ps. 122. R. 40. Temp. 104·2°.

Evening.—Asleep. Ps. 120. R. 36. Temp. 103·3°.

January 3.—Slept after the chloral and bromide of potassium; in other ways state is not sensibly altered. Bowels open; one motion partly solid. He is now awake, muttering and moaning; delirium; no spots. Ps. 128. R. 42. Temp. 103·5°. Brandy ℥iv. Eggs ij. To continue the chloral and bromide, and a mustard poultice to the neighbourhood of the heart.

Evening.—Hands cold; body hot. Respirations very irregular; pulse very much weaker. Eyes more sunken; belly not so prominent. Ps. 150. R. 40. Temp. 103·5°.

January 4.—If anything, is worse. Passed a very bad night. Had no draught. Bowels open once in bed; of a green colour. Took brandy ℥iv. and eggs ij. Put out tongue when asked; it shows a white fur, and dry. Ps. 144. R. 38. Temp. 104·5°.

2 P.M.—Essence of beef Oss. Ten grains of chloral hydrate at bedtime, and to be repeated every four hours. Ps. 128. R. 36.

Evening.—Ps. 128. R. 30. Temp. 103·6°.

January 5.—Passed a restless night. Had ten grains of chloral twice; one draught at midnight, and the second at 4·30 A.M. He was very restless and moaning, but became quieter after the second draught. He is now unconscious, but is taking nourishment a little better. Bowels open three times yesterday; not this morning. Ps. 134. R. 34. Temp. 103·4°.

Evening.—Quieter. Bowels open three times; very relaxed

during the day. Moans in his sleep. Is taking his food fairly well. Ps. 136. R. 30. Temp. 102·8°.

January 6, 10 A.M.—He is sleeping after a very bad night. Had only one dose of chloral, at 11·30 P.M.; but continued restless and noisy throughout the night. Bowels open once during the night. He takes all the stimulant ordered, though with some apparent reluctance. Is sleeping quietly now, and appears better. Ps. 130. R. 30. Temp. 101·8°.

Evening.—He slept undisturbed most of the day, and has taken plenty of nourishment.

January 7.—He slept very well without a draught. Bowels open once. Takes his food, and was sleeping last night when he was visited. Ps. 148. R. 40. Temp. 101·7°.

Evening.—Has seemed fairly well during the day. Bowels not open, but he seems to have some pain in his belly.

January 8.—In beginning of last night was noisy and restless. At midnight he took ℥ss. of morphia draught. This quieted his moaning, but he did not seem actually to sleep. He was unconscious, and seemed so low that the draught was not repeated. Bowels not opened since yesterday morning. This morning he is semi-conscious, with gasping, jerking respiration and a raised temperature. Is not taking nourishment nearly so well as for last few days. Ps. 140. R. 28. Temp. 102·8°.

Evening.—Belly swollen. Lying on back; eyes not closed, but turned up; mouth wide open and dry. Ps. 146. R. 38. Temp. 101·2°.

January 9.—Has wasted much during the week. There is much dulness and fulness, with tenderness in the epigastrium, extending to the right hypochondrium; the liver is thought to be much enlarged.

He has not had a good night. He takes food better again. This morning he is more sensible, but seems weaker. The pulse is not so strong, and the cough is not so vigorous. Bowels open once in the night. Ps. 150. R. 38. Would not have thermometer kept in axilla. It rose to 99·6°.

At 2 P.M. thermometer in axilla rose to 101·5°.

Evening.—Pupils very dilated; tongue coated white; belly not quite so tender or prominent. Ps. 148. R. 28. Temp. 101·6°.

January 10.—Has not had a good night. Bowels open once. Does not take his food well. Otherwise much the same as yesterday. Pulse is again a little firmer. Ps. 132. R. 36. Temp. 101°.

January 11.—Half conscious; he slept very little in the night, but was quiet, moaning occasionally. Bowels open once slightly. He seems weaker this morning; lips dry and much

sordes. He does not take all his brandy, and has some trouble in swallowing. Still a good deal of fulness in epigastrium. Pulse smaller, 136. R. 30. Temp. 99·4°, but this observation is imperfect owing to restlessness.

Up to this time the disease had been thought to be typhoid fever. Another opinion now began to be entertained—that of tuberculosis. Some bystanders, from the large size of the liver, began to think of abscess of that organ.

January 11.—Had a good night; bowels open once; he swallows better. Ps. 136.

Evening.—Had an enema of brandy and beef-tea in the morning because of dysphagia; not repeated because he can take his food better by the mouth.

January 12.—Has a papular rash out over belly where the poultices were a week ago. The upper part of belly is not so full. He rolls his head about and moans. Brandy ℥v. Ps. 52. R. 40. Temp. 100·4°.

January 13.—A fair night, and is much quieter. Bowels open once. No change. Takes food a little better again. Pulse rapid and more feeble than it has ever yet been. Much sordes on lips. There is no paralysis or squint. Ps. 144. Temp. 100°.

Evening.—Very restless.

January 14.—Very bad night. Bowels opened once. He resists brandy, but takes wine. He seems to understand what is said, but cannot speak; for he opens mouth when asked to do so. The rash over the belly is much more evident, and seems to be in great part purpuric. There is a rash on the back of his thighs, brownish, slight, fading.

January 15.—He has been fairly quiet during night. It is doubtful if this be due to sleep or exhaustion. He is much wasted. There are more sordes than ever about the mouth. He takes a fair amount of wine, ℥x. of which were ordered yesterday in place of ℥v. of brandy, but he still takes a little brandy with his egg. Pulse not to be felt at wrist; in femoral artery, 150.

Died on January 15, 11 P.M., very quietly.

Examination thirty-seven hours after death.—Body much wasted; rigor mortis absent. Red purpuric spots, almost confluent, over the lower part of chest and belly, said to be due to poultices. A small bed-sore over sacrum.

The spinal chord natural.

The dura mater of the spinal chord has a yellowish look; so has the dura mater of the brain where the convolutions are most prominent; the calvaria is thin; sutures symmetrical. Longitudinal sinus natural. Convolutions of brain not flattened, but while the dura mater is being examined about half an ounce of

fluid escaped from the meninges. The dura mater quite smooth. The vessels and meninges of brain quite natural; no tubercle around circle of Willis, or in Sylvian fissure. The ventricles of brain, central ganglia, cerebellum, pons and medulla natural. Weight of encephalon 1200 grm., or 43 oz. Sinuses and bones of base of skull natural.

The tongue covered with a thick coating of white material, not adherent. This material fills also greater part of the mouth, and lines the forepart of the velum, and turns round and covers the hind part for a few millimeters. At this place the material is thick, and rather closely adherent to the velum and bifid uvula; but the mucous membrane underneath is quite sound. The pharynx and œsophagus show precisely the same kind of stuff, which is apparent even to the cardia of the stomach; but it is here not adherent, and is in no way like false membrane. Under the microscope it shows a few confervoid growths and granular matter; but nothing else worth noting. It is probably nothing more than dried milk.

The left pleura holds about an ounce of clear yellow fluid; no signs of roughening or loss of transparency. The right pleura holds about four ounces of a turbid fluid, in which flakes of lymph float. The pleura itself, covering the lower lobe of the lung, is covered with a thickish layer of lymph.

The pericardium quite natural; it holds about a couple of drachms of a clear fluid. The cavities of the heart all hold fluid blood mixed with a few clots. The heart itself natural. No changes in the valves or muscular walls. Weight 4 oz., or 110 grm.

The epiglottis, larynx, and trachea natural. Bronchial glands natural. The lungs generally show emphysematous vesicles, especially along the margins. The upper two-thirds of the lower lobes on both sides are sunk below the level of the rest of the lung, and are of a bluish colour. They are solid, resistant, and do not crepitate. On section they are firm, purple in colour, not friable, and exude a small quantity of aerated fluid on pressure.

Spleen weighs 84 grm., or 3 oz.; it is purple, of natural consistence; the Malpighian bodies not very prominent.

No fluid in peritoneum. The contents of the small intestine at upper end are of a yellow-green colour. Peyer's patches quite natural, even to the ileo-cæcal valve. About a foot from this valve there are several patches, not limited to Peyer's patches, of great injection, which in spots have run on into hemorrhage. The large intestine contains solid fæcal matter.

The common duct is filled with yellow bile; the portal vein contains an ordinary clot; the gall bladder is distended with

yellow bile. The liver extends far away below the xiphoid cartilage; after being cut into, and the gall bladder opened, it weighs 1090 grm., or 39 oz. The surface is smooth, but in some places shows a very slight roughening on capsule, like grains of sand. It is of natural colour, but marked with white stellate lines, which are likewise seen very distinctly on cutting into liver. The tissue is tough and hard. The edges rounded.

The stomach shows none of the stuff which the gullet held. The mucous membrane looks opaque and macerated; it is highly pigmented towards the pylorus. The duodenum holds green-yellow contents. The tail of the pancreas natural; the head marked off by a sharply defined line from tail; head of a dark purple, almost hemorrhagic look, of lesser consistence than tail.

Inferior vena cava and aorta natural. The supra-renal capsules natural. The kidneys together weigh 168 grm., or 6 oz. Their capsule comes off with some trouble; the surface is yellowish white; the cortex is of natural breadth, but of a yellowish white colour, and the striation is almost lost; still the Malpighian corpuscles may be well discerned; the medulla very pale and white.

Ureters and bladder natural.

An examination with the microscope was made the same day.

Scrapings from the tumour at the head of the pancreas showed red corpuscles in abundance; large granular rounded cells about the size of liver cells, but with no distinct nucleus; in some a nucleus is apparent.

Heart fibres showed perfectly natural striation.

Liver cells abundant, of natural shape, with highly granular contents, and a well-shaped nucleus.

Kidneys: cells in tubules markedly granular, but the nucleus in all well seen, and cell well preserved.

Parts of the right and left lobes of the liver, having been hardened in chromic acid, were examined about February 6. Thin sections stained with carmine were mounted in glycerine. Whether examined with high or low power, sections from all parts of the liver showed the same appearances characteristic of cirrhosis. Bands of connective tissue surrounded single acini or groups of acini; the connective tissue was studded with lymphatic bodies, and showed a large number of round openings. The connective tissue did not seem to enter the lobules, which had a remarkably natural appearance. The rays of cells around the hepatic vein showed a perfectly natural radiation, and the cells themselves a polygonal outline, with a large nucleus and granular contents.

Case II.

Cirrhosis of the liver in an infant aged seventeen months, dying of tubercular meningitis—No history of syphilis discovered.—Joseph Taylor, aged 17 months, was brought to the out-patient room on August 30, 1876. He had then had diarrhœa for three weeks, and vomiting for a day. On September 21 he had an attack of convulsions, and was admitted on September 23 into Hope Ward, under the care of Dr. Black.

He was noticed to have a hydrocephalic head, thumbs clenched, toes bent. The temperature in rectum was 39·9° C. Double inguinal hernia. Bowels relaxed; motions green and slimy.

September 24.—Liver very large, filling the right hypochondrium and the epigastrium. He takes food fairly well. The alæ nasi dilate. He died on September 25 at 5 P.M.

For the foregoing notes I am indebted to Dr. Abercrombie.

I saw the mother of the child after its death, and she gave me the following account:—

The father was alive, aged 45, and had good health.

The mother, aged 32, always has had good health, save "pains in head like pressure" since childhood. She has never suffered from sore throat for any long time, or pains in the shins at night, or breakings out on the skin.

She does not know the year or the date of her marriage. She has never had any miscarriages, but has had four children, all born alive at full time; never any dead children.

First child, a boy, alive, eight years old. The last few months he has not been in very good health, and has had a cough and wasted. Second child, a girl, died when two months old of the hooping-cough, which she caught from her elder brother. She had no rash or breaking out on the skin, and no snuffles. Third child, a boy, died when one year and nine months old of diarrhœa and teething. He had the diarrhœa for nearly three months before he died; no snuffles nor skin eruption. The fourth child is the case now spoken of. He never had the snuffles, nor sores on his privates, nor rashes on his skin; but when a month old he had a bad eye, for which he was taken to the Eye Hospital near St. Martin's-in-the-Fields, and in another month the child was well. After this he had good health, and the mother hoped she should rear him, when a month before death he got the diarrhœa and fits, and died.

The mother was a watercress seller, and belonged to the very lowest of the London population.

Examination eighteen hours after death.—Body much wasted. Head very large. Skeleton rickety.

The skull is asymmetrical. The right parietal bone is much more prominent than the left. The sutures preserve their symmetry. There is no displacement of the coronal or sagittal sutures. The calvaria is thin; the fontanelles still open. The meninges very watery, with opaque white patches in places. The membranes at base of brain and in Sylvian fissure greatly thickened, showing many tubercles, large and small. The ventricles much distended with clear water. Central parts of brain are soft; pons and medulla natural.

Pleuræ natural; upper lobes of both lungs highly emphysematous; the back parts of lower lobes collapsed; no tubercles can be found in the lungs.

Pericardium natural. Heart small but natural.

Spleen natural; Malpighian bodies prominent; no tubercles. No increase in size of spleen.

Mesenteric glands coming from lower half of small intestine all enlarged; some cheesy, some diffluent. Of those from upper part, some are natural, others cheesy. The stomach natural; the small intestine shows only one ulcer, as large as a fourpenny piece, about midway between duodenum and cæcum. Peyer's patches, large, red. Large intestine natural.

Liver: glands in porta of liver natural. Gall bladder and ducts natural, patent. Portal vein natural. The umbilical vein obliterated. The liver free from adhesions, of natural size, that is, large for belly; smooth on surface, no tubercles apparent, of a pale yellow colour. On section, cut surface granular, mapped out by narrow lines into spaces the size of millet or hemp seeds; the tissue very tough, so that the finger could not be pressed into it.

Supra-renal capsules and kidneys quite natural.

Under the microscope the liver showed the usual characters of a moderate cirrhosis, with a large amount of fat in the cells of the circumference of the lobule. With a low power there were seen groups of liver acini separated from one another by narrow bands of fibrous tissue. With a higher power, the fibrous tissue was seen in many places to be coarsely fibrous, and to show long sweeping fibres around the lobules. In others the fibrillation was much less marked. Throughout the fibrous tissue there was abundance of lymphatic corpuscles. The fibrous tissue was sharply marked off from the circumference of the lobules; the cells nearest to it being, as a rule, filled with fat, but otherwise natural.

From the morbid anatomy point of view, these cases have but little interest. The naked-eye characters and the appearances seen under the microscope differ but little from those seen in the

cirrhosis of grown-up people. It is to the ætiology and symptoms that attention should be chiefly paid.

First as to the sex of the patient. Of the seventeen cases of primary cirrhosis which I have collected, seven were boys and ten were girls.

The two cases which I have just related were both boys, so, if these be counted, the proportion will be as nine to ten. There would thus seem to be little difference in the disposition of the sexes.

The ages of fourteen are between seven and thirteen; the other three are five and six. In my own the ages are seventeen months and twelve years. It will thus be seen that when cirrhosis is seen in children, it seems to choose, not the age of infancy, that is, from birth to seven years of age, but that of childhood, from seven to fourteen years. It is very rare before the first dentition be finished, excluding the cases which can undoubtedly be set down to congenital syphilis. In these the child is either still-born or lives but a few days, as in Schüppel's[1] and Dr. Wilks' cases,[2] although in F. Weber's case of cirrhosis in a still-born infant, nothing is said about syphilis.[3] Setting the early syphilitic cases aside, it would seem that cirrhosis becomes more common as age advances, and that from boyhood upward the disposition increases. I have not been able to meet with so many cases of cirrhosis in adolescence as might be looked for from the number of cases in childhood. I believe this is due to the disposition which exists to record the cases in childhood as the rarer, while the cases in adolescence are thought to be less uncommon.

Few things can be harder than to arrive at the truth with respect to the habits of these patients. It is nearly always impossible to rebut the insinuation that the little patient has taken, or been given, more than was good for it in the way of spirits or wine. Sir William Jenner has told me that, in one of his cases, the child asked for gin to be given to it soon after its admission into the Hospital; and in Dr. Murchison's case the father was a publican, and the boy was in the habit of drinking wine and water, especially between meals.[4] Still, the view that cirrhosis is due solely to an intemperate use of liquors seems hard to sustain, when it is considered that not merely domestic animals, but wild animals, suffer from cirrhosis. I am at a loss

[1] Schüppel, Arch. d. Heilkunde, 1870, Jahrg. xi. p. 74.
[2] Wilks, Trans. of the Path. Soc. of London, 1866, vol. xvii. p. 167.
[3] F. Weber, Beiträge zur path. Anat. der Neugebornen, III. Lief., Kiel, 1854, p. 47.
[4] Murchison, Trans. of the Path. Soc. of London, 1876, vol. xxvii. p. 199.

to know how these last could have alcohol introduced into their food; though it is well known that horses and other cattle are given mashes made with malt liquors, especially when sick; so that domestic animals cannot be considered free from all chance of alcoholic poisoning. But the amount introduced into their stomachs in this way must be very small, and, compared with the amount taken by human animals without risk of cirrhosis, of no account. It seems to me a highly probable conclusion that cirrhosis may arise independently of excess in alcohol.

The next important point in the ætiology of cirrhosis in children is the question of syphilis. Writers on infantile syphilis seem to be agreed, more especially the French and German pathologists, that cirrhosis and perihepatitis are one of the commonest results of congenital syphilis. In the case of the infant, no particulars of a syphilitic family history could be got. In the boy's case, the age seems to me rather too advanced for congenital syphilis; and after a most diligent search through the body, no other lesion suggestive of syphilis save the cirrhosis was found.

The ætiology of both of these cases seems therefore somewhat obscure.

The point to which I wish to draw special attention is the course which the symptoms ran in the boy. In most of the cases recorded the symptoms have been such as are commonly seen in grown-up people with ascites and slight jaundice. But in this case the symptoms were such as would point to a grave general disorder—high temperature, delirium, and the like. I am not acquainted with the records of any case in which like symptoms showed themselves. The high temperature is well worthy of note, as it remained high, from 103° to 104° F. and more, until within a week of death, and even then it was rarely below 100°. Delirium also has been seldom noted. There was a complete absence of jaundice.

Taking the symptoms and morbid appearances into account, it becomes necessary to consider how far cirrhosis in children, or even in grown-up people, is a general disorder, and not merely a local disease, limited to the liver. In the case of the boy, there were pleurisy, and a granular appearance of the epithelium of the kidney, as well as the cirrhosis. The fibres of the heart, however, showed a natural striation.[1] In Dr. Cayley's case, Mr. Needham found in the heart, stomach, spleen, kidneys, and

[1] I am sorry not to be able to give a more detailed account of the examination with the microscope. Nearly all my preparations were destroyed during my absence from England in the spring of the year.

brain changes closely akin to those in the liver;[1] and Dr. Burdon Sanderson, commenting on the late Dr. Hillier's case of cirrhosis in a child, suggests, on finding pericarditis, pleurisy, and cirrhosis together, that cirrhosis may be the expression of some general disease of the lymphatic tissue throughout the body, inasmuch as the pleuræ and serous membranes generally, with the sheaths of the biliary canals, are parts of the same lymphatic system.[2]

In the case of the infant, tubercle of the brain and intestines complicated the cirrhosis, and the intimate relation of tubercle with the lymphatic system is well known.

Acute yellow atrophy, when first described, was thought to be a local disease, a disturbance of the functions of the liver, causing general symptoms in its course, just as uræmia follows Bright's disease of the kidney. The more part of pathologists now believe that acute yellow atrophy is not a local but a general disease. Robin[3] and his followers[4] assert that acute yellow atrophy is nothing but an early stage of cirrhosis, for they find that the connective tissue of the liver is increased throughout, even before the liver cells undergo destruction.[5] In some cases this overgrowth of the connective tissue becomes most marked. Acute yellow atrophy is thought by many to be the result of the action of a poison either begotten in the body or brought into the body from without. Now the effects of an acute poisoning by alcohol bear the closest resemblance to this disease, so close that I do not think they can be now distinguished.[6] The results, therefore, of a chronic and acute poisoning by alcohol seem to vary only in degree and not in kind, for in both an overgrowth of the connective tissue of the liver is seen. And if the effects of the acute poisoning be a general disorder, why not the same in the chronic? It is well known that disease of the kidney commonly accompanies cirrhosis, though attention has been but rarely paid to the other glands in the body, or to the muscular system. The glands of the stomach and the cardiac fibres may well be studied by forthcoming anatomists.

Since I wrote the above, Dr. Hayden has published in the July number of the "Dublin Journal of Medical Science" a case of cirrhosis in a boy of fourteen, complicated with "cirr-

[1] Cayley, Trans. of the Path. Soc. of London, 1876, vol. xxvii. p. 195.
[2] Burdon Sanderson, reported by Hillier, Trans. of the Clinical Society of London, 1868, vol. i. p. 107.
[3] Robin, Mémoires lus à la Société de Biologie, 1857, p. 9.
[4] Fritz, Gaz. des Hôp., 1863, p. 81.
[5] Winiwarter, Med. Jahrbb. herausgeg. v. d. kk. Gesellschaft d. AA. zu Wien, 1872, p. 259.
[6] Lendet, Gaz. méd. de Paris, 1860, p. 405.

hosis" of the kidneys, pleurisy, and pericarditis. He was not a drunkard. In many particulars it reminds the reader of the case commented on by Dr. Burdon Sanderson, and adds something more to the view that cirrhosis is a general disease.

The following are the references to the cases of cirrhosis in children which I have met with. The list does not pretend to be complete.

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A. W. Foot.—Dublin Journal of the Medical Sciences, 1873, vol. lvi. p. 337.

Gee.—St. Bartholomew's Hospital Reports, 1871, vol. vii. p. 144. This case was complicated by complete obliteration of the mouths of the hepatic veins, and presented more the characters of a nutmeg than of a cirrhotic liver.

Griffiths.—Trans. of the Pathological Society of London, 1876, vol. xxvii. p. 186.

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CASES JUST MENTIONED.

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PRIMARY (?) MELANOTIC CANCER OF THE LIVER.

I have here used the term cancer, as a convenient expression to signify a malignant new growth. According to its histological characters, this specimen would appear to have been a melano-sarcoma carcinomatodes. Primary melanotic tumours of the liver are very uncommon; at all events, this is the first case that I have seen during the seven years that I have watched the post-mortem examinations at the Hospital.

William James Slous, aged 42, was admitted into John's Ward on September 9, 1876, under the care of Dr. Church.

For the clinical notes I am indebted to Dr. Verco.

His illness began about four months ago with jaundice, which came on gradually. He left off work nearly three months ago, at which time he first noticed his belly to be big.

He says he has never drunk spirits habitually. He has no rigors, though occasionally he sweats profusely. He has never been out of England or had dysentery.

He has wasted greatly during his illness. There is no loss of appetite; no cardialgia; no vomiting; no hæmatemesis or melæna.

He is much wasted; his eyes are sunk.

The liver dulness begins at the fifth space in the right nipple line; there is dulness at both bases behind, but there are no unnatural sounds. The heart sounds are natural, save some reduplication of the first.

Belly tense, prominent; above the umbilicus a solid mass fills both hypochondria, both loins, and the umbilical region. It is immoveable, and almost quite smooth on the surface. There is a small nodule, the size of a pea, to the right of the middle line. The edge is well defined, hard, thick, and uneven. There is a little fluctuation, very superficial, in the right hypochondrium, apparently from the presence of peritoneal fluid. There is a palpable friction-rub generally. There is very little tenderness except at the edge close to the navel. There is no œdema of the feet, The skin is deeply jaundiced. It has itched ever since the jaundice came on, but is less troublesome now. Pulse, 68; regular, full, strong, deliberate; artery rather tough.

On October 5, it is noted that the nodules on the liver are more distinct.

He became thinner, the tumour not changing much in physical character. The disease ran a painless course, and the patient died on February 2, 1877.

Examination thirty hours after death.—Body greatly wasted and deeply jaundiced; rigor mortis persistent, but not well marked. Several small purple spots on legs, especially below knees and on arms. On opening the belly a quantity of bilious fluid escapes. Left lobe of liver extends 180 mm. below xiphoid cartilage in middle line, and 50 mm. below umbilicus. The right lobe is not so large; in line of gall bladder it extends 90 mm. below the ribs; its surface is here and there covered with small patches of clotted blood. The left pleura covered with a thin layer of recent exudation, with a few hemorrhagic spots beneath the exudation. Right pleura empty, but shows a few spots of recent exudation.

Pericardium natural. Heart natural; valves and left auricular endocardium yellow; weight, 250 grm. = 8½ oz.

Right lung: base slightly œdematous, and edges emphysematous. Left: the upper lobe shows two patches of solid resistant tissue, granular on section, pale yellow in colour, and sinking in water, but raised above the surface of the surrounding tissue. These patches are bordered by œdematous tissue. In the middle of the lower lobe is a solid patch having the same characters as the two above, but deep purple in colour.

Spleen not larger than natural; purple on section.

Small intestines only ten feet in length. Mucous membrane natural; colour of contents of upper part yellowish; contents very small in amount; no hemorrhages on mucous membrane. The large intestine contains a little thin light yellow fæces.

The bile ducts, dissected in the porta as far as the branching into right and left hepatic ducts, are nowhere found distended, but are stained dark yellow throughout. The gall bladder is remarkably small; contains clear fluid; no gall stones.

Stomach and pancreas natural.

Kidneys: jaundiced; capsule slips off with great ease; they are of natural size; structure of cortex distinct; branching lines of pyramids are green in colour.

Liver weighs 4800 grm. = 9½ lb. It preserves roughly its natural shape; its capsule is somewhat roughened, and covered with small spots, yellowish, white, and opaque, varying from a pin's point to peas in size; these larger apparently formed by confluence. On a larger scale the surface is marked by deep green prominences from peas to apples in size. These are small in the left, large in the right lobe; at the back part of the right lobe there are one or two which lie close to the organ, looking like lymphatic glands, as they are almost separated from the liver. On section all these tumours are seen to hold a dark green pulpy material, bounded by a thick, almost cartilaginous, white capsule, which in some cases sends in septa, incompletely dividing the cyst. The pulp can be readily washed away, leaving the capsule.

No opening into the cysts can be demonstrated, although the attempt is made in many. In the thickest part of the right lobe is one of these large bodies almost the size of an orange, and in this the contents looked to the naked eye like the layers of fibrin seen in aneurysmal sacs, only of a dark colour.

Under the microscope, the day of examination, the cells from the pulp of the cysts could not be distinguished from liver cells. They were polygonal in shape, with large, well-formed nucleus, and green granules in quantity by the side of the nucleus.

The lumbar glands showed a blackish green surface on section.

Parts of the right and left lobes, containing some of the smaller nodules, were hardened in chromic acid; thin sections were cut and coloured with carmine or logwood. They were mounted in glycerine, and looked at with Hartnack's microscope, Ocular 3, Objective 7.

The following appearances were made out:—The nodule of new growth was sharply marked off from the neighbouring liver tissue by a broad band of connective tissue. In the larger nodules this is well seen with the naked eye, and forms the investing white capsule spoken of above. On the outside of the capsule are several layers of firmly compressed liver cells, which become almost spindle shaped from pressure. Within the growth itself are the cells already spoken of as having the greatest resemblance to liver cells. If anything, they are a trifle smaller than natural liver cells, but in other respects the resemblance is nearly complete. They are deeply pigmented, the pigment being most abundant in the neighbourhood of the large nucleus. The stroma supporting the cells is very delicate in some places, forming alveoli, containing many cells; in others, surrounding but one cell.

In the liver tissue itself, the connective tissue is increased in places here and there. Throughout the liver there is much pigment; some within the cells; other masses round in section or long, apparently between the cells, and forming casts of the capillary ducts.

The diagnosis made by Dr. Church in this case during the patient's life was cancer of the liver, and after death the first impression certainly was that the tumours were melanotic cancer. But on more careful examination there seemed difficulties in the way of this opinion. First, the thick, almost cartilaginous, capsule which surrounded the tumours, and which seemed perfectly continuous; at least no openings could be demonstrated. Then the first rough examination of the scrapings of the tumours showed cells which could not be distinguished from liver cells, and the liver tissue itself appeared deeply jaundiced and coarsely granular. A doubt therefore arose if the tumours were really new growths, or whether they were not portions of the liver tissue separated off from the rest by a local development of connective tissue, the liver in general being somewhat cirrhotic. But a more careful examination did not bear out this view. The tumours were clearly not hepatic tissue, although the cells so closely resembled liver cells. They showed no trace of arrangement in rays or acini, but a structure closely akin to carcinoma or sarcoma, or to a form of tumour showing the characters of

both in some degree, such as Virchow would call a sarcoma carcinomatodes. It is indeed rare for a melanotic tumour in the liver to have cells so very like the liver cells; it seems more common for the cells to be spindle shaped;[1] but in a report by Dr. Burdon Sanderson and Dr. Henry Green on some specimens of Mr. Andrew Clarke's of disseminated melanosis, it is said that "the pigmented liver cells so closely resemble the cells proper to the new growth, that it is impossible to determine whether or not the latter are the offspring of the hepatic elements."[2] In the liver and lungs the growths were not distinctly defined, but in the spleen there was a "distinct fibrous capsule, and in some septa passed inwards from the capsule and subdivided the growth," precisely as in this case in the liver.

My friend, Dr. Samuel West, who has examined sections of this liver, has suggested to me that the new growth is one primarily affecting the epithelium of the gall ducts. This view appears to be a very probable one, as it would explain the presence of the capsule of the new growths, and also suggest a cause for their pigmentation, as they would at once receive a supply of green and dark pigment from the bile in the ducts. But it has already been noticed that these fibrous capsules have been seen in the spleen, an organ without ducts; and I cannot consider it proved that in this case the liver was the primary organ seized with melanosis. Though the skin certainly showed no tumour of any size, and in the wasted state of the patient it was unlikely that any swelling of any size would have escaped notice, yet no special dissection of the limbs was made. Neither was any examination of the orbit made, a matter for very considerable regret, for I must acknowledge that, at the end of the examination of the viscera, my mind was led away from thoughts of melanotic cancer to the suggestions offered by the first rough examination with the microscope. For it will be owned on all sides, that if secondary melanosis of the liver be rare, primary melanosis is still rarer, so that Virchow says that it is almost unknown.[3] Notwithstanding, Virchow seems inclined to admit Frerichs' case[4] among the primary melanosis, notwithstanding the absence of any special examination of the skin or orbit. In this case there were certainly no large tumours of the skin and no symptoms of mischief within the orbit; so that if Frerichs' case be one of primary melanosis of the liver, this ought likewise to be allowed the same title.

[1] Payne, Trans. of the Pathological Society of London, 1873, vol. xxiv. p. 134.
[2] Andrew Clarke, ibid., 1872, vol. xxiii. p. 254.
[3] Virchow, Die krankhaften Geschwülste, Berlin, 1865, Bd. ii. p. 285.
[4] Frerichs, Klinik d. Leberkrankheiten, Braunschweig, 1861, Bd. ii. p. 319.

NOTES

OF A

CASE OF PAROXYSMAL HÆMATURIA.

BY

CLEMENT GODSON, M.D.

In the exhaustive paper on this affection by Dr. Wickham Legg, published in vol. x. of these Reports, is the following statement:—"Of the cases now on record, there is only one in which the disease has attacked a woman. The earliest age at which it has been seen is two years. There are three cases on record of the disease attacking boys of eight or nine; but the most common time for a first attack is between the ages of twenty and forty years."

Having met with an instance in a little girl aged 2½ years, it seems important that the case should be recorded.

The child was brought to me by her mother on August 15, 1874. She presented every appearance of health, a bright intelligent girl of florid complexion and fair hair. A bottle was handed me containing a fluid resembling tawny port wine, which was stated to be the urine passed by her in the early morning. I obtained the following history. All that had been voided on the previous day was of a natural colour, while on the two days prior to this that passed had been of a dark mahogany colour. Early in May this condition had been first observed, when it lasted for two or three days, and the attacks had recurred at almost regular intervals of four weeks ever since. They always seemed to terminate with a discharge of gravel. They were generally ushered in with cold and shivering, the child exclaiming, "Baby not well." There was no vomiting or retching; the appetite was always impaired at these times. There was no knowledge of exposure to cold, other than that the child had a habit of kicking off the bed-clothes. There were no manifesta-

tions of rickets. There had been no fall or injury of any kind. While with me the little girl passed her water, which was of a pale straw colour. I ordered some powders, each containing one grain of gallic acid with a little white sugar, to be dissolved in a tea-spoonful of warm water, and to be given every four hours, so long as the urine was discoloured; at other times six grains of bicarbonate of potash, with three minims of tincture of henbane, some syrup, and dill water, three times during the day. I directed that the child should be clothed in flannel drawers, and every care be taken to avoid chills.

The urine was examined on the same day by my friend Mr. Knowsley Thornton and myself, and we agreed that it contained numerous blood cells, a few crystals of oxalate of lime, and an immense quantity of uricacid crystals; microscopic calculi. A few days subsequently some of the same urine was examined by Mr. Symons, Demonstrator of Practical Physiology at St. Bartholomew's Hospital, and he reported, after very careful testing :—

"Uric acid (small crystals) in abundance.

Acid phosphates in considerable excess.

Albumen, a trace.

No casts or blood cells to be detected."

The parents reported, that after returning home from her visit to me, the child "passed water very dark, and also afterwards, on the same day. In the evening she commenced taking the powders, and very soon her water became clear, and was accompanied with a quantity of fine particles of brown sand. This condition continued for two days, when the urine became apparently normal. The powders were then discontinued, and the alkaline mixture commenced. As soon as the sand had passed baby seemed well."

Later on I received the following account :—

"The child is very well; she has only had one attack since August, a slight one on the last day of October; only one emission of dark urine." From this time up to the present, just three years, no further symptoms have been manifested; the little girl has kept in good health, has grown and developed.

It may therefore be fairly inferred that no renal disease exists; and the attacks appear to have been due to the presence of the uric acid crystals. I am desirous to call attention to one point especially. Mr Thornton and I, who examined the dark urine when fresh, observed that it contained numerous blood cells. Mr. Symons, an able histologist, who looked at the urine with the microscope a day or two later, found none. This bears out the observation made by Dr. Legg, that though blood cells are to be seen in the urine immediately after it is voided, they are not to be recognised after it has been standing for some days.

INTOLERANCE OF LIGHT.

BY

BOWATER J. VERNON.

As of old, intolerance of light is still a prominent and characteristic feature of certain diseases of the eye. In many cases it has lost nothing of its distressing intensity, and it must be confessed that our treatment of it has not gained much in certainty or in precision. There is, in fact, some reason to fear that the advance which the study of ophthalmology has undoubtedly made within the last few years has been in those departments which are more exact and scientific, and which possess the charm of novelty, rather than in the pursuit of everyday clinical observation.

The writings of the very distinguished surgeons who treated specially of diseases of the eye in the last generation, notably those of Sir W. Lawrence, of Tyrrell, and of Mackenzie, contain most graphic sketches of many of the forms of disease which can be met with everyday in our out-patient rooms; and no writer of the present time can venture to improve upon the work of men who were indeed masters of their art, as it was known in their day. Their portraits of disease are as true now as they were then, and a restatement, with reclassification, is nearly all that recent experience will warrant by way of change. In works of this period, intolerance of light was spoken of as being in itself one of the symptoms of a scrofulous constitution; so much so, that, when very prominent, the disease was then and there termed scrofulous; and we read of strumous ophthalmia, scrofulous iritis, strumous keratitis, and the like; and in some instances, where there was no appreciable external lesion, Mr Tyrrell[1] wrote of a

[1] Diseases of the Eye, vol. i. p. 152.

"scrofulous morbid sensibility of the retina." In these, and such-like expressions, all that could be implied was, that special modifications of these diseases were met with in persons of scrofulous habits or constitution. How vague was the term, and in how many different senses it was used, may be seen in the observations of Sir W. Lawrence.[1]

"Two kinds of constitution, differing considerably in some respects, are observed in persons called scrofulous. In one, there is a pale and bloated countenance, a swelling of the upper lip and septum of the nose, and a tumid abdomen. The mucous membrane of the stomach and bowels is easily disordered by errors of diet, or by trifling causes which would have little or no effect on other persons. When these important organs are disturbed, the nutrition of the entire body is more or less impaired. There is a languid state of the circulation, so that the skin is pale and rough, and the extremities are cold. The muscular flesh is loose and flabby, and there is a kind of torpor in all the functions, bodily and mental.

"In the other set of subjects, the integuments are thin, and the ramifications of the cutaneous veins are distinctly seen. There is an almost unnatural colour in the cheeks. The circulation is rapid, the nervous system irritable, and both are easily excited. The various functions of body and mind are performed quickly. A premature development of intellect is often observed in such children, and they are affected powerfully by all external influences. We cannot suppose that the phenomena and treatment of disease will be the same in the two kinds of constitution just described, though the term scrofulous is used in both instances."

At the time when Sir W. Lawrence wrote, it was said that "nine-tenths of the ophthalmic inflammations in children at Vienna were strumous;"[2] and at Breslau the proportion was said[3] to be greater still; for "of one hundred cases of ophthalmia in children, ninety-five or more were scrofulous."

It is conceded that typical and extreme forms of scrofulous disease are not so frequently met with as was the case even a few years back. Whether it is so or not, it will probably be admitted that the greater number of children who attend our out-patient rooms belong to the second rather than to the first of the two categories which have been sketched above; and it is probable that their sickly appearance is due rather to the effect of insufficient and unwholesome food, and to the general unhealthy state of their surroundings, than to any special defect or peculiarity of constitution.

[1] Diseases of the Eye, 1833, p. 243. [2] Beer., Lehre, vol. i. p. 588.
[3] Benedict, Handbuch, vol. ii. p. 165.

It is, I think, certain that the term "strumous ophthalmia" is less familiar than in former days; the disease to which it was especially applied being now spoken of as phlyctenular keratitis, on account of the anatomical features which it presents; and by some writers as herpes of the cornea, in consequence of its supposed neurotic origin. The variety of iritis in children which was called scrofulous is now very rarely seen, or it may have been that the term was applied to cases which are now considered to be of syphilitic origin, and which are associated with peculiar changes within the cornea, and with remarkable defects of the nose and teeth, as described by Mr. Hutchinson. The cases of "scrofulous irritability of the retina," spoken of by Mr. Tyrrell, may now be traced to some inherent structural defect of the eyeball itself, or as the result of reflex irritation of the fifth nerve in consequence of disease elsewhere.

In this country, at the present day, and under ordinary circumstances, we meet with intolerance of light in some, but by no means in all, affections of the conjunctivæ. It is with injuries and diseases of the cornea, however, that the symptom attains its greatest prominence; and it is to be noted that its severity is generally in the inverse ratio to the extent of injury or structural lesion. Thus, superficial injuries, such as slight wounds, abrasions, scratches, and ulcers, so long as they implicate the outer layers only of the cornea, are almost invariably attended with extreme intolerance; but as soon as the deeper layers are involved, as in the case of a perforating ulcer, the pain and the intolerance at once disappear or are much diminished. All affections of the cornea may be attended with intolerance; but when the deeper layers, which are not so richly endowed with nerves, are involved, its occurrence is the exception rather than the rule. The exceptional cases are generally complicated with disease of the eyelids or with iritis.

When there is any great degree of intolerance, it is sure to be associated with spasmodic contractions of the orbicularis muscle (blepharospasmus), which is brought about by the reflex action of the fifth nerve upon the facial nerve, the motor nerve of the orbicularis. So intimate is the association, that intolerance of light almost necessarily entails the spasmodic action of the eyelids.

The sensitive nerves which supply the eyeball may be divided into two sets—those which pass through the lenticular ganglion, and those which take their origin directly from the nasal nerve. The conjunctivæ are supplied by nerves which do not pass through the lenticular ganglion; the cornea and iris are supplied by both sets. Upon this plan of innervation, the difference between the sensibility of the conjunctivæ and of the cornea is said to depend.

Affections of the latter without intolerance of light are rarely seen, while those of the conjunctivæ without it are of everyday occurrence. Physiological experiments and clinical observation agree that neither the retina nor the optic nerves are the seat of intolerance, but that it is due to some irritation upon the fifth nerve, and especially of those portions of it which are connected with the lenticular ganglion. The irritation of the dental nerves by decayed teeth is well known as a fertile source of intolerance, and it often happens that corneal affections are much aggravated during the period of dentition.

Serous iritis either with or without ulcers of the cornea, which is usually looked upon as rheumatic in its origin, is generally a very painful affection, and is attended with an extreme degree of intolerance. Again, the first warning of sympathetic inflammation is to be found in the shrinking from light some little time before there is any evidence of structural change; and in a distinct set of cases, which, for want of a better term, may be called cases of hysterical asthenopia, the intolerance will be most obstinate and resisting.

Everybody knows the severe pain which the presence of a foreign body within the eyelids entails, and if it chance to be imbedded in the cornea, it will produce an amount of distress out of all proportion to the injury inflicted. The abrasion of the corneal epithelium only will set up an extreme degree of irritation, which will require very decided treatment. When there is a foreign body present, there can be no rest till it has been removed, a proceeding which is sometimes easily carried out, but, on the other hand, it will sometimes require a good light and practised fingers for its accomplishment. When removed, there is no better plan of treatment than the old-fashioned method of dropping in oil; and, if at hand, a few drops of a strong solution of atropine will diminish the acute sensibility of the nerves, which have been exposed and injured. In a few hours' time the epithelium will be renewed, but till that takes place, the eye should be tied up in such a way that no friction whatever can take place between the eyelid and the surface of the cornea.

In the catarrhal form of inflammation of the conjunctiva it is the epithelial layers which are especially involved. In purulent conjunctivitis the submucous tissue and the thickness of the lid are implicated, and we rarely hear complaints of pain or see any intolerance so long as the cornea is unhurt. But in the kind of ophthalmia known as phlyctenular or pustular, it appears that we have to deal with a distinct affection of the terminal branches of the nerves, in fact with a neurosis, as the result of which, white cells accumulate at the extremity of a nerve, beneath the

epithelium, and give rise to the appearance of minute elevated vesicles (phlyctenules). The contents of these vesicles soon become opaque and puriform (pustules); each vesicle is, as it were, the focus of a small network of congested vessels; they are generally one or two in number, and their usual position is the margin of the cornea. Curiously enough, the presence or absence of pain and intolerance will determine whether the cornea is or is not implicated. When the boundary-line has been over-stepped, even by a single vesicle, you almost invariably find all those distressing symptoms present which have been described under the familiar term "strumous ophthalmia." So long, however, as the cornea is intact, this form of inflammation is essentially harmless. The phlyctenules disperse or burst, and then dry up, and this may be accelerated in a surprising degree by the insufflation of calomel in powder, or by the insertion between the eyelids of a small portion of ointment made of the yellow oxide of mercury, as recommended by Pagenstecher; and as the children who suffer are generally pale and in feeble health, it will be well to administer iron in some shape internally.

Phlyctenular keratitis, herpes corneæ, the disease long known as "strumous ophthalmia." No sooner is the structure of the cornea implicated, than at once the symptoms become much more urgent, and are distressing to witness; treatment becomes very tedious, and the results are anything but satisfactory, for at least two reasons; the one, that the disease has a strong tendency to recur; and the other, that a very large number of eyes receive more or less permanent injury. The main features of the attack have the same character now as in the days when Mr. Tyrrell wrote.[1]

The child is quite unable to open his eyes, and the influence of a very moderate degree of light greatly augments his suffering. He complains of great heat around the eye, and describes the secretion which streams over his cheek as hot or scalding; occasionally he is troubled with violent fits of sneezing. From the excessive intolerance of light he will lie for hours upon his stomach and bury his face in a pillow, or will endeavour to exclude the light more effectually by the aid of his handkerchief or the hands, which he will press closely on the affected organs. But if the hands are forcibly withdrawn for the purpose of examination, the palpebræ are found closely compressed together, and they are corrugated by violent, and sometimes spasmodic, action of the orbicular muscles. Further, it is very common to find the face distorted by the action of the other muscles of this region not connected with the eye. If the attack has been of long standing, the surfaces of the eyelids and of the cheeks are frequently red and

[1] Tyrrell, *op. cit.*, p. 151.

excoriated from the irritation of the scalding secretion, and from the friction and pressure of the hands of the patient, who will often scream and stamp upon the floor in his distress. The examination of the cornea is almost impossible, but it is well to remember that the evidence of disease is generally infinitesimally small when it is compared with the extreme urgency of the symptoms. It is, however, advisable that one thorough examination should be made in the early stages of the attack. A judicious mixture of force and persuasion may succeed in this, but in a hospital a few inhalations of chloroform make the most effectual means to this end. In some of the very aggravated cases, chloroform should be employed frequently—as often, in fact, as it is thought advisable to ensure the proper application of local remedies. This method of treatment can, of course, be best carried out within the wards of a hospital. When the surface of the eye can be properly examined, it often happens that there is very little appearance of any structural lesion, although, as a rule, one or more minute irregular opacities are to be seen at or near the centre of the cornea. In some instances there is a distinct ulcer, with yellow infiltration of the tissue around it; but just as frequently there is but a small opacity (macula), to which a few scattered blood-vessels pass from the conjunctiva across the cornea. As a rule, both eyes are affected, but if one is free from disease it will display an equal amount of intolerance with the other.

According to Ivanoff, the disease has its origin in the track of the small corneal nerves. Collections of white cells take place beneath the epithelium, and form the well-known vesicles. The extreme pain and intolerance are the direct result of irritation of the nerve fibres, and the amount of injury inflicted upon the cornea in different cases depends entirely upon whether or not the surrounding tissue of the cornea is involved. In a few fortunate instances the vesicle dries up, and the surface of the cornea is completely restored; but, as a rule, there remains more or less of permanent opacity, although to so slight an extent as to require careful examination for its detection.

As regards the treatment of these cases, no very precise rules can be laid down. Whether or not the disease can be justly ascribed to any peculiarity of constitution—formerly called scrofulous, there is no doubt that the more urgent symptoms will often yield when some source of irritation elsewhere than in the eyes has been discovered and removed. So frequently is an attack connected with errors of diet, that the disease has been called by men of experience "gastric ophthalmia;" and if we reject the term itself, it will nevertheless remind us how necessary it is to watch very carefully the diet and the general regime of nurseries which

are generally supposed to be well regulated ; for although the disease is most frequently met with amongst hospital patients, it is no less true that the pampered and over-fed children of well-to-do persons occasionally suffer most severely.

Foremost amongst local remedies, and one which should be firmly persevered with, stands belladonna in some shape or form. The extract dissolved in boiling water makes a most soothing fomentation, but it sometimes happens that the incessant warmth and moisture which attend its use will give rise to great cutaneous irritability, and to the efflorescence of herpetic vesicles upon the parts around the eyes. On the other hand, the solutions of atropine do not always give satisfactory results. They have more than once been known to cause dangerous symptoms of poisoning ; but the objection to them is rather of another kind, that they are uncertain and inert. The gush of tears which follows their application has something to do with this. At other times these solutions are found to be exceedingly irritating, for their composition is unstable. Under such circumstances, it may be possible to gain the soothing effect by the use of a liniment in which the extract has been rubbed up with glycerine. In any case, it will be found that, as soon as the full effect can be produced upon the pupil, the most distressing of the symptoms will subside. On the whole, it will be found extremely difficult to produce the full effect of belladonna upon the eyes of out-patients without considerable delay and without great waste of material ; and I think that it will be found more satisfactory to see the solution properly applied twice a week, than to trust it to inattentive hands for more frequent use. Another remedy which I have found of very great value in numberless cases is the cold-water douche to the nape of the neck. One or two applications in the morning will some times dispel the intolerance as by a charm. This plan, however, can hardly be properly carried out except in a hospital, or under the favourable conditions of private practice. When the acute stage has passed, and the cornea is left roughened, and with its surface faceted with the scars of minute vesicles, the local application of calomel or of the yellow mercurial ointment will once more be found of great service. An objection to this ointment is that it decomposes, changes its colour, and becomes irritating, so that it should be used when freshly made or not at all. Again, neither of these remedies should be employed when there is much intolerance, nor when there is ulceration of the cornea. Their effect is probably in great measure mechanical, and when used under suitable circumstances, their value is very great ; but their indiscriminate use in affections of the cornea has sometimes brought ridicule upon the medical adviser.

The ordinary and very various plans of general treatment which have been found useful are familiar to everybody, but I would put upon record, that in the summer months, when the disease of the eye has been supplemented with a turgid mucous membrane within the nose, and with thickening of the upper lip, I have often found chlorate of potash in large doses a most useful medicine.

Although it sometimes happens that a child may pass through an attack as above described, and may escape with his eyes unhurt, it more often happens that one or both of them will be permanently injured, to the extent of one or more small macule, no bigger than a pin's head perhaps, but, situated, as they generally are, in the very centre of the cornea, over the pupil, they are most detrimental to good sight. No doubt the impairment of sight which such an opacity will cause depends much upon the circumstances of the patient, and to some extent upon his individual disposition ; for in the case of nervous and sensitive patients, these faint opacities not only prevent accurate vision, but they cause great annoyance by the distortion and multiplication of small objects. In some instances relief may be found in the employment of weak concave glasses, but, as a rule, the child soon learns to suppress the image of the faulty eye, and thus deprives himself of the advantages of binocular vision.

A more immediate, and a very disheartening result, is the extreme tendency to recurrence which these cases often exhibit. In spite of every care on the part of the medical attendant, the slightest irregularity of health or habit is sure to be the signal for the renewal of the intolerance and its attendant spasm of the eyelids. The attacks frequently recur at stated times—in the spring and autumn, for example ; they appear as the result of any kind of external irritation, however trivial, such as exposure to wind and sunlight ; the intolerance is again extreme, with copious lachrymation. The ciliary vessels may appear enlarged, and there may be a circumcorneal zone of congestion ; but beyond a slight haziness around the original seat of disease, there is no other change visible. These recurrences are not usually very severe, but they take place almost without warning, and the disease may as suddenly disappear. They interfere sadly with education, and are exceedingly difficult to treat, inasmuch as the surgeon has to overcome a tendency to disease rather than disease itself. As this habit of recurrence will, under favourable conditions, wear itself out by degrees, it is well to do what we can to ward off all possible sources of irritation. In attempting this, much help may be derived from some form of counter-irritation. The application of iodine paint over the brow or behind the ear is often of great service, and still better results have been obtained by the insertion

of a small seton, a single silk thread behind the ear; this causes but little inconvenience, and when concealed by the hair, can be worn without interfering with school-life.

Another unfortunate result of the formation of small corneal opacities is the occurrence of strabismus. The exact mode by which this is brought about is not very clear, and probably more than one factor is concerned. The explanation commonly given is, that in consequence of the opacity of the cornea, the distinctness of the retinal images of the affected eye is more or less impaired, and the difference in the clearness and intensity of the retinal images of the two eyes is often very confusing and annoying to the patient; and in order to escape from this annoyance, he involuntarily squints with the affected eye, so that the rays from the object may impinge upon a more peripheral, and therefore less sensitive, portion of the retina, and the image of this eye be consequently so much weakened in intensity, as not to prove any longer an annoyance. The direction in which the deviation may take place is generally determined by the strength of the muscles, and is almost invariably inwards, or inwards and upwards. The image of the squinting eye will be gradually suppressed, and then amblyopia from disease of the eye will be superadded to the weakness of sight caused by the original affection. It has been pointed out by Pagenstecher that hypermetropia exists in very many of these cases, and must be regarded as the true cause of the strabismus; while other authorities, and especially Donders, think that the inflammation which causes the corneal opacity may extend to some of the muscles, and at first bring on a spasmodic, and then an organic, contraction of the muscular tissue.

Such squints as these are not favourable cases for operation, unless it be for the sake of some improvement in personal appearance.

Intolerance of light is also occasionally a symptom of great importance, pointing to the existence of what may be termed sympathetic irritation, arising in one eye as the result of some injury previously inflicted upon the fellow-eye. It is one of the earliest symptoms of this condition, and if the warning be attended to, and the injured eye be at once removed, the vision of the other and sound eye may be preserved intact. Clinical observation shows more and more clearly that many cases of sympathetic inflammation are preceded by a premonitory stage, which may be called a stage of irritation only, where as yet there is no structural lesion. Most certainly all cases are not preceded in this way, and this period of incubation, so to speak, is not as a rule a very lengthy one, although Mr. Carter has put on record some observations which show that sympathetic irritation may

occasionally exist for a very considerable time, and with no development of destructive inflammation.

Hysterical asthenopia.—The anomalous and wide range of symptoms which are now included in the term "asthenopia" formerly admitted of no very precise explanation, and we consequently find them arranged under very different headings in the writings of the older ophthalmic surgeons. The announcement by Donders that very many of them were to be explained by the difficulty in accommodating for near objects and for prolonged work by eyes which were naturally deficient in refractive power (hypermetropia), was shown to be so well founded that it led to a complete reaction of opinion, during which there was some danger of its being thought that nearly all functional defects of the eye were directly or indirectly the result of errors of refraction, and were to be cured by the use of proper glasses. There is abundant evidence to show, if necessary, that many a young man's education and career have been checked, if not entirely spoiled, because the difficulty with which he pursued his studies admitted of no rational explanation, and consequently of no relief. On the other hand, anybody who has any great experience of the out-patient rooms of a large hospital will soon come to the opinion that there is a distinct set of individuals whose symptoms are those of aggravated asthenopia, and yet in whose eyes the error in refraction is so trifling, that it is impossible to suppose that it can be the sole, or even the chief, cause of so much inconvenience. Such cases are for the most part young women. To the ordinary symptoms of asthenopia are added those of hysteria. Intolerance of light is nearly always present, and this is sometimes aggravated by the habit of wearing dark glasses, especially amongst the better class of patients. Although these cases bear a general resemblance to the ordinary form of accommodative asthenopia, yet there are distinctive features between them. In the asthenopia of hypermetropes, there is an undoubted error in refraction, and the pain and sense of fatigue complained of can be referred to the efforts to accommodate; and they are increased or diminished in proportion as the exertion of the ciliary muscle is prolonged or suspended. The pain, too, is generally referred to the eyeball, or to the parts immediately around it. In the hysterical form, however, the pains are exaggerated and perpetually shifting, sometimes localised in one situation, and sometimes in another. They come and go independently of any particular exertion of the eye. Indeed, these patients give up work altogether, and profess themselves unable to do anything. There is no tenderness on pressure over the track of any particular branch of the fifth nerve, as there probably would be in the case of an ordinary neuralgia. The symptoms

are much aggravated by bright light, and by any cause which in any degree disturbs or agitates the nervous system.

Such cases used to be described under the head of hyperæsthesia oculi, and one of their very remarkable features is the variation in severity which occurs from time to time. Sometimes these patients will feel very well, and at others, for no very evident reason, will express themselves as feeling most miserable, and are evidently in great distress.

A special study of cases of this kind has been made by Dr. Foerster of Breslau,[1] and he has been able in many instances to connect these symptoms with disorders of the uterine functions. No doubt many ordinary cases of asthenopia are much aggravated in seasons of disordered health; but the very severe cases which we sometimes meet with are so tedious, and unmanageable by ordinary means, that it is a real gain to be able to trace their origin to any structural lesions such as Dr. Foerster has described.

Not many years ago, young men, wretchedly out of health in many ways, were frequently seen in the out-patient rooms, who at times could hardly open their eyes, while at others they were tormented and made miserable by the apparition of all kinds of muscæ volitantes. Many of these patients were suffering from long-standing gleet, and very often, rightly or wrongly, were supposed to be the victims of self-abuse. Such cases were at any time most difficult to treat, and it is fortunate that it is very rarely nowadays that any such present themselves.

[1] Handbuch der Gesammt Augenheilkunde, vol. vii. p. 88.

FATTY TUMOURS IN INFANCY AND CHILDHOOD.

BY

HENRY T. BUTLIN.

Although children are often well nourished or *fat*, the occurrence of excessive deposit of fat is not common in any form. There are cases on record of excessive obesity in quite young children,[1] and even in new-born infants, but they are not nearly so frequent as records of similar conditions in adults. There are also cases of what may be called "partial obesity,"—cases in which the adipose tissue of a certain region of the body is greatly in excess of the adipose tissue generally, and yet the characters of "a tumour" are wanting. Of such a nature is the case related by Lebert[2] of a female, aged nineteen, the left side of whose abdomen was the seat of enormous increase of its fat. This condition was first noticed when the patient was six months old, and was thought to have been congenital. The growth of the mass had been in proportion to the growth of the maiden, and when she ceased to grow, the left side of the abdomen also stopped growing; so that from first to last it retained the same relative proportion to the size of the patient. The greater part of it was removed by Dieffenbach, who, finding no definite or circumscribed tumour, left a layer of fat still covering the abdominal muscles. Lebert calls this a "lipoma diffusum."

Bearing a certain relation to this condition, and at the same time closely allied to elephantiasis, is the congenital hypertrophy which occasionally affects a limb or a portion of a limb; as, for instance, one or more of the toes or fingers. A case of this nature

[1] Wardell, Remarks on Obesity, 1849; Wadd, Cursory Remarks on Corpulency.
[2] Lebert, Abhandlungen aus dem gebiete der prak. Chirurgie, 1848.

has just occurred in the Hospital in the person of a girl ten years old.[1] The great toe and inner side of the foot were exceedingly large, and so much out of proportion to the rest of the foot, that the toe was amputated by Mr. Smith. The phalanges were considerably longer and larger than normal, but the great bulk of the toe was due to hypertrophy of the subcutaneous fat. Yet this toe was to a certain extent well proportioned. It had grown as the child grew, and had throughout retained the same relative proportion to the rest of the foot. In an excellent article on these congenital hypertrophies, Busch[2] describes and figures a somewhat similar but still more troublesome condition. The patient was a young man, several of whose toes where hypertrophied, and whose foot was so much enlarged by irregular outgrowths of fat in the form of large tumours, that after amputation the foot weighed more than 7 lb. In this case the bones were enlarged, but had not preserved their due proportions; some of the joints were obliterated, and the whole foot was exceedingly deformed. The affection was congenital, at first somewhat resembling the affection last described; but the relative proportions had been more and more disturbed by undue and irregular growth of the diseased parts of the foot. Such outgrowths of adipose tissue as these can scarcely be considered as tumours, although they fulfil one of the conditions belonging to the definition of a tumour, viz., their growth is to a certain extent independent of the growth and development of the rest of the body. But their occurrence in connection with extensive coincident disease of other structures gives them an entirely different character, and renders it unnecessary to consider them further.

Fatty tumours, properly so called, are not often met with during infancy and childhood. Indeed, so uncommon are they, that Paget and Moore[3] speak thus of them: "Fatty tumours come under treatment in young persons and adults of all ages. They are very rare in children," and Mr. Pollock[4] "had not been able to meet with a single instance on record of a fatty tumour commencing in infancy." The various works on the surgery of childhood either say very little on the subject of fatty tumours or totally ignore it. During the last four years and a half, two cases have come under my notice in the Hospital. The first case occurred during the year 1873, in a child[5] twelve years of age, who had a large oval tumour of the left side of the neck, lying

[1] Lucas Ward Book, vol. iv. p. 410.
[2] Busch, Langenbeck's Archiv, Bd. vii. s. 187.
[3] Paget and Moore, Holmes' System, art. Tumours.
[4] Pollock, Path. Soc. Trans., vol. viii. p. 360.
[5] Lucas Ward Book, vol. ii. p. 72.

superficial to the sterno-mastoid muscle. It was said to have been growing slowly during three years. At a consultation held upon the case, doubt was expressed as to the nature of the tumour, which felt like fat, but was thought not to be fat on account of the youth of the patient. It was at first treated by injections of iodine, on the supposition that it was of glandular origin; but it got larger and larger, and was at last removed by Mr. Smith, under whose care the patient was. It proved to be a fatty tumour, encapsuled, adherent to the surrounding tissues, but removed without any real difficulty. With the fat, of which it was chiefly composed, was mingled a much larger proportion of fibrous tissue than is usually found in lipomata.

The second case also occurred in Mr. Smith's wards, in the person of a girl[1] seven years old. This child had a very large tumour of the calf, which had been first noticed when she was a year old. The tumour was removed with the help of Esmarch's bandage, but even then not without some difficulty, as it passed between the bones into the front of the leg. It was a large fatty tumour, perfectly encapsuled, containing a much greater quantity of fibrous tissue than is generally the case in lipomata, and in addition traversed by a number of fine bands of striated muscular fibre. The operation was followed by an attack of pyæmia, with effusion of pus into the knee-joint, necessitating amputation of the thigh, after which the child made a good recovery.

These tumours illustrate some of the features in which the fatty tumours of childhood differ from those of adults.

The majority of them, probably the large majority, are congenital. The tumour of the calf described above was probably congenital, but was not noticed until the first attempts at walking drew attention to the legs. One of the most remarkable cases illustrating this point is that described by Gruber[2] of an infant girl who was born with a large tumour behind the rectum, covering the coccyx and sacrum. It was almost double the size of the child's head, of globular shape, with a small base, its surface generally normal in appearance, except at the summit, where there was redness and excoriation, from which blood flowed so freely that the tumour was obliged to be at once removed. Its weight after removal was $2\frac{1}{4}$ lb., whilst the total weight of the child minus the tumour was only $5\frac{1}{2}$ lb. It consisted of peculiar-looking fat, forming many lobes, which were bound together by fine connective tissue; it exhibited calcified spots and points of ossification; and the coccyx was continued into it in the form of a finely tapering cartilaginous point to the distance of three-quarters of an

[1] Path. Soc. Trans., 1877; Lucas Ward Book, vol. v. p. 206.
[2] Gruber, Schmidt's Jahrbuch, Bd. xxx. 1841, s. 184

inch. Twelve hours after the operation the child died. Arnold[1] describes a still more wonderful infant, a child who survived its birth six days in spite of a huge tumour filling its pharynx and mouth, projecting out of the latter like a hideous hypertrophied tongue, and growing far up into the cavity of the skull through its base. It was supposed to have grown from the substance of the tongue, and was a lipoma, inasmuch as it consisted chiefly of large lobes of fat, but it also contained fibrous tissue, cartilage, sarcoma tissue, cysts, and even striated muscular fibres, which were supposed to have been included within it during its gradual growth.

Although they are so frequently congenital, there is no evidence to show that these tumours are hereditary. Nor are they multiple and symmetrical, like the multiple and symmetrical bony tumours occurring not seldom in children.

Their growth is, for the most part, slow and steady, but there are several cases on record in which it has been most rapid. Thus Closset[2] reports a congenital tumour of the foot in a boy six years old, which had increased during the last year from the size of a hen's egg to that of a child's head. And Suttina[3] describes a tumour of the loin in an infant, which grew so quickly that the rapidity of its growth rendered the diagnosis uncertain between lipoma and sarcoma.

Even with slow growth they often attain a very considerable size. The tumour removed from the calf of J. G.[4] was as large as a child's head. Mr. Pollock[5] exhibited to the Pathological Society some years ago a tumour weighing nearly 13 lb. removed from the back of a girl seven years old. Dohlhoff's[6] case of tumour of the dorsum of the foot in a boy of eleven, reached so large a size that the boy could no longer walk. But most wonderful in proportion is the case already mentioned, described by Gruber,[7] of a tumour weighing $2\frac{1}{4}$ lb. in a new-born child whose weight without the tumour was no more than $5\frac{1}{2}$ lb.

The seat of fatty tumours in children is much more varied than in adults. Thus several cases have been reported of lipoma of the foot,[8] both of the dorsal and of the plantar aspects. The

[1] Arnold, Virchow's Archiv, Bd. l. s. 482.
[2] Closset, Deutsche Zeitschrift. f. Chir., 1873, Bd. ii. s. 553.
[3] Suttina, Schmidt's Jahrbuch, Bd. cli. s. 57.
[4] See case described, p. 183.
[5] Pollock, Path. Soc. Trans., vol. viii. p. 360.
[6] Dohlhoff, Schmidt's Jahrbuch, Bd. xxv., 1840, s. 85.
[7] Gruber, *loc. cit.*
[8] Gay, Path. Trans., vol. xiv. p. 243; Dohlhoff, *loc. cit.*; Closset, *loc. cit.*

tongue,[1] the pharynx,[2] middle of the calf,[3] neck of the radius,[4] zygomatic fossa,[5] have all been the seat of such tumours. The side of the neck appears to be the most frequent seat. Lebert[6] describes a most formidable specimen removed by Dieffenbach from the neck of a young man twenty-three years old, where it had been growing since the patient was six. It was as big as a man's head, had grown slowly and regularly, and now extended from the spine round the side of the neck and beyond the middle line. The sacrum and coccyx,[7] and the loin,[8] stand next to the neck as favourite seats; but whereas those of the loin and upper part of the back appear to arise from the subcutaneous or intermuscular fat, and occur after birth, those of the sacral and coccygeal region are almost invariably congenital, and often have a much deeper and more important origin. Gruber's case, in which the coccyx was continued into the middle of a tumour, has already been mentioned. Athol Johnson removed a tumour from over the sacrum which lay partly within the spinal canal, pressed upon the spinal cord, and produced serious symptoms of spinal irritation.

Bryant[9] has called attention to a peculiarity which the fatty tumours of childhood occasionally present in being diffused and without capsule. He speaks of it as if it were of common occurrence, whereas it is the exception, not the rule, save in those conditions described at the beginning, of this paper. There is a peculiarity, however, which may be frequently observed, namely, the mixed nature of these growths. They are usually much firmer than the lipomata of adults, on account of the much larger quantity of fibrous tissue which they contain. The tumour of the calf described above was an excellent example of this. It contained almost as much fibrous tissue as fat, the fibrous tissue forming much larger and thicker trabeculæ than usual, separating the lobes and lobules of fat to a much wider distance, and knitting the whole together into a compact mass. This tumour also contained striated muscular fibre, like the great tumour described by Arnold.[10] Bone[11] has been several times mentioned amongst the constituents of these mixed tumours, but unless this has been confirmed by microscopical examination, it would be much easier

[1] Bastien, Bulletins de le Société Anatomique, 1854, p. 349; Arnold, *loc. cit.*
[2] Lanbl, Beobachtungen aus dem Franz Josef Kinder Spit. in Prag., 1860, s. 181.
[3] Case described above. [4] Smith, Path. Soc. Trans., vol. xix. p. 344.
[5] Wood, Path. Soc. Trans., vol. xxvi. p. 190.
[6] Lebert, Abhandlungen aus dem gebiete der prak. Chir., Berlin, 1848, s. 115.
[7] Johnson, Path. Soc. Trans., vol. viii. p. 16; Gruber, *loc. cit.*
[8] Suttina, *loc. cit.*; Pollock, *loc. cit.*
[9] Bryant, The Surgical Diseases of Childhood, 1863.
[10] Arnold, *loc. cit.* [11] Bastien, *loc. cit.*; Smith, *loc. cit.*; Gruber, *loc. cit.*

to believe that these bony points and masses were points and masses of calcareous degeneration. Wood[1] brought a case before the Pathological Society of a tumour composed of fat and mucous tissue. It was described by the Morbid Growth Committee as a myxoma containing a variable amount of fat; in either case, it calls to mind the close relation between these two tissues which Virchow[2] believed to exist. Weber[3] has given an account of a mixed growth consisting of telangiectasis, fat, and fibrous tissue, which he believed to originate as a nævus in which fat and fibrous tissue had been developed as a secondary change. Walther's[4] celebrated case has not been included in this paper, because it appears much more likely to belong to the class fibroma (F. molluscum) than lipoma, otherwise it would afford another example of the connection of fatty tumour with telangiectasis.

The origin of the fatty tumours of childhood is a most interesting question. No doubt many of them arise, as they do at a later age, from the subcutaneous or intermuscular adipose tissue, or the fat of some other part. A few of them may originate in the degeneration of nævi, as just mentioned. But there is reason to believe that many of them have a totally different origin. It seems very probable that they commence as fibrous or fibrocellular tumours, and that the formation of fat is a later change, not very late perhaps, but occurring soon after the first appearance of the tumour, and proceeding almost collaterally with its growth. The large quantity of fibrous tissue contained in most of these tumours, the ill-developed nature of the adipose tissue as shown by the microscope, and the strange fact that nearly half the cases collected are stated to have been attached to bone or periosteum,[5] sometimes immediately, sometimes by a fibrous pedicle, whilst several of them are stated actually to have arisen from bone, have led me to this belief. A less likely bed than the periosteum for the origin of a fatty tumour can scarcely be imagined, whereas it is one of the ordinary seats of origin of fibroma.

The diagnosis of fatty tumours in young subjects is occasionally rendered difficult by the large quantity of fibrous tissue they contain, by the strange positions in which they often grow, and by the fact that they are believed to be still more uncommon in children than they really are. Their slow growth, however, and

[1] Wood, *loc. cit.* [2] Virchow, Krankhäften Geschwülste, Bd. i.
[3] Weber, Müller's Archiv, 1851, p. 74.
[4] Walther, Die Angebohren Fetthantgeschwülste, Landshut, 1814.
[5] Smith, *loc. cit.*, vol. xvii. p. 286; Holmes, Path. Trans., vol. xvi. p. 236; Gruber, *loc. cit.*; Wood, *loc. cit.*; Closset, *loc. cit.*; Dohlhoff, *loc. cit.*; Jallet, Gaz. des Hôpitaux, 1867, p. 194; Lebert, *loc. cit.*

long duration have usually been sufficient to make it evident that they are innocent tumours, so that even when the exact nature of the tumour has not been certain before removal, the uncertainty has not led to any serious error of treatment. In Gay's[1] case of fatty tumour of the sole, the foot was amputated, not so much because the recurrence of the growth and its rapid progress led to the belief that it was malignant, as because the child appeared too weakly to recover the hæmorrhage which must have attended its dissection.

The treatment, fortunately, as a rule, is simple enough—removal wherever it can be accomplished. Tumours about the spinal column, more particularly those about the sacrum and coccyx, require to be dealt with much more cautiously than those of other parts, as they occasionally have deep and important attachments, rendering their complete removal improbable, or even impossible. But even such tumours have been removed without that apparent increase of danger which might have been expected, or the operation has been stayed before its completion has led to the damage of any very important structure.

[1] Gay, *loc. cit.*

CASE

OF

ANEURYSM IN THE GLUTEAL REGION AFTER A PUNCTURED WOUND.

BY

E. L. HUSSEY.

A man, aged fifty-three years, a clerk and warehouseman in a large china shop, in fairly good health, but not of strictly temperate habits, was admitted into the Radcliffe Infirmary, Oxford, under my care, on the 27th of April 1877, with a large aneurysmal swelling in the left gluteal region, about the size of two doubled fists, the result of a wound received on the 10th of January.

History.—On the evening of the 10th of January, during some altercation about drink, his wife—a woman of violent temper, and then under the influence of drink—struck him in the left gluteal region with a short broad dagger. He was on his feet at the moment, trying to avoid the blow as she was pursuing him. He ran out of the house, to a friend's about fifty yards off, with the blood flowing freely from the wound. He fell down in the street, "in a pool of blood," and was at first supposed to be dead. He was brought home by the neighbours; and the nearest medical practitioner, Mr. Druce, was called to him. Upon Mr. Druce's arrival, the bleeding had ceased; and there was not any swelling to be felt underneath the skin.

Without resting at home, as directed, the man went to his work the next day. The outer wound soon healed. He felt little inconvenience from the injury, and did not make any complaint. Some three or four weeks afterwards, on rising from his chair one evening, he felt a pain at the part, and on putting his hand there, he found a small substance deep under the skin,—

"like a small core," he described it. He afterwards applied to Mr. Weaving, the medical officer of the sick club, of which he was a member.

On the 18th of March I saw him with Mr. Weaving. Behind the trochanter major there was a swelling about the size of a small orange, deep under the skin and muscle, with a cicatrix wide and spread out, rather more than an inch in length, prominent, and almost pointing in the middle of the swelling. The swelling was tense and elastic; it could not be emptied or lessened in size by pressure. The thrill or whiz of an aneurysm was clearly detected by the ear applied to the part: there was not any distinct impulse communicated to the hand. There could be no doubt that the swelling was a false aneurysm, the result of an injury; and that there had been a wound of the gluteal artery, or one of its chief branches. The nature of the case was pointed out to the patient, and the necessity for an operation. Rest in bed was advised, with moderate diet.

Notwithstanding the advice so given, he continued at his business; and walked backwards and forwards to his home, a distance of a mile and a half, twice a day. Increasing pain and the inconvenience of moving the limb compelled him at length to keep at home and to take to his bed.

At the time of his admission into the Infirmary (27th April) the swelling had increased much in size—it was fully three times as large. The enlargement was most visible at the sides. The shape was oval, and the cicatrix was not so prominent and pointing. The long axis of the swelling was in the transverse direction. The outline was not so clearly defined. In other respects the character of the swelling was not altered.

Treatment.—On the 30th of April the patient was put under chloroform, and Lister's large clamp or tourniquet placed on the aorta below the umbilicus. An incision about eight inches long was made through the integument over the swelling, in a line from the anterior part of the crest of the ilium toward the middle of the sacrum, and passing through the cicatrix. A thin, but tough, membrane covering a cyst was then exposed in the middle of the line of incision, bulging forwards in a globular form. In attempting to deepen the incision the cyst was punctured, and a great mass of arterial blood—fluid and in clots—was immediately ejected with great force. The opening in the cyst was enlarged, and my left hand thrust to the bottom, making pressure with the fore and middle fingers upon the spot from which the blood issued, against what seemed to be a strong fascia or ligament. The flow of blood was stopped by the pressure. The original incision through the integument was then lengthened toward the

front by about three inches; the clotted blood was cleared from the cyst, and the wound wiped out with a sponge. At the bottom of the cavity an opening or slit about a quarter of an inch long was seen, from which arterial blood was ejected in a full stream with a strong impulse, upon slackening the pressure made with my fingers. The cavity was evidently the sac of an aneurysm; and the slit or opening was a wound in a large artery. The edges of the slit were rather thickened, and were not so clearly defined as in a recent wound of a large artery made with a sharp instrument. The wounded vessel, and the part of the sac immediately around it and connected with it, was raised with two pairs of forceps; the tough membranous lining was a little cleared or scratched away with the point of the scalpel, and an aneurysm needle, armed with a double thread of strong twine, was passed underneath, using some degree of force in passing it. The two threads were then tied separately, one on each side of the opening. The one toward the sacrum, the proximal in the course of the circulation, was tied first, and the end left long: a feeble pulsation was observed to be communicated to this ligature. The hemorrhage continued, though without arterial jet or impulse, until the other ligature, that toward the trochanter, the distal in the course of the circulation, was drawn tight; it then ceased. The knot of this ligature was cut off short and left in the wound. One muscular branch continued to bleed after the man had recovered from chloroform, and was tied with a fine thread. The edges of the wound were brought together and fixed with interrupted sutures and strips of plaster. A compress of lint with cerate was laid over it, and a flannel roller bound round the pelvis.

During the operation the man lay on his right side, with his knees partly bent; and the tourniquet slipped from the position in which it had been placed. It seemed to me, however, that the bleeding was to some extent checked when the instrument was properly in position.

The blood which escaped from the sac, fluid and in clot, was about a pound and a half in quantity. In the further steps of the operation very little more was lost. Some of the outer part of the clot from the cyst was in layers, resembling the "laminated coagula" in the sac of an ordinary aneurysm, though not so tough in consistence.

The man recovered quickly from the effects of the operation, and was carried at once to the ward.

On the morning of the 6th of May, the ligature applied on the sacral side of the wounded artery was seen to be lying loose between the edges of the wound, and by the side of it a thin piece of sloughing tissue about an inch long. The outer dressing

was changed without anything unusual being observed. About two hours afterwards—between eleven and twelve o'clock—the patient called the attention of the nurse to some blood which he found was flowing from the wound. Before the House-Surgeon reached the ward several ounces had escaped. The patient was put under chloroform. The sutures were divided, and the edges of the wound separated. Some clots were cleared away, and a small vessel was found to be bleeding. A ligature applied to the bleeding point did not hold, and the hemorrhage continued. Another ligature was applied, including within the ligature some of the surrounding tissue; the hemorrhage then ceased. The wound was then drawn together with strips of plaster, and a roller was applied round the pelvis as at first.

The next day—the 7th—about an hour after the dressings were changed, and before I had left the house after seeing the patient, the nurse observed blood flowing freely from the lower part of the wound. The wound was again opened, and arterial blood was seen flowing from the part where the ligatures had been applied. The bleeding seemed to come from the knot of the distal or trochanteric ligature, which still held fast. A sharp-pointed needle, armed with a thread of strong twine, was thrust in, about an inch nearer to the trochanter, so as to pass under the line in which I supposed the trunk of the wounded artery lay. On withdrawing the needle, arterial blood issued freely from the puncture made by the needle. The needle, armed again in the same way, was then thrust through at a point about half an inch further off, toward the trochanteric side, and the thread tied. After this the bleeding ceased. The wound was left open, being merely covered with a piece of linen rag, spread with cerate.

On the 9th, between four and five o'clock in the afternoon, hemorrhage again returned. Upon separating the edges of the wound, the House-Physician observed arterial blood flowing from the point at which the proximal or sacral ligature had been placed. He seized the point with forceps, and twisted it. Altogether about four or five ounces of blood had escaped. About six o'clock I saw him; and I passed an aneurysm needle, with a thread of strong twine, deep under the point from which the blood had been observed to flow, and tied the thread. The patient was too low in general power for me to have any reasonable hope from a further operation, such as ligature of the iliac artery within the pelvis.

On the 11th, about half-past seven in the morning, the House-Surgeon was again called, on account of a fresh loss of blood, to the extent of about four or five ounces. The bleeding stopped

upon a compress of lint being applied, and held for a short time with the finger at the part.

The progress of the patient and the appearance of the wound were not satisfactory. There was not any healthy secretion from the wound. After the first few days the odour from it was more like that from mortification than from a large open wound or suppurating surface. Under the frequent returns of hemorrhage the patient's strength began to fail. From the first he showed little of the strength and resolution which best show the power a patient has to recover after a surgical operation.

On the evening of the 15th some slight bleeding was observed: it was easily stopped by pressure. A little after midnight the bleeding returned more freely. Upon clearing the bottom of the wound from the threads and the sloughing mass in which they were entangled, the open mouth of a large artery was observed situated deep at the bottom. This was transfixed with a tenaculum and tied. No further bleeding took place.

The man, whose strength had been failing during the last few days, slowly sank, and died, exhausted, about three o'clock the following morning, the 16th.

The body was examined the next day. The internal iliac vein of the left side, with several of the tributary branches, was of a uniform dusky red colour; as were also the external and common iliac veins of the same side, and the lower part of the ascending vena cava. No pus or lymph was observed within them. On the right side the iliac veins and their branches were natural in colour and appearance, and did not exhibit any morbid signs. The arteries within the pelvis were uninjured. The trunk of the gluteal artery was traced through its course to the point where it divides. The main artery and the ascending branch were not injured. The artery which had been wounded was found to be the large descending branch. The various small vessels could not be traced in the mass of sloughing tissue surrounding the site of the aneurysm.

Remarks.—The operation of securing the wounded artery by opening the aneurysmal sac was not found to be of so formidable a character as I supposed it would be from having read the cases which have been recorded.

The wound in the vessel was exposed without difficulty by laying the sac open. By passing the hand at once to the bottom of the sac, in the line in which the stream of warm blood was felt by the fingers, the further hemorrhage was checked by pressure, and the flow of blood was under command.

The amount of blood lost at the time of operation was not

greater than I have seen in some cases where a large artery has been wounded, or an aneurysmal sac opened.

In the cases recorded by Mr. John Bell (Principles of Surgery, vol. i., 1801), and by Mr. Syme (Observations on Clinical Surgery, 1861), the patients recovered. There was not, therefore, any opportunity given of learning by dissection whether it was, as they supposed, the trunk of the gluteal artery itself that was wounded, or one of the primary branches, as in this case.

Some of the difficulties mentioned by other surgeons may perhaps have arisen from that circumstance, if the main artery was wounded. The shorter trunk of the main artery outside the pelvis allows less space for compression with the fingers during the operation undertaken for securing it, and less space, of course, for placing a ligature round it.

In the case mentioned by Mr. Guthrie (On the Diseases and Injuries of Arteries, &c., 1830), the particulars are not given with sufficient clearness to show what vessel was injured, or whether the wound was that of a single branch; and the short note of the examination after death does not afford information upon either of these points.

Additional note.—Mr. Briscoe has told me of the following case, which was under treatment in the Radcliffe Infirmary when he was the House-Surgeon.

A woodman, forty-one years of age, was admitted into the Infirmary on the 9th of July 1846, under the care of the late Mr. Fisher, with a large and deep incised wound in the right gluteal region, behind the trochanter, received five days before admission.

The man was at work with other men felling timber in a wood about twelve miles from Oxford. As he was stooping down he received a blow upon the buttock with the axe from one of the other men. He was knocked down by the blow. The wound bled profusely, but the bleeding soon stopped. With very little delay the edges of the wound were brought together with strips of plaster by a neighbouring practitioner, who was quickly in attendance. The hemorrhage, however, returned more than once, though not to the same alarming extent, and the man was brought to the Infirmary.

There was not any bleeding at the time of admission, but at different times afterwards, in the course of the next three or four days, the bleeding returned to an extent to make it clear that a large artery had been wounded.

After about a week, the late Mr. Cleoburey took charge of the patient during Mr. Fisher's illness. The wound was opened. The spot from which the House-Surgeon had observed that the bleeding issued was where the gluteal artery comes out from the

pelvis, and turns forward. This was examined carefully, and search made for an open vessel, but none could be detected. As a matter of precaution and security, a curved needle armed with a strong thread was passed deeply through all the tissues, and the ligature drawn firmly round the needle; the needle was then left in the wound.

No further hemorrhage took place. The wound healed slowly; and the man left the Infirmary on the 16th of September, in no way the worse for the injury.

ON

COMPLETE INTRA-PERITONEAL LIGATURE

OF THE

PEDICLE IN OVARIOTOMY.

BY

ALBAN DORAN.

In the twelfth volume of these Reports I discussed at some length several questions relating to the impaction of foreign bodies in the tissues. I endeavoured to prove that the archives of modern surgery, British and foreign, offered strong evidence in support of two facts. In the first place, small inorganic foreign bodies have been known in numerous instances to remain embedded in different parts of the body without exciting any pathological changes. Secondly, such fortunate results are not the rule, foreign bodies generally inducing morbid conditions in the structures surrounding them, changes which are most likely to terminate in suppurative inflammation.

It is by the clinical history of accidental wounds that the surgeon is guided when compelled to inflict surgical wounds on patients for the cure of disease or for the relief of suffering. Knowing what kinds of injuries from sharp instruments heal by first intention, and under what circumstances they are enabled to heal in that desirable fashion, he ensures that kind of union, when he requires it, by inflicting similar wounds with keen-edged knives, and by placing the patient under similarly favourable circumstances. Aware that crushes are followed by comparatively little hemorrhage, the surgeon uses the écraseur in cases where the use of the knife might produce a loss of blood obstructive to the operator, if not absolutely beyond his control.

On precisely the same principle the surgeon recognises the mischief likely to arise from foreign bodies remaining in the tissues. From time immemorial he has avoided the permanent introduction of such substances into the body, excepting when he seeks to

produce counter-irritation. When necessity has compelled him to pass threads or pins into the tissues for such purposes as the arrest of hemorrhage, he has ever been eager to remove them at the earliest opportunity consistent with safety. Again, one of the chief advantages claimed by the advocates of compression for the cure of aneurysm, and of torsion for the arrest of hemorrhage, is the avoidance of even a temporary presence of ligatures or acupressure needles in the organism.

On the other hand, the immunity from inflammation observed when certain foreign bodies remain long in the tissues, though an assured fact, rests nevertheless on the evidence of comparatively few cases. Hence surgery has for long refrained from profiting by the knowledge of this fact. At length it has so profited, and, strange to say, not in a minor operation, but in one so beset with dangers that it is of paramount importance to consider how every individual element of risk may be neutralised or eliminated.

When the pedicle of an ovarian tumour is too short to allow the use of the clamp, it is now the practice among the most experienced operators to transfix it with several ligatures, to cut short the ends of those ligatures, to return the pedicle within the abdominal cavity, and to close the wound. This practice may conveniently be termed "complete intra-peritoneal ligature," for the sake of brevity, and in contradistinction to the system, now obsolete, of leaving the extremities of the threads uncut and dependent from the external wound.

"Nothing but the large amount of success which has recently followed the adoption of this most unsurgical method appears to me to justify it. Its disadvantages as regards the probabilities of peritonitis and the risk of hemorrhage from the slipping of the ligature (it being part of the plan to cut the peduncle close to the ligature) are evident."[1] These are the words of a well-known hospital surgeon, written within the last fifteen years, and still retained in a popular text-book. Yet complete intra-peritoneal ligature is now justified, not only by the success of the bold earlier ovariotomists, but also by a precise knowledge of the changes produced in the stump of the ligatured pedicle, changes which are now known to consist, not in gangrene or diffused peritonitis, once considered inevitable, but in processes which are harmless from the first, and ultimately beneficial.

These processes I have myself had the opportunity of observing in a case where death occurred a very few days after operation. But before describing the specimen of ligatured pedicle which I examined in this particular instance, it may be interesting to con-

[1] A System of Surgery, edited by T. Holmes, M.A., art., Surgical Diseases of Women.

sider the history of the complete intra-peritoneal ligature as performed by the earlier operators. These surgeons had not the advantage of the scientific sanction which that practice has gained from the now well-known experiments of Spiegelberg and Waldeyer on the effects of the ligature when applied to the cornua of the uterus in animals.[1] Before those investigations were made public the practice must be considered almost, one may say, empirical. Much valuable information on the subject is scattered among the pages of certain special works, particularly the standard productions of Mr. Spencer Wells[2] and Dr. Peaslee.[3] In order, however, to discuss the matter with precision, it is very advisable to refer to the original records of the early ovariotomists.

Dr. M'Dowell, admitted by Mr. Wells to be "the first rational ovariotomist," did not adopt the complete intra-peritoneal ligature. His practice was to leave the ligatures uncut and hanging from the lower end of the incision in the abdominal walls.

But Dr. Nathan Smith of Connecticut, the second American ovariotomist, in his first operation in 1821, not only ligatured two arteries in the omentum with strips of leather from a kid-glove, but also tied two arteries in the pedicle. This is in accordance with principles recognised by the most experienced modern operators; ligature of the pedicle, as a whole, being hazardous, since the single thread is apt to slip. The ends of all the ligatures were cut short, and the external wound closed, the stump of the pedicle having been returned into the abdominal cavity. Dr. Smith, then, was the first to adopt the complete intra-peritoneal ligature. The patient recovered.

In 1829 Dr. David Rogers of New York ligatured separately several large vessels in the pedicle of an ovarian cyst, and returned the stump of the pedicle with the ligatures cut short. The operation was perfectly successful. In 1835 Dr. Billinger adopted the same proceeding with satisfactory results.

Lizars's first *bona fide* ovariotomy was successful, but the ligature encircling the pedicle was "carefully left out" of the external incision. In relating the next case, which ended fatally from peritonitis, he says:[4] "I now gave this enormous mass to my assistant, Mr. Macrae, passed a ligature round the pedicle, and tied it firmly, and then cut close to the tumour, securing three open-mouthed vessels of the pedicle. . . . . I now stitched up the wound, carefully avoiding the intestines and omentum."

The ligatures of the pedicle, or, more strictly speaking, of the

[1] Virchow's Archiv, 1868.
[2] Diseases of the Ovaries, their Diagnosis and Treatment.
[3] Ovarian Tumours, their Pathology, Diagnosis, and Treatment.
[4] Observations on Extraction of Diseased Ovaria, Edinburgh, 1825.

vessels of the pedicle, were evidently not left uncut and dependent from the wound, for the careful Scotch surgeon would surely have recorded the fact had it been so, as in the preceding case. Thus Lizars's second ovariotomy was the first instance of complete intra-peritoneal ligature ever performed in the British Isles. "The peritoneum investing the parietes which adhered to the tumour, and also those portions of this membrane investing the colon and small intestines which adhered to the tumour, were of a bluish-black appearance, and tore with ease under the fingers, being evidently gangrenous." After expressing his belief that the patient should have been bled the night after operation, Lizars remarks: "This probably would not have saved her, *for such contusion was inflicted* that the stamina of life were apparently not capable to stand such a shock and repair the evil." There is, then, clear evidence that death was due to the bruising of the peritoneum, and the fatal result of this operation throws no discredit on the treatment of the pedicle.

Lizars's third operation was incomplete, and throws no light on the subject of this paper, nor need we refer to Dr. Granville's cases. But Jeaffreson of Framlingham in 1836 ligatured an ovarian pedicle as a whole, and cut short the ends of the thread. This case was perfectly successful. The history of Mr. Phillips's ovariotomy, performed in 1840, is very instructive. The stump of the pedicle was tied as a whole, and returned into the abdomen with the ligatures cut short. The patient died on the fourth day. "Upon examining the Fallopian tube, the ligature was found in its place; but it was evident that, from its hypertrophied condition, it resisted the necessary constriction (although Mr. Samwell had used much force), and the extravasation was a consequence of oozing from the extremity of the tube. *That oozing, however, had long ceased, for nature had blocked up the vessels.*" Thus wrote Mr. Phillips in the "London Medical Gazette," October 1840. A few ounces of undecomposed blood were found in the peritoneum. Dr. Peaslee, in quoting this case,[1] appears to infer that death was caused by the loosening of the ligature, or, as Phillips would express it, by the resistance of the pedicle to necessary constriction. But on reading the case itself as recorded by the operator, it will be found that the patient was suffering from choleraic diarrhœa the day before operation, unknown to her medical attendants. In fact, she died with severe choleraic symptoms; moreover, the above quotation shows that the hemorrhage had been slight, and was checked by natural processes.

Thus complete intra-peritoneal ligature was frequently adopted with success, or, at the worst, without being the cause of fatal

[1] *Op. cit.*

results, in these early cases of ovariotomy, when the surgeon acted at an enormous risk, without anæsthetics, and without taking precautions now known to be essential by the light of greater experience. Moreover, such necessary hygienic measures as washing out the peritoneum with antiseptics was not ventured upon. In short, I think it is clear, from the above records, that the practice in question was justified by clinical experience on the human subject before the experiments of the German physiologists.

These experiments showed the changes that actually take place in intra-peritoneal ligature of the stumps of excised portions of the horns of the uterus in bitches. A communication between the distal and proximal parts of the stump is established by inflammatory plastic effusion, and the ligature is unravelled by granulation-cells insinuating themselves between its fibres.

Dr. Tyler Smith appears to have been the first authority who regularly and systematically advocated complete intra-peritoneal ligature. Recently it has been adopted in hundreds of successful cases where the pedicle has been found too short for the clamp to be safely applied. Ligatures of bleeding vessels in the omentum are also cut short. Mr. Spencer Wells informs me that, on one occasion, he left as many as forty ligatures in the abdominal cavity without any evil effects. In the more perilous operations for the removal of solid growths of the uterus, complete intra-peritoneal ligature may also be practised with impunity; this is proved by Mr. Knowsley Thornton's case, recorded in the "Medical Times and Gazette," April 1877. That gentleman strongly advocates the use of the silk ligature, even in cases where the clamp has hitherto been thought advisable.

An important section of contemporary medical literature furnishes us with a strong proof that complete intra-peritoneal ligature of the ovarian pedicle is firmly established. The student finds it advocated in those educational works on the theory and practice of his profession which he studies when preparing for examination, and retains, or ought to retain, in his library for consultation and reference in after-life.

Mr. Erichsen approves of this mode of ligature, at the same time epitomising its history as follows:[1]—

"Some of the earlier American ovariotomists, especially D. L. Rogers, cut the ligatures short, and returned the stump of the pedicle into the wound. This practice was revived by Tyler Smith in 1861, and has been adopted by him, by T. Bryant, and others, with the happiest results, the ligatures either becoming en-

[1] Science and Art of Surgery, 6th ed., 1872. In the seventh edition Mr. Erichsen expresses a strong preference for the clamp, but retains the remarks above quoted, excepting the last sentence.

capsuled or being discharged after a time through a suppurating track. It appears to me that if the ligature be used, this is the best method to be adopted; and if practised with carbolised ligatures, it would probably be most successful."

Mr. Holmes states, still more decidedly: "When the clamp cannot be fixed on the pedicle of the tumour, on account of its proximity to the uterus, without injudicious traction on that organ, the best plan is to perforate the pedicle with a needle threaded with stout wire, and tie it in halves, the ends of the ligature having been flattened down so as not to irritate the neighbouring parts, and after cutting away the tumour down to within about half an inch from the ligature, drop the pedicle back into the belly. In a case treated successfully in this way, I searched some time afterwards carefully for the wire by palpation from the abdominal wall and from the vagina, but could elicit no sensation of its presence."[1]

In the second edition of his "Practice of Surgery," Mr. Bryant makes some interesting observations on the history and on the advisability of complete intra-peritoneal ligature. He describes the pathological appearances noted in the pedicle in one case of his own, and in another recorded by Dr. Peaslee in a Transatlantic medical serial. A reference to Mr. Bryant's work will show that the intervals between the operation and the death of the patients in these two cases are similar to those to which I am about to refer. This surgeon concludes his disquisition on the subject by asserting that "with short and broad pedicles, in which the vessels are usually small, the cautery may be employed, or the pedicle ligatured in two or more parts with whip-cord, the ends of the ligatures cut off and dropped in, and the wound afterwards closed."

The precise pathological changes produced in the ovarian pedicle by complete intra-peritoneal ligature have been well displayed in the two following cases, one of which has been under my own observation, and has not hitherto been recorded.

In 1872 Dr. Bantock exhibited before the Obstetrical Society the stump of an ovarian pedicle from a patient who died of cancer one year after double ovariotomy had been performed upon her. The hempen ligature applied, with its ends cut short, to one of the pedicles, was found on dissection to have been completely absorbed excepting its knot, which remained as a hard body the size of a hemp-seed, covered by peritoneum. The bulging of the tissues over each side of the groove formed by the ligature had brought the strangulated portion of the stump at once into close contact with the unstrangulated proximal part. Through the slight irritation produced at first by the pressure of the ligature,

[1] A Treatise on Surgery, its Principles and Practice, 1875.

the proximal part had thrown out plastic lymph, which had conveyed nutritive plasma and also capillaries to the distal portion of the stump, and thus saved it from gangrene. In a case like this, the stump ultimately atrophies, for reasons evident to any surgeon with a superficial knowledge of pathology. As for the ligature, it is destroyed in the manner demonstrated by the experiments of Spiegelberg and Waldeyer.

In the summer of this year I made a post-mortem examination of a patient, aged thirty-seven, who died in the Samaritan Hospital from septicæmia, on the sixth day after the removal of a large multilocular ovarian cyst. The pedicle had been treated by complete intra-peritoneal ligature, and there was no evidence that death was in any way due to this method of treatment.

The stump of the pedicle was an inch broad, and its inner border was about a quarter of an inch from the fundus of the uterus. It was not in a sloughy condition, nor was it even congested. It was separated from the appendages of the uterus by four silk ligatures, none of which had produced ulceration, but all were covered with bands of lymph bridging over the constriction they had produced in the tissues which they encircled. A more interesting feature remains to be noticed. The outer extremity of the distal side of the pedicle was already very firmly united to the broad ligament by well-organised lymph. This plastic effusion must have been exuded some days before death, prior to the onset of septicæmia, and therefore very shortly after the operation. The lymph covering the ligatures was evidently more recent; it also showed signs of breaking down, its plasticity being diminished by the constitutional disorder which had proved fatal to the patient.

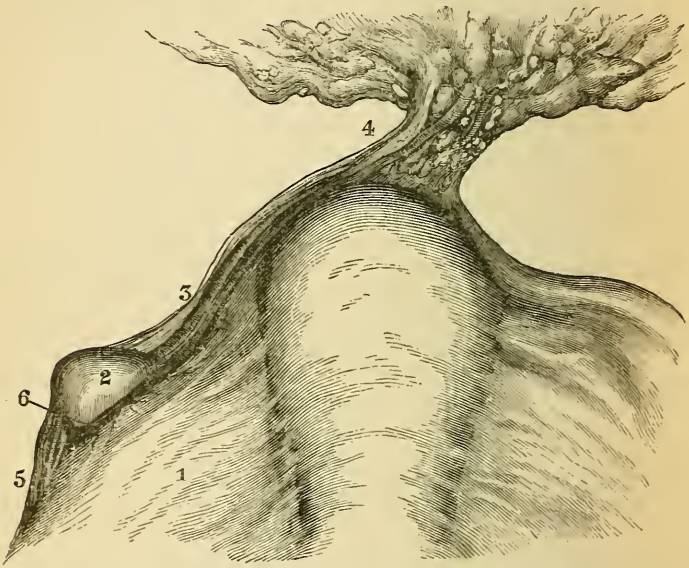
This early and intimate adhesion of the distal part of the stump of the pedicle to the broad ligament is represented in the appended lithograph, for which I am indebted to Mr. C. Berjeau.[1] It might be suggested that the adhesion existed before operation, the ligature being passed under it. Such, however, was not the case, as I carefully observed the pedicle and the process of ligature when Dr. Bantock removed the ovarian tumour. The specimen may be seen in the Museum of the Royal College of Surgeons, Pathological Series, No. 1642 C.

Thus the surgeon need no longer dread any evil effects when he thinks it desirable to leave ligatures enclosed in the abdominal cavity after a serious and complicated operation. Hence small foreign bodies do not necessarily produce disastrous consequences, even when impacted in a wounded structure in the neighbourhood

[1] In the engraving, an asterisk points to the adhesion.

of delicate organs irritated by disease, and by unavoidable surgical manipulations.

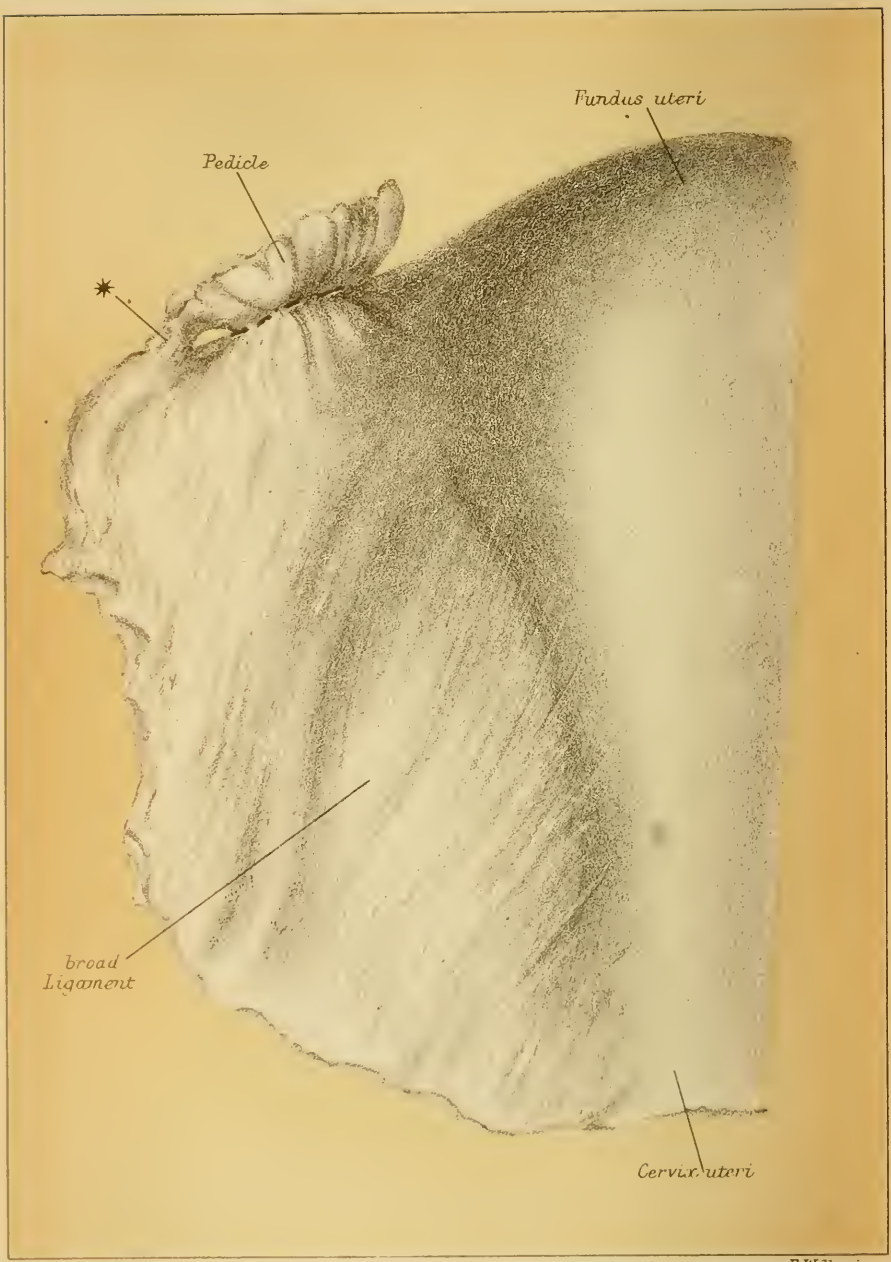
From the success of the complete intra-peritoneal method of ligature in ovariotomy we may deduce the important corollary, that necessity may justify the setting aside of surgical principles previously considered as laws never to be violated with impunity. Such deviation from precedent may yet be applied with advantage to other operations.



1. Broad ligament. 2. Stump of pedicle. 3. Adhesion between pedicle and 4. Portion of omentum adherent to fundus uteri. 5. Adhesion to broad ligament. 6. Line of ligature.

Note.—Since sending the above to the press, Dr. Bantock has drawn my attention to another specimen, showing the effects of complete intra-peritoneal ligature on an ovarian pedicle. Hempen thread had been employed, on the theory that that material shrinks when moistened, and thus tightens its hold on any structure around which it may be tied.

This specimen is from a girl eighteen years of age. On 5th April 1876, Dr. Bantock removed a large multilocular tumour, weighing seven pounds, from her left ovary. The patient made a good recovery, but in the following October the right ovary became the seat of a sarcoma. She was readmitted into the Samaritan Hospital, where she had undergone the first operation, and the abdomi-



C. Berjeau, lith. E. Weller, imp.

Complete Intra-peritoneal Ligature.

Stump of Pedicle of Ovarian Tumour,
Sixth day after operation.

nal cavity was opened, but on account of the character of the tumour it was not deemed advisable to remove it. The patient died on November 3, 1876. The annexed woodcut shows the appearance of the stump of the pedicle of the left ovary seven months after ligature. Towards the middle line it is connected by a long, thin, and very vascular adhesion to a portion of omentum, which has become adherent to the fundus uteri. Externally another vascular band has formed between its outer border and the broad ligament. This outer band is homologous to that in the specimen figured in the lithograph. The free upper edge of the stump has curled in and formed an adhesion to its anterior surface, so that the whole has made itself, so to speak, into a cyst. A deep groove marks the position of the hempen ligature, not a trace of which remains.

MEDICAL CASES.

BY

SAMUEL WEST, M.B.

A.—Three Cases of Fever with Diarrhœa—In Two Ulceration in large Intestine, with Abscess in the Liver.

Case I.

Edwin E., aged 18, packer, was admitted under the care of Dr. Andrew on May 18, with the following history:—

He had never been strong; had been troubled with cough, worse in winter, for many years, with slight expectoration and night sweats, but had never spat blood; had had two attacks of measles and one of scarlet fever, from which he had recovered completely.

On March 23 was seized with shivering and frontal headache. His head was very hot; he had a cough and night sweats; and from this time he has continually lost flesh. He was indoors for fourteen days, and then was well enough to go to work, though the pains in the head and the cough continued.

On May 12 he was taken with diarrhœa, B. O. five or six times a day; this has continued till now; has noticed no blood in the stools. Has no pain; is slightly delirious at night.

Present condition:—Patient with flushed cheeks; very thin and weak. Sordes on lips. Tongue moist, slightly furred, and slight central streak. No physical signs in chest in front; back not examined. Spleen not enlarged. Abdomen not distended. Well-marked gurgling in right iliac fossa. No spots. Urine, 1013; slight trace of albumen. Appetite very bad. Is very thirsty. P. 120; dichrotous. T. 104·2°.

10 P.M.—T. 104·3°. Passed three motions in bed.

May 20.—Motions, eight or nine during twenty-four hours; grass-green, like chopped spinach; copious, fluid. T. 103·8°. P. 146. Very delirious during night. One small spot on right side of

epigastrium, which is not characteristic at all. Slight improvement of resonance at left apex. No alteration in urine. En. amyli c̄. opio repeated at intervals.

10·30 P.M.—T. 104·8°. P. 160. R. 56.

May 21.—Fidgety and delirious during night. T. 103·4°. P. 144. R. 52. Motions of same character passed in bed. Dusky flush over face. Sordes on lips. Slight gurgling in right iliac fossa. Spot of yesterday more prominent, but less red. Slight subsultus. Urine contains a trace of albumen.

3 P.M.—Bath, tepid, at 90° F., reduced to 78° F. Before bath T. 105·8°. P. 144. R. 52. In bath fifteen minutes; complained of feeling cold. Five minutes after bath, T. 100·0°. P. 120. R. 48.

6 P.M.—T. 103·4°.

10 P.M.—T. 104·0°. Has been more comfortable, and has slept well. P. 132.

May 22.—Slept well. No delirium. Motions, nine in twenty-four hours; same in character. No pain in abdomen, but lies with knees drawn up. T. 103·4°. P. 120, R. 36. 10 P.M.—T. 102·8°.

May 23.—Dusky complexion. Drowsy and stupid. Motions, eight in twenty-four hours. Not quite so green. A good deal of general bronchitis. T. 102·4.° P. 144.

10 P.M.—Muttering. T. 103·4.° P. 144. R. 44.

May 24.—Motions, seven; not so green; mixed with whitish flakes, probably milk. T. 102·4.° P. 140. R. 44. At 5 P.M., T. 103·8°

May 25.—Motions passed continually in bed for last twelve hours; of same character. En. am. c̄. op. repeated. T. 104·3°. P. 106. R. 36. Bath at 89° F. In bath for five minutes, when he began to shiver.

2·30 P.M.—T. 103·4°. P. 128. R. 36.

6 P.M.—T. 104·1°. Wet pack. At 9 P.M., T. 103·0°. At midnight, T. 103·4°. Motions less frequent.

May 26.—Restless night. Motions, twelve in twenty-four hours; less green, more yellow and powdery. T. 101·8°. P. 120. R. 30.

10 P.M.—T. 102·8°. P. 124. R. 32.

May 27.—Motions, seven. Patient seems brighter. T. 101·8°. P. 140. R. 32.

11 P.M.—T. 101·4°. P. 112. R. 34.

May 28.—Motions, eleven; less green, more solid. T. 101·2°. P. 120. R. 36.

11 P.M.—T. 102°. P. 120.

May 29.—Motions, eight. T. 101·5°. P. 120.

Midnight.—T. 102·0°. P. 120. R. 30.
May 30.—Motions, six. T. 101·6°.
Midnight.—T. 101·4°.
May 31.—Motions, eleven; still very green. T. 99·0°. P. 96. R. 30.
Midnight.—T. 101·4°. P. 108. R. 30.
June 1.—Motions, five. T. 101·8°.
11 P.M.—T. 100·4.
June 2.—Motions, four; less green. T. 100·8°. E. 101·2°.
June 3.—Motions, four. T. 99·2°.
10 P.M.—T. 100·8°.
June 4.—Motions, three. T. 101·2°. E. 99·8°.
June 5.—Motions, three. T. 100°. Great pain in left wrist and knee; worse on movement; hot; not swollen. E. 99·2°.
June 6.—Motions, two. T. 98·3°. E. 98·4°.
26th day, June 7.—Motions, three; becoming brown. T. 100·8°. E. 97·0.
June 8.—Motions, three; greyish, liquid, powdery. T. 98·6°. E. 98·0°.
June 9.—Motions, four.
June 11.—Motions, three.
June 13.—Bowels not open.
June 14.—Bowels open once; solid, well formed.

From this time the bowels became regular, the patient having generally only one motion in the day, solid and natural. The temperature never rose again, and in July he was sent up to Highgate, where he made a complete recovery.

This case, if it had stood alone, would, in spite of the absence of typical spots and the peculiar characters of the stools, have been regarded as a case of typhoid fever; but the following cases, which were under observation in the Hospital about the same time, place it in a different light.

Case II.

James S., aged 27, admitted into the Hospital under the care of Dr. Andrew on June 18, complaining of pain in the abdomen and diarrhœa, which had lasted for a fortnight. For some days he had felt ill, but he had had no shivering or sickness from which his illness could be dated. The patient was very weak and looked ill. There was a general indistinct mottling over the whole skin. The abdomen was distended, tympanitic, and tender. T. 102·8°. P. 130; dichrotous, slightly irregular. R. 54.

June 19.—Has slept hardly more than one hour during [the

night. Is very weak and feeble, so that complete examination of the chest is impossible. Alæ nasi dilating. Patient complains of pain on right side, where there are found the physical signs of pleuro-pneumonia of that base. Apex of heart half inch outside left nipple. The mottling has disappeared. No spots. Tongue moist and creamy. B. O. four times since admission; copious, liquid, powdery. T. 101·8°. P. 132. R. 60.

Evening.—T. 102·6°. P. 132. R. 54. Cough troublesome; sputa bronchitic.

June 20.—Slept fairly. Slight increase of dulness upwards on right side. Physical signs still those of pneumonia. Slight dulness at left base in front. Abdomen more tympanitic. No pain. Motions, nine; golden-yellow, liquid, and powdery. T. 102·2°. P. 120; dichrotous. R. 50.

Evening.—En. amyli c. opio. T. 101·8°. P. 144. R. 56.

June 21.—Slept fairly. No delirium. Abdomen more distended, tympanitic. Tongue inclined to be dry; red central streak; fur at sides. Motions, ten; golden-yellow, two with greenish matter in them. Slight crepitation at left base. T. 100·8°. P. 132. R. 70. Given bismuth and catechu.

Evening.—T. 100·0°. P. 120. R. 60. Sick once.

June 22.—Slept fairly. Coughs but little. Expectoration scanty. Motions, thirteen; golden-yellow, one slightly bloody. T. 99·4°. P. 132; much weaker. R. 60.

Evening.—T. 101·2°. P. 140. R. 60.

June 23.—More restless; talking in sleep; lies on right side. Crepitation marked at left base in front. Motions, nine; same in character. T. 100·4. P. 144. R. 60.

Evening.—T. 100·8°. P. 132. R. 60.

June 24.—Sick once. Abdomen less tympanitic. Crepitation more abundant on left side; dulness less on right, Tongue moist. Motions, thirteen; copious, flaky, greenish. T. 98·4°. P. 144. R. 60.

Evening.—T. 99·8. P. 132. R. 48.

June 25.—Slept fairly. Tongue slightly dry. Breathing more laboured. Abdomen as before. No ascites. Cough and expectoration slight. Dulness increased upwards on left side, with occasional crepitation and bronchial breathing. On the right side, coarse respiratory sounds, but less dulness. Heart sounds very feeble and hurried, occasionally irregular both in force and frequency. Motions, nine; copious, one very green, like solution of sulphate of copper; in two are clots of blood; perhaps in all nearly half a pint of blood. T. 102°. P. 144. R. 60.

Evening.—T. 101·2°. P. 132. R. 60.

June 26.—Abdomen more tympanitic. Pain in supra-pubic

region. Motions, nine; green, flaky; blood with two, but not much. T. 99·8°. P. 144. R. 72.

Evening.—T. 102·5°. P. 144. R. 60.

June 27.—Tongue much less coated. Chest signs as before. No spots. Motions, eleven; the last four very green, like solution of sulphate of copper; no more blood. T. 102·8°. P. 160. R. 54.

Evening.—T. 100·0° (?). P. 136. R. 44.

June 28.—Motions, eight; still green. This colour becomes more marked after the motion has been exposed to the air for some time; at first the colour is a deep yellow. T. 100·3°. P. 148. R. 50.

Evening.—Sweating profusely. T. not raised. P. 140. R. 52.

June 29.—Motions, nine; same in character but less copious. T. 99·5°. P. 140. R. 56.

Evening.—P. 144. R. 48.

June 30.—Motions, ten; less green and less copious. Abundant crepitation at right base in front, but less dulness. Dulness at left base has almost disappeared. T. 99·5°. P. 150. R. 60.

Evening.—T. 98·8°. P. 144. R. 48.

July 1.—Motions, five; the last more solid. T. 97·8°. P. 132. R. 50.

Evening.—T. 98·8°. P. 144. R. 54.

July 2.—Restless during the night. Motions, six; same in character. Right foot and leg œdematous; slight enlargement in course of femoral vein. Abdomen tympanitic. Round swelling fluctuating in epigastrium, 3 inches in diameter, 2½ inches above umbilicus; dull on percussion; dulness continuous with liver dulness, and painful on pressure. Liver seems to extend as low as umbilicus. Veins enlarged on lower part of sternum, especially on the right side. Lips and tongue tremulous. Face pale and drawn. Heart's apex nearly two inches outside left nipple. T. 97·8°. P. 144. R. 60.

2 P.M.—Abscess tapped with aspirator; about 4 oz. of very viscid, tenacious, chocolate-pink fluid (micros.: granular detritus, fatty cells, liver cells (?), pus and blood cells).

11 P.M.—Has not slept. Has perspired a great deal; cold clammy sweat. T. below normal. P. 144. R. 50.

Slept fairly during the night until early morning, when he woke up; appeared to faint, and died suddenly.

Post-mortem twelve hours after death.—Body emaciated. Abdomen distended. Diaphragm reached middle of third intercostal space on right side and of fourth on left. A small amount of serous fluid in the pericardium. Heart healthy.

Lungs: both bases collapsed and congested, yielding on section a serous fluid devoid of air. In other respects healthy. Right base adherent to the diaphragm.

The peritoneal cavity contained a good deal of serous fluid, but there was no recent peritonitis.

Small intestines slightly congested and œdematous, but otherwise natural. The large intestines were œdematous, with enlarged and swollen follicles scattered in moderate abundance, some of which had broken down to form round ulcers, for the most part as large as a pea. The mucous membrane between them was œdematous, but not otherwise altered. In the cæcum and in the rectum the ulcers were much larger, irregular in shape, for the most part oval transversely, with red hyperæmic borders and prominent margins. The mucous membrane hung in shreds from the edges, and floated out on pouring a stream of water over them, and exposed the muscularis laid bare beneath. There was no trace of peritoneal inflammation anywhere over these ulcers.

Some small ulcers were also found in the vermiform appendix.

The contents of the intestines, small as well as large, were grass-green in colour. The liver was very considerably enlarged; upwards to the third intercostal space on the right side and to the fourth on the left, downwards to the level of the umbilicus. Slight adhesions, easily broken down with the fingers, between it and the peritoneum and the anterior part of the diaphragm. Posteriorly and on the right side the diaphragm and capsule of the liver were so closely adherent that they had to be removed together. Another dense adhesion was found between the middle of the left lobe and the abdominal walls, through this the puncture of a trocar was traced into the cavity of an abscess the size of a small apple (1½ inch in diameter).

In the posterior and superior part of the right lobe was a large fluctuating swelling, from which about a pint and a half of slimy, greenish-yellow, opaque, acid-smelling, purulent fluid escaped on section.

The upper wall of the abscess cavity was formed by the diaphragm; the outer walls by liver substance which hung in arborescent shreds into the cavity.

Two other abscesses the same in character were found deep in the same lobe. These were about 1½ inch in diameter, while others much smaller in size (a pin's head to a cherry) were fairly numerous.

In the left lobe two others were discovered, one the size of a walnut; the other that described above, into which the trocar puncture led.

The lobulus Spigelii contained one also as large as a walnut.

These abscesses one and all possessed the characters given above, viz., the irregular walls and viscid contents. The liver tissue round them was in a state of active inflammation and purulent infiltration.

Gall-bladder empty and contracted.

A large adenoma verum lay imbedded in the centre of the liver (irregularly circular, about two inches in diameter), in the right lobe, close to the longitudinal fissure.

A large ante-mortem clot, in process of softening, lay in the right branch of the portal vein. It was not adherent to the vessel, nor were the coats of the vessel inflamed.

Both femoral veins, and the vena cava as far as the diaphragm, were filled with a softening ante-mortem clot, but the heart contained no ante-mortem clots. Nowhere were the clots adherent, or the coats of the vessel inflamed.

Slight œdema of both feet, but not extending to the legs.

Kidneys, slight parenchymatous inflammation. Spleen slightly enlarged and congested.

Case III.

For the notes of this case I am indebted to the kindness of Dr. Abercrombie.

Elizabeth S., aged 18, admitted into Hope Ward, under the care of Dr. Black, 21st May 1877.

Taken ill suddenly twelve days ago, with vomiting and pain in abdomen.

Has remained in bed ever since.

Constipation more or less till yesterday, then diarrhœa began.

Bowels open more than every half-hour during the night. No blood in motions.

Has had a little cough since she has been ill.

No one else ill in the house.

On admission, face pallid; no expression of pain.

Tongue pale, moist, thickly furred down the centre.

Can lie in any position.

P. 128; small, reg. R. 40. T. 100·5°.

One suspicious spot over sternum, but not typical of typhoid.

No physical signs of disease in chest.

Abdomen slightly distended, tympanitic. No spots seen. Tender all over, especially in left hypochondrium and right iliac fossa.

Spleen not felt. Retched this morning, but did not vomit. Urine no albumen.

Bowels open once; motion said to be slimy.

3·45 P.M.—Bowels open three times since; one motion was pale and loose, another bright yellow and semi-solid. P. 128. T. 100·2°.

10·15 P.M.—Nine motions since last note; not seen; no blood. A good deal of pain when the bowels act. P. 128. R. 48. T. 100·8°. Hst. cretæ arom. c. tr. opii, m. x. p. r. n.

May 22.—Rather a restless night; dull flush on cheeks; listless expression. Bowels open twice; both motions rather copious; quite loose; bright yellow-ochre. P. 124. R. 48. T. 100·9°.

2 P.M.—Breathing thoracic. Abdomen fixed; no spots. B. O. 3; motions as before. P. 120. T. 102·8°.

10·30 P.M.—Sub-delirium. B. O. 5; motions scanty; no blood. P. 120. R. 40. T. 101·2°.

May 23.—Slept a little. B. O. 4; motions pale; no blood. Abdomen less tense; no spots. P. 120. R. 36. T. 100·0°.

10·15 P.M.—A very violent shooting pain came on in abdomen about two hours ago and persists. B. O. 5; no blood. P. 120. R. 40. T. 100·2°.

May 24.—Slept well. B. O. 3; motions loose, powdery yellow-ochre. Still has the pain in abdomen, just to left of umbilicus; it is unaffected by pressure. No spots on chest or abdomen. P. 120. R. 42. T. 102·2°.

2·45 P.M.—B. O. 1. P. 128. T. 102·7°. No spots on chest, abdomen, back, or arms.

11 P.M.—Much pain in abdomen. B. O. 5; motions quite loose, light yellow. P. 126. R. 36. T. 101·6°.

May 25.—B. O. 3. P. 128. T. 101·4°.

10·30 P.M.—B. O. 4. P. 120. T. 101·2°.

May 26.—B. O. 3. Abdomen less distended; no spots. Bronchial breathing and crepitant râles over right root and right posterior base.

3 P.M.—B. O. 1; motion light, partly formed. P. 120. T. 102·4°.

May 27.—B. O. 4. P. 112. T. 100·8°.

10·45 P.M.—B. O. 5. P. 120. T. 100·8°.

May 28.—B. O. 3; motions loose, light brown. P. 120. T. 100°0.

Right back dull; bronchial breathing and râles. E. B. O. 4. P. 100. T. 99·2°.

May 29.—B. O. 2. P. 120. T. 100·6.° Abundant sudamina over abdomen. Dulness over right back, breathing hard, and bronchophony.

May 30.—B. O. 5. P. 128. T. n.

10 P.M.—B. O. 2; motions light, part formed. P. 126. T. 100·0°

May 31.—B. O. 2. P. 116. T. 101·8°.

During the month of June there was but little change; constant elevation of temperature (from 100° to 103·5°), with increased frequency of pulse and persistent diarrhœa; from three to five loose motions a day. The chest symptoms cleared up, but very slowly, so that at the end of this month dulness was noted with abundant moist râles on the right side.

On July 7th it was noted that there was a tender spot in the right iliac fossa, near the crest of the ilium.

Towards the end of this month she was able to sit up for a little while, but the high temperature, diarrhœa, and localised abdominal tenderness persisted without much alteration.

In the first week in August her temperature went down to normal, and continued so for about ten days, during which time she was able to get down into the square every day. The diarrhœa continued.

On August 19th her appetite failed, and her temperature was considerably raised. From this date she kept her bed, and until the end of the month her temperature continued above 102°; her pulse was very frequent, and she passed about three loose motions a day, always light coloured and powdery.

September 4.—B. O. several times; motions loose. P. 108. T. n. Chest quite natural, abdomen slightly tense and tender. Urine no albumen.

For the next three days her temperature remained normal.

On September 8th, in the afternoon, after a copious action of the bowels, she was seized with a sudden pain in the abdomen, followed by great collapse, from which she never thoroughly rallied, and died on the following day.

On examination of the body after death, the small intestine was found natural throughout, Peyer's patches being quite natural. In the cæcum there was a small oval ulcer, half an inch in length, its base being formed by thickened peritoneum, and having smooth, non-everted edges. The small intestine was adherent to the cæcum at this point, and here there was a small localised abscess in the peritoneum.

About the middle of the descending colon there was another similar ulcer of about the same size, and here too there was a localised abscess, with adhesion of the small intestine to the large.

In neither of the ulcers had perforation taken place.

The rest of the large intestine was natural.

The mesenteric glands were not enlarged.

There was slight recent general peritonitis.

On the upper surface of the left lobe of the liver, and separating this from the diaphragm, was a localised abscess, which communicated with one in the left lobe of the liver, about the size of a walnut.

In the right lobe of the liver there was another abscess a little larger. No thrombi were discovered in any of the branches of the portal vein.

There was a little turbid fluid in both pleuræ.

Other viscera natural.

Briefly summed up, the history of these three cases is as follows:—

Case I.

Extreme fever. Extreme diarrhœa. Grass-green motions, which never contained blood. No pain in hepatic region. Pneumonia and obstinate bronchitis.

Case II.

Moderate fever. Profuse diarrhœa. Grass-green motions, some containing blood. Pneumonia. Abscess in the liver. Ulcers throughout the large intestine, most extensive in cæcum and rectum.

Case III.

Moderate fever. Diarrhœa obstinate, and in commencement of disease profuse. Motions not grass-green, containing no blood at any time. Pneumonia. Abscess in the liver. Two small ulcers in the colon.

In the first two cases the disease ran an acute course. In the first a crisis occurred on the twenty-sixth day. In the second, the patient died in the fourth week. The last case became chronic, and was terminated by death in the middle of the fourth month.

They all agreed in the absence of enlargement of the spleen and of characteristic typhoid spots, and in the fact that none of the patients had ever lived out of London, and that there was no history of dysentery.

In spite of the strong clinical resemblance which these cases bear in many respects to typhoid fever, the pathological changes which post-mortem examination of the last two cases disclosed conclusively prove that they cannot be classed under this head. Not only were the characteristic changes of typhoid fever

absent, but others rarely, if ever, found in connection with it, were present. Peyer's patches, and indeed the whole small intestine, showed in neither case any evidence of past or present disease, while in the large intestine ulcerations of a peculiar nature existed, and with these numerous abscesses in the liver.

The ulcers in Case II. were large, extensive, and almost diphtheritic in character, while in Case III. they were very small, and bore no relation whatever to the severity of the symptoms during life; but in neither case did the intestines present the characters of dysentery—an opinion which was confirmed by Mr. Prentis, whose long residence in India has given him ample opportunities of observing dysentery of all kinds and in all stages. He kindly allows me to state that the condition of the intestines was unlike any that he ever found in cases of dysentery, and this opinion the clinical symptoms support even more strongly than the pathological appearances. Dysentery then must be excluded; but that the disease is of some specific nature, both the course the cases ran and their pathology renders probable, but it is not clear what this specific nature is. If typhoid, restricted to the large intestine, be suggested, it may be urged, that in all the statistics given of the disease only one case is placed on record in which the changes were limited to the large intestine, and even this seems to be somewhat doubtful; and further that abscess in the liver as a consequence of typhoid is almost unknown.

Nor can the abscesses in the liver and the changes in the intestines be regarded as accidental coincidences. The strong clinical resemblance of the cases to one another forbids this; nay, rather suggests a close specific relation between the two. So that we are justified in placing together such cases as these in a group by themselves, and separating them from the class of typhoid fever to which they approximate clinically, but from which they are evidently to be distinguished.

B.—Two Cases of Pneumothorax.

Case I.

(*Access marked by severe attack of pain.*)

Isabel D., aged 15, admitted under Dr. Andrew on May 25, complaining of constant uncontrollable sickness. She had had a similar attack at the same time last year, which lasted for six weeks, from which she recovered completely. She was said to have had three attacks of slight hæmoptysis on the last two days before admission. No history of hæmaturia or fits. Catamenia have never commenced.

Patient was much emaciated; lay on the right side with her knees drawn up because of pain in the right inguinal region. Tongue dry, central streak. No diarrhœa. No headache. Urine natural. T. 98·0°.

Vomiting several times during the day, attended by great pain in epigastrium.

At both apices the respiration was slightly coarse, especially on right side, and here the voc. vibr. were increased somewhat, but no crepitation was audible.

The fundus of the eye was normal.

There was no evidence of gastric disease, so that the vomiting was evidently reflex, but its cause was obscure. There was no evidence of cerebral mischief, none of abdominal affections, none of disease of the genito-urinary tract, and the slight symptoms at the apices which she was thought to have on admission cleared up so that it seemed too much to attribute all her trouble to tubercle.

The sickness continued in spite of treatment, and the patient had to be fed by enemata, which she was able to retain. These symptoms continued from May 26 to July 4, without abatement or change in her general condition. Early on the morning of the 4th, she woke up shrieking with pain in the left side. Her face was pale and pinched. P. 140, weak. R. 36. Her heart was beating under the right nipple, being evidently displaced by pneumothorax of the left side. The chest was tapped with a fine canula, and air escaped, but in no great quantity.

The rest of the case is simply told. The patient grew gradually weaker, but the chest was not again tapped, as there was no dyspnœa, and her great weakness seemed to contra-indicate operative interference. Her temperature never rose, though the pulse was always very rapid, 140, 150, and the respirations on the average about 40.

She lingered on for some days, and finally died exhausted on May 14th.

Post-mortem.—Body much wasted. The chest, abdomen, thighs, and insteps covered with confluent purpuric spots; on the chest and abdomen they form one large hemorrhage.

The membranes of the brain quite natural; as also the lateral ventricles and central ganglia. On the right side of the floor of the fourth ventricle is a tumour the size of a pea, softened in the middle, and containing fluid like pus (a tubercle).

The left side of the chest contains air and pus. The left lung is collapsed and bound down by recent adhesions to the back of the chest. At the lower part of the upper lobe is an opening

through the pleura into a cavity. The upper lobe is broken down into an irregular cavity. The rest of the lung shows masses of caseous matter, some solid, others softening. The right pleura is a little roughened. At the apex of the right lung is a large cheesy mass, the size of a walnut. Rest of the lung free. Bronchial glands cheesy. Heart small but natural.

Spleen large. Liver, kidneys, and aorta natural. Stomach excessively contracted, but no disease.

Intestine natural, but Peyer's patches show extensive follicular ulceration; the last before the illocæcal valve has an ulcer in it as large as a pea.

The chief feature of interest in this case was the uncontrollable sickness. The occurrence of pneumothorax was regarded at the time as sufficient evidence of its cause, viz., tubercular phthisis; and this, with the absence of any other nerve symptom, was thought to be conclusive against its cerebral origin; but nevertheless, post-mortem, a tumour was found in the medulla, so that the sickness was after all possibly cerebral.

Case II.

(*The only symptom slowly increasing dyspnœa.*)

William B., aged 41, gave this history. He had been quite well until four months before admission, when he caught cold and was attacked by a bad cough, brought on by "hard living." Loss of flesh, night sweats. No hæmoptysis. No dyspnœa. He had never had any sudden attack of dyspnœa or cough; but on examination he was found to have pneumothorax of the left side.

His temperature was high, at night usually 103°, with a morning fall. His respirations were 36 on admission, but two days later dyspnœa intervened, and the distress became so extreme, that the side was punctured with a fine trocar and canula, and a large quantity of air escaped with a small quantity of sweet pus. This was followed by great relief, and the temperature fell to 99·7°. For two days he was better, although the pulse and respirations were rapid, 140 and 44, and the temperature rose again to 102·0°, but on the third day he died quite suddenly after sitting up to eat his breakfast.

Post-mortem.—Some chronic mischief was found in both lungs with small cavities, one small one had communicated with the pleural cavity near the root of the left lung. The left pleura was much thickened, contained air and about forty ounces of pus. The lung was collapsed. At the back of the left lung, a little above and close to the root, was a small opening as large as a split pea, leading into a cavity of the size of a hazel-nut. The

rest of the upper lobe was riddled with cavities; none in the lower lobe, which was collapsed.

In the right lung, which was generally emphysematous, were numerous solid masses of varying size, from walnuts to peas. They were grey, granular, and on section solid, and in places breaking down to form cavities.

Small tubercular ulcers in lower part of small intestine. Kidney small; capsules adherent; cortex reduced, somewhat granular.

Other organs healthy.

C.—Tubercular Meningitis.

In two cases of tubercular meningitis, the pupils were found to be constantly dilating and contracting, so that in the short space of a minute or two the pupil would pass rapidly from a condition of extreme dilatation to one of extreme contraction, and *vice versâ*. In both cases the patients were completely unconscious, and this peculiarity lasted for many days before their death.

The diagnosis was in both cases confirmed by post-mortem examination.

D.—Two Cases of Anæmia, with Microscopical Changes in the Blood, as Illustrating the Pathology of Pernicious Anæmia.

Case I.

In a case of severe jaundice, probably depending on gall-stones, the patient being in a state of the most extreme anæmia, and so weak that the least exertion caused fainting, the blood, on being examined, presented the characters described as pathognomonic of idiopathic anæmia.

The cells were considerably diminished in number, and the blood very watery. The red cells did not form rouleaux, were much diminished in size, hardly half as large as a healthy red cell, most irregular in shape, and deeply coloured, resembling the bodies described by Lebert as tubercle corpuscles. The white cells were in relative excess. In this one particular only did the blood in the case differ from that described in idiopathic or pernicious anæmia. The patient, however, recovered, and with his improvement his blood gradually returned to its normal condition; and on his leaving the Hospital this note is recorded:—

Red blood cells nearly natural again; here and there one still misshapen. They form rouleaux; the white are no longer in excess.

Case II.

In a case of extreme anæmia, of which the cause could not be found, and in which post-mortem no lesion was discovered beyond fatty degeneration of heart, liver, and kidneys, all probably secondary to the anæmia, the blood was found during life profoundly altered. In addition to the hydræmia, which was considerable, the red cells were much increased in volume, half as large again. They were swollen, had lost their disc-like form, and become pear-shaped. The colouring matter had separated from the general mass of the cell, and was collected into a round strongly refracting globule in one corner of the cell. In some cases it had escaped, and a pale, greyish, slightly granular cell-body remained. The white cells were, so far as could be judged, in their proper proportion to the red.

Clinically this was a case of pernicious anæmia, and these two cases together tend to show that the changes of the blood described as pathognomonic of pernicious anæmia are not really so distinctive as authors assert.

HYPERTROPHIC CIRRHOSIS:

A CASE, WITH REMARKS.

BY

SAMUEL WEST, M.B.

I propose to give an account in this paper of a case which presented many of the features during life of acute yellow atrophy, but which proved on post-mortem examination to be one of hypertrophic cirrhosis. I shall endeavour briefly to describe this disease, and to discuss the chief points of interest which it presents. For the notes of this case and for the account of the post-mortem I have to express my thanks to Dr. Ormerod.

Frederick C., aged 2 years, was admitted into the Hospital on 3d April.

The following history was obtained:—The patient was in the Hospital about four months ago for a severe scald occasioned by his rolling a kettle of boiling water upon himself. Extract from the Surgical Registrar's note:—"November 29, 1876. Scald of whole of abdomen, of right hand and forearm, of right thigh and leg, chiefly of the first and second degree (erythema and vesication). Constitutional symptoms slight. Sleeps well, breathes well, &c. Carron oil, cotton wool.

"December 5.—The temperature, which was 98·4° on December 1st, has since then been gradually rising. To-day it is 104·0°. Face flushed, nostrils dilated, breathing quick.

"December 21.—No harm came of the high temperature. Discharged nearly well."

Since this time the patient remained quite well until three weeks ago, when the father noticed that the child vomited, became jaundiced, and that the urine was high coloured.

April 3.—Admitted to the Hospital with slight jaundice. T. 101·0°.

April 4.—Marks of scald over region of liver. Liver enlarged. Bowels very relaxed; motions coloured yellow. Urine acid, containing bile pigments; no albumen. P. 120. T. 99·8°.

April 5.—Ordered D. L.; Pil. hydr. c. cret. co., gr. iiss. statim; Sodæ bicarb., gr. iii.; spir. chlor., m. iii.; aq. carui., ʒi., 6ts horis.

April 6.—Slept well. Cries constantly when awake. Bowels rather less relaxed. Jaundice increased.

April 7.—Tenderness over liver. Bowels not so relaxed. T. 99·0°.

April 8.—Eats and drinks greedily. Not sick. B. O. 2.

April 9.—Sleeps much. Not sick. Motions solid, white.

April 10.—Sleeps a great deal. Jaundice increasing. Motions less frequent; quite white. P. 120. R. 32. T. 98·4°.

April 11.—Sleeping very soundly, almost comatose. Has not taken his food. Pupils large; do not react. T. 68·0°. Hydr. subchlor., gr. iii. statim; Spir. ammon. aromat., m. v.; spir. ætheris., m. v.; syrupi, ʒss., aquæ ad ʒii., 4ts horis.

3 P.M.—Less deeply asleep. Screams piercingly when awake. P. 120. T. 98·0°.

10 P.M.—Has had occasionally slight convulsions. Grinds his teeth. Some dark-coloured fluid was brought up by the mouth. P. 64. R. 32. T. 98·0°.

April 12.—Died at 2 A.M. this morning.

The history of the case thus briefly summed up is this:—Gradually increasing jaundice, without symptoms of gall-stones, hepatalgia, high temperature, hæmatemesis, coma, convulsions, death.

Post-mortem examination.—Universal bile staining. Large bile duct pervious. No bile in intestines or in gall-bladder. Liver small in size, flabby, watery in appearance, stained with bile, and hyperæmic. Spleen natural. Kidneys, slight cloudy swelling of cortex. Stomach contained a dark fluid, apparently altered blood, and similar to that vomited before death. No trace of duodenal ulcer. Lungs and heart natural; some caseous bronchial glands. Brain not examined.

The diagnosis made during life was that of acute yellow atrophy, but the case presented several features worthy of note. First, The age. Acute yellow atrophy in children is an extremely rare disease. Dr. Charles West[1] met with only one case in his large experience, and that at the age of 4½ years; but a few other cases are recorded by Dr. Duckworth and Dr. Tuckwell.[2] In the present case the child was only two years old. Secondly, The

[1] Diseases of Children, Dr. Charles West.

[2] St. Bartholomew's Hospital Reports, vol. vii. and vol. x.

cause. There was no history or evidence of syphilis, so that this must be excluded; nor of any other affection to which the attack could be directly referred, so that we are driven to consider the possibility of the hepatic lesion being associated with the scald on the abdomen of four months before. Dr. Murchison[1] gives a case, the clinical symptoms of which strongly resemble those present in this instance, and in which they were apparently referable to the effects of a chill. Thirdly, The extreme variation in the number of the pulse shortly before death; and, fourthly, The condition of the liver which microscopical examination disclosed, and of which the description follows here.

I must take the opportunity of thanking Dr. Church for his kindness in placing the liver at my disposal.

Microscopical examination.—(Liver hardened in alcohol and gum, and stained with hæmatoxylin.)

Fig. 1 shows the appearance of a section under a low power (Hartnack, obj. 2; ocular 3).

The lobules are distinctly marked off from each other by a zone of dense tissue completely surrounding them, in which numerous duct-like structures ramify. The lobules vary somewhat in size. The cells are no longer arranged in radiating rows as in the normal liver, and the central vein is remarkably distinct. A higher power (No. 7) shows this new tissue to be connective tissue, and fibrillar, rich in cells, partly spindle-shaped and partly round, the round being in great excess. Imbedded in this tissue lie the duct-like structures referred to. Processes of this tissue pass inwards in an irregularly radiating direction towards the central vein (fig. 2), forming a network with thick trabeculæ and irregular meshes. The connective tissue in the lobules differs only from that round them in containing fewer round cells, and being less distinctly fibrillar.

The gland cells have in great measure disappeared, and the few that remain are smaller than normal, granular, and pigmented.

The meshes are occupied partly by liver cells and partly by dilated blood vessels.[2] In some a blood vessel and cells lie side by side; in others only one or two cells are seen; and in others only a blood vessel.

The new growth of connective tissue seems to develop from the periphery towards the central vein, displacing the liver cells, and breaking the rows up into irregular clusters, and subsequently leading to their atrophy and disappearance by pressure.

[1] Murchison, Diseases of Liver, Case clxxix.

[2] Cf. Figure Hayem Arch. de Physiol., 1874, p. 157.

In this case the cells undergo a granular and pigmentary, but not a fatty degeneration, as is sometimes observed.

The duct-like structures vary considerably in size; the largest resemble bile ducts, are lined with cuboid epithelium, and filled full of cells, so that in most there is no lumen to be seen. In those of medium size the cells are smaller, and stained more deeply with hæmatoxylin; these, as well as the smallest, branch considerably, and are arranged for the most part concentrically with the lobule, while the branches run centripetally towards the central vein.

One and all have no proper wall distinct from the connective tissue by which they are surrounded.

To such cases as this the name of hypertrophic cirrhosis has been given. This must not be confounded with cirrhosis with hypertrophy, for any cirrhosis may be hypertrophic in some stage. By hypertrophic cirrhosis is meant a special form, with a definite clinical history and peculiar anatomical lesion. The preceding description of the microscopical appearances of this liver is sufficient for the pathological anatomy of the disease, which consists in a considerable new growth of connective tissue following the course of the bile ducts, and a large development of new bile ducts. "Hepatite interstitielle extra et intra lobulaire; catarrhe et développement anormal des canalicules biliares."[2] "Periangiocholitis chronica," (*cf.* fig. 4).

The clinical history is equally characteristic. Chronic icterus with exacerbations, attended by hepatalgia, fever, enlargement of liver, and gradually increasing icterus. Persistent enlargement of liver. No ascites, no dilatation of abdominal veins. Death with symptoms of icterus gravis.

All these symptoms stand in strong contrast with those of ordinary cirrhosis. Icterus is not usual. The liver is always, except in the earliest stage, atrophied. Ascites and dilatation of abdominal veins is marked. Hepatalgia and fever absent; and death occurs from various causes, gradual asthenia, ascites, gastro-intestinal hemorrhage, profuse alvine discharge, pneumonia, &c.,[3]—but not from icterus gravis.

Hypertrophic cirrhosis is interesting chiefly in its relations, on the one hand, to ordinary cirrhosis, and, on the other, to icterus gravis.

Taking anatomical grounds as the basis of his classification, M. Charcot divides cirrhosis into three groups.[4]

[1] Hanot, Cirrh. Hypertroph., Paris, 1876, Ollivier, L'Union Medicale, 1871.

[2] Hanot, ut supra, p. 30.

[3] Bristowe, Medicine, p. 296.

[4] Charcot et Gombault, Différentes formes de la Cirrhose du foie, Arch. de Phys., Ser. 2, iii.

1. *Atrophic* (granular hobnailed liver, common cirrhosis, cirrhosis of Lænnec), in which the fibrous tissue follows the branches of the portal vein (cirrhose d'origine veineuse), and forms a network with irregular meshes (annular), each including a varying number of lobuli (multilobular). This we may call *portal cirrhosis.*

2. *Hypertrophic,* in which the fibrous tissue develops round each lobule (monolobular), insulating it (insular), and following the ramification of the biliary ducts (cirrhose d'origine biliaire). To this we may give the name of *biliary cirrhosis.*

3. The third is a rare form, which has at present been observed only in some few cases of hereditary syphilis in young children. In this the new fibrous tissue develops in the lobule (intralobular) round the individual liver cells (monocellular).

From its rarity this class need not be discussed, but the two first are of importance, as corresponding with the two great groups into which most of the clinical cases fall.

Although the two processes may co-exist, may develop side by side, yet that they are essentially distinct and independent of each other is amply proved.

Experiment demonstrates that after ligature of the vein or duct,[1] the fibrous tissue which develops in consequence of the irritation is in great measure restricted to the course of the vessel ligatured, according as it be vein or duct; and in the case described, by reference to fig. 4 it will be seen how sharply defined the seat of the new tissue is, how dense it is round the ducts, how comparatively free the portal veins are.

The hypertrophic form, with its characteristic development of new bile ducts, may be produced by any lesion which sets up chronic inflammation of the ducts—*e.g.*, calculus, stricture, &c.[2] (*cf.* fig. 3); but it may also arise independently of these mechanical causes, as a lesion originating primarily in the ducts themselves. It then appears to commence, not in the great ducts, and to spread from them backward to the small ducts, as in those cases due to mechanical obstruction, but to originate in the middle-sized and small ducts, as a more or less idiopathic disease, one of which we do not at any rate yet know the etiology. It is to this, as it may be called, idiopathic form that the name of *hypertrophic cirrhosis* is limited, but the distinction is based upon an etiological, and not upon any anatomical difference in the process.

[1] Charcot et Gombault, Alterations du foie par lig. du canal choled., Arch. de Phys., 2d ser. tom. iii.; Wickham Legg, St. Bartholomew's Hospital Reports, 1872, 1873; Solowieff, Centralblatt f. d. med. Wissensch., 1872, No. 22.

[2] Kelsch et Keiner, Arch. de Phys., Nov.–Dec. 1876; Charcot et Gombault, Diff. formes, ut supra, p. 470; Cornil et Ranvier, Histol. Path., p. 924.

The relation of the new ducts to the disease is not yet satisfactorily determined, *i.e.*, whether they be old ducts which have persisted and become visible by atrophy of the liver cells,[1] or new ducts produced either by a process of budding from the old,[2] or by a transformation of the rows of liver cells.[3] The microscopical evidence seems in favour of the view that they are new formations, and that their development is an active process, and an essential part of the disease.

The chief interest centres perhaps in the relation of hypertrophic cirrhosis to icterus gravis.

Icterus gravis is a clinical expression rather than a pathological entity, and corresponds with varying pathological conditions. Anatomically we must distinguish at any rate three groups.

1. Acute fatty degeneration, of which the type is phosphorus poisoning.[4]

2. Hepatitis parenchymatosa with acute atrophy of cells. This would include most of the cases of acute yellow atrophy following pregnancy, &c.

3. Cirrhosis with acute degeneration of cells.

In all these groups the process is essentially acute, but in the first two it occurs in a previously healthy organ, and in the last in an organ already considerably diseased.

Of icterus gravis following cirrhosis of portal origin I have not been able to find any instances on record; but it is a not uncommon termination of cirrhosis of biliary origin, as we have seen, and especially of that to which the name of hypertrophic cirrhosis has been given.[5]

From this it is clear that we must not look to icterus gravis for any one anatomical lesion; and as uræmia is associated with very different conditions of the kidneys, so is icterus gravis with very variable conditions of the liver. The one factor common to the series is the acute destruction of liver cells,[6] and icterus

[1] Cornil, Arch. de Phys., Mars–Mai, 1876.

[2] Cornil et Ranvier, Histol. Pathol., p. 920.

[3] Kelsch et Kiener, Arch. de Phys., Nov.–Dec., 1876.

[4] Prof. Dr. Steiner, Jahrb. f. Kinderkeilk., 1871, p. 428 (akute atrophie der Leber aus Fett. degeneration); Ad. Dupre, Icterus gravis bei Schwangern und Wöchnerinnen, Strassburg, 1873; Humbert Mollière, L'ictère grave, Paris, 1875; Winiwarter, Stricker's Jahrbuch, 1872; Frerichs, Liver, Syd. Soc.; Picot, J. Anat. et Phys., Mai–Juin, 1872 (acute yellow atrophy following syphilis, with considerable new growth of connective tissue in liver).

[5] Murchison, Liver, Case ciii., following occlusion of duct by gall-stones. "A. Y. A. is frequent in some epidemics of catarrhal jaundice and in jaundice from syphilis." *Cf.* Case clxxix. for a case very similar to that described, but no microscopic examination is given.

[6] Frerichs, Liver, Acholia, vol. i. Syd. Soc. 193; Trousseau, Syd. Soc., iv. 318; Budd, Liver, p. 224; Jaccoud, Clin. Lect.; Murchison, Liver.

Fig. i

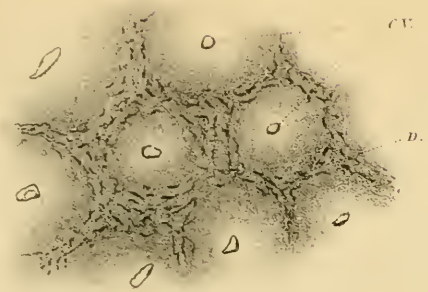


Fig. ii

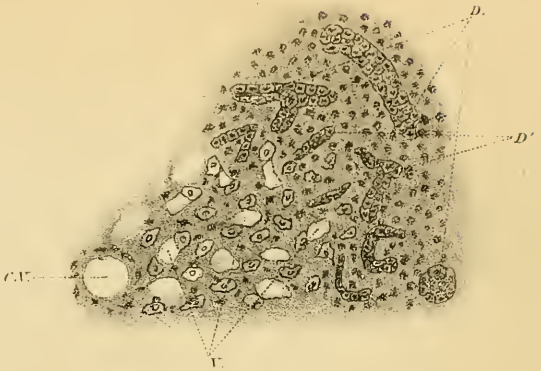


Fig. iii

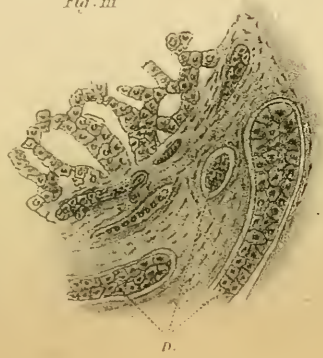
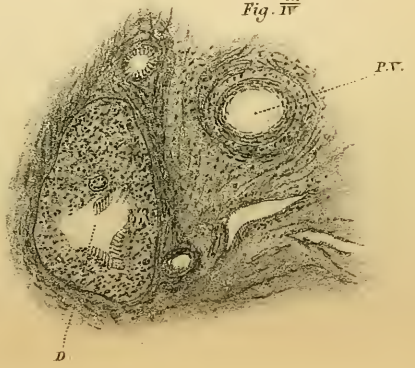


Fig. iv



gravis stands, it appears, in direct relation with this; but it is not yet clear whether the relation is one of cause and effect, or whether they are both the effects of some one antecedent cause at present unknown, *e.g.*, of some general intoxication or deep-seated blood change.

The *sine qua non* is that the atrophy be acute, but this may occur in a previously healthy liver (Groups 1 and 2), or in one already diseased (Group 3). Yet, on the whole, icterus gravis is rare in chronic affections of the liver, but in these diseases is far more common in the biliary form, *i.e.*, in cirrhosis of biliary origin. We can see a possible reason why this should be so. The cells of the liver depend for their nutrition upon the blood supplied by the portal vein. In portal cirrhosis the supply is gradually diminished, the cells consequently atrophy gradually and disappear. In biliary cirrhosis, however, the blood supply remains constant, but the cells, owing to the obstruction which exists to the passage of bile, are called upon to perform the normal amount of work under great disadvantages. They are overworked. A very slight increase in the amount of work demanded from them may lead to utter breakdown and complete disorganisation (Acholia, icterus gravis).

Whether hypertrophic cirrhosis will maintain the position claimed for it as a distinct disease, experience alone can determine; but this at any rate is evident, that under the clinical headings of cirrhosis and icterus gravis we have classed together a very heterogeneous mass of materials,[1] which require very careful sifting, and in performing this process the anatomical lesion must be our sole guide.

DESCRIPTION OF FIGURES.

Fig. 1.—Hartnack, Oc. 3, Obj. 2.

CV. Central veins.

D. New connective tissue round the lobules with the new ducts.

Fig. 2.—Oc. 3, Obj. 8.

CV. Central vein.

D. New ducts in the new connective tissue. The ducts were seen to have no proper wall separate from the connective tissue, and to have no lumen.

D'. Smaller ducts running radially towards the central vein.

V. Dilated blood-vessel in the lobule. The connective tissue has spread into the lobule towards the central vein. It contains fewer nuclei than that round the lobule. The hepatic cells are irregularly scattered about, are atrophied and granular.

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[1] Frerichs, Liver, vol. i. p. 95.

Fig. 3.—Oc. 3, Obj. 7.

Section of a liver from a patient in which the mouth of the ductus communis choledochus was occluded by a small cancer, showing a similar new growth of ducts in the newly formed connective tissue.

Fig. 4.—Oc. 3, Obj. 4.

PV. A portal vein.

D. A large bile duct. The sheath of the duct is stuffed with small cells, and round this the connective tissue is dense and compact. While the sheath of the portal vein contains hardly any cells, and though surrounded too by new connective tissue, this is not nearly so dense or so compact.

A CASE

OF

ANEURYSM OF THE RIGHT SUBCLAVIAN.

FOR WHICH AMPUTATION WAS PERFORMED AT THE SHOULDER-JOINT.

BY

LUTHER HOLDEN

Thomas Lambert, aged 44, a hale-looking farm labourer, was admitted into St. Bartholomew's Hospital on the 13th of May 1876, with an aneurysm of the right subclavian artery. The tumour is about the size of a small orange. It occupies the whole of the supra-clavicular fossa, extends inwards under the sterno-cleido mastoid, backwards beneath the trapezius, and downwards beneath the cavicle, which is pushed forwards and partially absorbed. There is slight pulsation over the episternal notch. The pulse at the right wrist is weaker than that of the left, and slightly delayed. The right arm is much wasted. He complains of constant and severe pain shooting from the shoulder along the inner side of the arm.

History.—Two years or more ago, while lifting a heavy weight, he felt a sudden pain over the right shoulder. A few weeks afterwards he noticed a small swelling just above his right collar-bone. This swelling increased at first very gradually; but has grown much more rapidly during the last six months. The pain has become of late so severe that he has been compelled to give up work, and to seek (for the first time) the advice of a surgeon, who sent him to St. Bartholomew's Hospital.

As in this case any attempt to place a ligature on the proximal side of the aneurysm offered no chance of success, it was resolved

to try the effect of ice upon the tumour, the ice to be applied in a bag; the patient being kept perfectly still in bed with the arm supported on a pillow. Only half diet allowed, with half a pint of milk daily.

September 25.—The result of the ice treatment, which the patient has borne uninterruptedly for nineteen weeks with great fortitude, is not satisfactory. Although the aneurysm appears a little smaller and harder, and its pulsation is less at its upper part, still it is more prominent and softer towards the clavicle, which is more thinned by absorption. The patient is decidedly weaker.

After a consultation, it was agreed to try direct uninterrupted pressure on the aneurysm. This is to be done by a bag containing 3 lb. of small shot. The diet to be restricted to meat, 4 oz., bread, 4 oz., milk, 5 oz., water, 5 oz., in twenty-four hours.

October 20.—The pressure and restricted diet have been steadily continued for twenty-one days. Patient has borne it extremely well. But the aneurysm is not materially diminished, nor is the pain down the arm less.

It was next resolved to try the effect of simultaneous compression of the brachial and common carotid. This was done on three successive days, namely, for eight hours on the first day; for ten hours on the second; and for eight hours on the third; making a total of twenty-six hours. During the five last hours of the third day chloroform was given, the patient being unable to bear the pressure any longer.

This compression of the arteries produced no change in the aneurysm, but very great (for a time alarming) exhaustion of the patient. It was noticed that pressure on the common carotid, firm enough to stop the flow of blood, lowered the pulse even to syncope, due probably to the pressure on the pneumo-gastric nerve.

October 23.—It was now determined to give up the restricted diet, and to restore, if possible, the exhausted strength of the patient, so that he might be able to undergo, as a last resort (if thought justifiable), amputation at the shoulder-joint.

November 10.—The patient has regained his strength and his spirits. The aneuryism beats more forcibly than ever, and makes daily perceptible progress toward the surface. All trace of the clavicle is lost in the general swelling. There is a dusky red circle of skin three inches in circumference over its most prominent part. As a fatal crisis was obviously imminent, a consultation was once more held. The majority of the surgeons present were of opinion that amputation of the arm was justifiable as giving a last, though remote chance, of inducing the formation of a clot and the ultimate contraction of the aneurysm.

November 11.—With the full concurrence of the patient, who was told of his critical position, the arm was amputated at the shoulder. The axillary artery was first exposed by an incision through the pectoral muscles, and tied close to the coracoid process, so that the ligature might be above an abnormal bifurcation of the artery into two large branches. The operation was then completed in the usual way. The wound was dressed with carbolised oil. The pulsation of the aneurysm was most distinctly diminished after the removal of the arm. The patient quickly recovered from the shock of the operation, and had several hours of refreshing sleep.

For the report of the subsequent progress of the case, I am indebted to my House-Surgeon, Mr. F. S. Eve, who watched it throughout its long course with the most assiduous care.

2nd day after operation.—Pulse, 162. Temperature, 104·0°.

Aneurysm is firmer, and pulsates only slightly. Slept the greater part of last night.

9·30 P.M.—Pulse, 150. Temperature, 103·0°. Has taken plenty of nourishment, and some brandy during day. Morph. inject. subcutan., gr. $\frac{1}{3}$.

3rd day.—Pulse, 132. Temperature, 101·5°.

Did not sleep after 12·30 A.M. Takes nourishment well. Stump dressed with carbolised oiled lint; dressings saturated with sanious serum; edges of wound have adhered except at the angles, where openings were left for drainage. No particular change in the aneurysm. Has a troublesome cough.

4th day.—Pulse, 116. Temperature, 100·8°.

Passed a good night. Looks better. Pulsation in aneurysm less forcible than it was last night. Takes food well. Stump looks well; abundant discharge of sero-purulent fluid. Bowels not open since the operation.

9·15 P.M.—Ol. ricini, ℥ss. Pulse, 120. Temperature, 102·0°.

5th day.—Pulse, 120. Temperature, 101·5°.

Did not sleep well. To have a morphia injection every night. Aneurysm slightly smaller and firmer, as if it contained a clot; skin covering it somewhat hot. Stump looks well; dressed with lint soaked in a solution of Condy's fluid and covered with gutta-percha tissue. The carbolised oil dressing was discontinued because it produced sooty urine.

2·30 P.M.—Temperature, 103·0°. 10·45 P.M.—Pulse, 110. Temperature, 101·4°. Bowels freely relieved.

6th day.—Pulse, 130. Temperature, 102·2°.

The aneurysm seems considerably smaller and firmer; the pulsation is only just visible. Stump going on well. Took some fish to-day. Vin. rubri, ℥iv. daily.

11 P.M.—Pulse, 120. Temperature, 102·0°.

7th day.—Pulse, 120. Temperature, 103·0°.

Did not sleep well. Complains of distension of abdomen from flatus, which has troubled him for the last day or two. Stump looks well; abundant discharge of somewhat offensive sanious serum and pus.

10 P.M.—Pulse, 120. Temperature, 101·6°.

8th day.—Pulse, 106. Temperature, 101·4°.

Has passed a very bad night; is very much troubled with a cough, which gives great pain. Edges of wound nearly entirely healed. Aneurysm slightly smaller and firmer. Pulsation less forcible.

The patient continued to progress favourably until the 14th day. The aneurysm gradually diminished in size until it did not project above the level of the surrounding parts. The temperature still continued high, and ranged from 99·3° to 102·0°; the pulse ranged from 100 to 116.

14th day.—Pulse, 112. Temperature, 102·0°. Respirations, 24.

Has a catching pain on right side. Impaired resonance and fine crepitation at base of right lung. Sputum slightly rusty.

15th day.—Pulse, 102. Temperature, 100·0°.

Cough better. No more rusty sputum, but viscid and purulent. Wound syringed with a solution of Condy's fluid, and dressed with lint soaked in the same solution twice every day.

He continued in much the same condition until the 24th day, on the whole improving, but troubled with a cough and occasional slight pain in the right side. The temperature ranged from 98·8° to 102·0°, being generally about 100·0°, and higher in the evening than in the morning.

24th day.—Pulse, 120. Temperature, 102·7°.

Ligature came away. During the last two or three days there has been considerable swelling, heat, and tenderness over the aneurysm. These untoward symptoms are more marked to-day, and raise a strong suspicion that the aneurysm is leaking.

26th day.—Pulse, 132. Temperature, 102·2°.

Aneurysm very tender, apparently increased in size, skin covering it hot and red. Stimulants gradually diminished. Stump healed, except a small opening in front. Cough painfully distressing.

29th day.—Pulse, 136. Temperature, morning, 102·5°; evening, 103·5°.

Skin inflamed over a wider surface. Much pain in the stump and the tumour, particularly when he coughs.

30th day.—Pulse, 150. Temperature, morning, 101·4°; evening, 101·0°.

Aneurysmal swelling increasing in size.

31st day.—Pulse, 140. Temperature, morning, 99·0°; evening, 101·0°. Respirations, 32.

The skin covering the aneurysm and surrounding parts is more red, hot, and tender. There is great swelling and brawny induration of the cellular tissue, extending also below the clavicle, over the scapula, and even to the integuments of the stump itself. A heaving pulsation is felt over the whole swelling. There is a superficial slough about the size of a crown-piece over the most prominent point of the aneurysm. Patient perspires profusely. Cough very distressing, and accompanied by a considerable quantity of muco-purulent expectoration.

32d day.—Pulse, 140. Temperature, morning, 100·0°; evening, 103·0°.

Very little change in aneurysm. Delirious last night. Takes ℥x. of port wine and ℥ij. of brandy in the twenty-four hours.

33d day.—Condition the same.

34th day.—Pulse, 140. Temperature, 99·4°. Respirations, 44.

Copious and fetid discharge of pus from opening in stump. Very little change in aneurysm; slough extending slowly; it has given way at one point, and discharges offensive watery fluid.

11 P.M.—Pulse, 148. Temperature, 101·5°. Respirations, 60.

Urgent dyspnœa; expectoration profuse and difficult; perspiring profusely. Morphia injection as usual.

35th day.—Pulse, 140. Temperature, morning, 98·2°; evening, 102·0°. Respirations, 46.

Delirious last night; parts around aneurysm more swollen; pulsation distinctly diffused throughout the whole mass. Cough very distressing.

36th day.—Pulse, 132. Temperature, 98·2°. Respirations, 44.

Delirium continues at night. Much weaker. Sinking from exhaustion.

37th day.—Pulse, 120. Temperature, 100·0°.

Died at 11·30 P.M.

Post-mortem examination thirty-eight hours after death.—Body somewhat emaciated. A large patch of sloughing integument covered the most prominent part of the tumour.

Stump.—Almost entirely healed. An abscess extended from the axilla downwards over the ribs.

Thorax.—The aneurysm projected into the right pleural cavity, compressing the apex of the right lung.

Lungs.—One to two pints of sanious serum in right pleural cavity; some recent adhesion. Left pleura covered with a layer of partially organised lymph. Mucous membrane of trachea and bronchi injected, and the bronchi filled with frothy mucus.

Heart. — Hypertrophy of left ventricle. Aorta dilated to nearly twice the ordinary size; very atheromatous, with calcareous patches. Innominate artery atheromatous, and dilated to the size of a normal aorta.

Subclavian artery (right).—Surrounding the second and third portions of the subclavian and the first portion of the axillary arteries was a mass of semi-solid blood clot, which lay in a sac formed by the matting together of the surrounding structures. The clot was divisible into two portions, an outer, the more recent; and an inner, firmer, more pliable and smaller, adherent to the outside of the aneurysmal sac.

The aneurysm involved the second and third portions of the subclavian artery. There was a wide slit across its upper surface, near its commencement.

The sac appeared to have collapsed considerably, for it was an inch or more distant from the clavicle, the under surface of which was eroded, and presented several very sharp points of bone towards the aneurysm

Liver, spleen, and kidneys healthy.

The preceding case, so far as I can ascertain, is the second on record of amputation at the shoulder-joint for a subclavian aneurysm.

It may reasonably be asked on what grounds such an extreme measure can be justified. First, there is the recognised principle, advocated by Brasdor and Wardrop, that an obstruction to the circulation beyond the aneurysm may have the effect of curing it. Secondly, there is the not unreasonable hope that the clotting and contraction of the sac may be promoted by the removal of the "vis a fronte" and "the attractive power. of the tissues" to be furnished with blood. Thirdly, there is the precedent established by the case[1] of Professor Spence, in which the aneurysm after the amputation of the arm diminished to one-third of its previous size, and the patient lived for four years afterwards. Lastly, in the face of impending death, was it not right to give the patient, at his own request, the last chance of prolonging life?

The immediate results of the operation seemed to promise

[1] Case recorded in the Trans. of the Royal Med. Chir. Soc. of London, vol. lii. p. 306.

success. The aneurysm began to harden and contract. The ligature came away on the 24th day. All was going on well. Soon afterwards severe bronchitis with pleurisy came on. The patient was distressed by violent fits of coughing and difficult expectoration. During one of these fits he begged to be raised so as to sit up in bed. It was just at this time, I suspect, that the sac gave way, and permitted the leakage which, combined with the chest mischief, proved fatal.

A CASE

OF

FRACTURE THROUGH THE BASE OF THE ODONTOID PROCESS.

BY

FREDERIC S. EVE.

(*The patient survived two and a half hours.*)

Andrew G., aged 20 years. While walking along the street, he was struck on the upper and back part of his head by an empty packing-case, which fell from a crane on the third or fourth story of a warehouse. He was stunned by the force of the blow.

When seen in the Hospital Surgery, immediately after the accident, he was quite conscious, and gave his name and address. He complained of difficulty in breathing, and said that he wanted to cough but could not. He was quite unable to perform a forced expiration. Respiration was entirely superior thoracic; not the slightest movement of the abdomen could be observed during inspiration. This fact was particularly noted and fully appreciated at the time. There was slight retrocession of the lateral parts of the thorax on inspiration.

His head lay back helplessly on the stretcher; both arms and legs were paralysed.

No irregularity or undue prominence of the spines of the cervical vertebræ could be detected.

Face pale, extremities warm, the shock of the injury being comparatively slight. Pulse, 90, regular. Temperature, 98°. Respirations, 36. There was urgent dyspnœa. At each inspiration the sterno-mastoids, sterno-hyoids, and sterno-thyroids stood out most prominently, drawing up the superior portion of the thorax, which alone moved on inspiration.

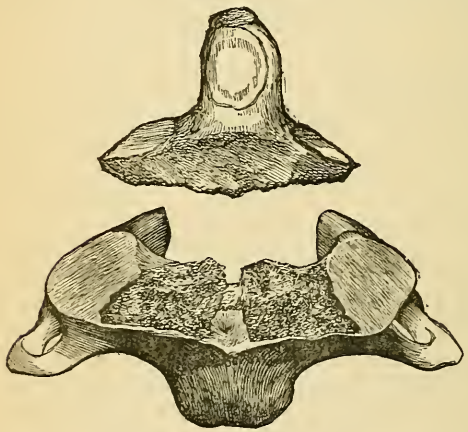
He was admitted into the Hospital under the care of Mr. Holden.

Especial care was taken to support the head while moving him from the stretcher into bed, an injury high up in the cervical spine having been diagnosed. Priapism was noticed.

The dyspnœa increased; he became dusky, and soon insensible, but retained for some time the power of swallowing. He died 2½ hours after sustaining the injury.

Post-mortem.—The neck was extremely loose and moveable, but no displacement of the vertebræ could be felt.

A fracture was found through the body of the axis at the base of the odontoid process; the fracture passed very obliquely from behind downward and forward, beginning posteriorly at the point where the nutrient artery enters the bone, and terminating at the centre of the greatest concavity on the anterior surface of the body; laterally it passed through the upper part of the superior articular facets.



The atlas with the odontoid process was dislocated forward; the displacement was easily reduced by raising the shoulders and allowing the head to fall back, and as easily reproduced by raising the head.

The spinal cord, considerably below the medulla, was nipped between the posterior arch of the atlas and the sharp fractured edge of the body of the axis; it was softened and ecchymosed, especially the posterior half, but not lacerated.

A small amount of blood was extravasated around the fracture.

The organs were healthy. The lungs and right side of the heart were engorged with blood.

Remarks.—The question naturally arises, in what manner could respiration be carried on after an injury to the cord just below the medulla, producing general paralysis of the body? I have no hesitation in stating that respiration was carried on almost entirely by the sterno-mastoid muscles, aided by the

sterno-hyoids and thyroids, the hyoid bone being fixed by the mylo- and thyro-hyoid muscles. In the absence of absolute proof to the contrary, I am bound to assume that the diaphragm was acting, however feebly, although no movement of the abdomen on respiration could be observed. Such being probably the case, the explanation would lie in the fact that the anterior segment of the cord was much less injured than the posterior.

The obliquity of the fracture prevented displacement of the atlas and odontoid process backward (in which case the medulla itself would have been pressed upon), but readily allowed the head to fall forward, thereby causing pressure on a lower part of the cord.

Although a very rare injury, yet fracture of the odontoid process appears to be the most common lesion affecting the first two vertebræ or their connections.

Dr. Stephen Smith[1] has proved, by a series of experiments, that much less force is required to fracture the odontoid process than to rupture the transverse ligament or fracture the anterior arch of the atlas; he also shows that the check ligaments, by the traction which they exert on the process when the head is violently driven forward or backward, have an important share in the production of this injury.

Malgaigne[2] describes fracture of the odontoid process as a variety of dislocation forward of the atlas. He speaks of it in the following terms under luxation forward of the atlas:—"Much more frequent than the others, they have been observed in two conditions: simple, or with rupture of the ligaments; complicated with fracture of the odontoid process, and even of the atlas." This view is not, I think, strictly correct; in many of the cases cited below, the displacement forward of the atlas upon the axis did not occur until some time after the accident, being then secondary to the fracture, and permitted in most cases by a gradual yielding of the ligaments connecting the two bones; while in three cases no dislocation occurred.

I have been able to collect twelve cases, including that described above, of fracture of the odontoid process from indirect violence, that is, other than gunshot injuries. Five were complicated with fracture of the atlas.

Two cases recovered completely. Of these, the following occurred in the practice of Dr. Bayard:[3]—

Charlotte M., aged six years, fell a distance of five feet, striking

[1] On Fractures of the Odontoid Process, American Journal of Medical Science, October 1871.

[2] Fractures et Luxations.

[3] Hamilton, Fracture and Dislocations.

her head and neck. She complained of pain on moving the head, and when walking supported the chin with her hands. Had no paralysis, nor was any deformity of the neck noticed.

Two months after the accident she had an attack of convulsions, followed by paralysis of the trunk and limbs; she remained in this condition three months, when an improvement began, and in seven months she was quite recovered.

Two and a half years after the fall a post-pharyngeal abscess formed, from which a piece of bone escaped, which was decided to be the odontoid process.

The other is the well-known case related by B. Phillips.[1] A farm labourer fell headlong from a hayrick; he was stunned for a short time, and then walked to the house of his medical man. He resumed his occupation in two days. A month after the accident there was a swelling in the throat, causing some difficulty in swallowing and thickness of speech; he was unable to turn his head. He lived for a year after the accident, death being caused by general dropsy. Post-mortem, the odontoid process was found to have been fractured at its base, and displaced forward with the anterior segment of the atlas, the latter bone having been fractured behind the articular processes. Bony union had taken place between the posterior segment of the atlas and the body of the axis. The specimen is preserved in the Museum of the Middlesex Hospital. This, I believe, is the only specimen of the kind to be found in the London museums.

In the following case,[2] although it terminated fatally, death was not due to pressure on the cord. A man aged 28 years fell from his horse; he remounted, and had ridden two and a half miles when something in his neck gave way with a snap, and he immediately fell to the ground insensible.

Symptoms.—A prominence was felt at the upper part of the nape of the neck; his chin rested on the sternum; rotation of the head caused acute pain; crepitation was felt. He had no paralytic symptoms. After a time he was able to walk, having his head supported by an apparatus. Abscesses subsequently formed about the neck, and he died of hectic and exhaustion two years after the accident.

The odontoid process was fractured at its base, and the posterior arch of the atlas broken across.

While the preceding cases show that this injury may be recovered from without any treatment, the following clearly

[1] Holmes's System, vol. ii. p. 396.
[2] Ed. Med. Surg. Jour., vol. lxiv. p. 527.

indicate that recovery might have been attained had the nature of the injury been recognised and proper treatment adopted.

A milkman[1] was thrown violently out of his cart, alighting on his head and face. He took some brandy and then drove home. Was unable to rotate his head; there was an unusual prominence at the upper part of his neck, with much bruising and swelling; no paralysis. He resumed his work in nine days, but complained of pain in his neck, which grew worse as time went on. One evening, about five months after the accident, he had a feeling of numbness in his left side, and afterwards in the right. Next morning he could stand only when supported, and shuffled his feet in walking; while dressing he sat down and immediately expired.

Autopsy.—The odontoid process was fractured and displaced forward with the atlas; it lay horizontally, with its fractured end pressing on the spinal cord.

Two cases related by Malgaigne lived one month and six days, and four months and a half respectively One had no paralysis. The patient, a porter, fell under a weight he was carrying. He continued his work for a month, when he sought advice for a deformity of his neck, which had existed since the accident. The head was inclined forward and to the left, but could be raised to its natural position. There was a depression at the upper part of the neck. He died in violent convulsions six days after admission to the hospital.

Post-mortem.—The odontoid process was found fractured.

The other case was that of a boy aged fifteen, who was thrown to the ground and received blows upon the back of his head and neck. The movements of the neck were embarrassed, and the head inclined forward. He continued well for nine weeks, when he lost the use of his extremities, and entered the hospital. A hard swelling was noticed at the upper part of the back of his neck. He died a week later.

Post-mortem.—The atlas was luxated forward obliquely, the right side inclining most forward. The odontoid process was fractured at its base, and lay nearly horizontally; it was united by bone to the axis.

The vertebral canal was contracted, by the displacement of the atlas, to a transverse fissure, two lines broad on the left and four-fifths of a line on the right side.[2]

I cannot omit the two following cases, as they bear directly on the most singular feature of this injury, viz., the absence of symptoms for a considerable time after the injury, and their

[1] Hamilton, op. cit., p. 165, reported by Dr. Parker.

[2] Malgaigne, op. cit., tom. ii. pp. 327, 329.

gradual development. I will, however, quote them as briefly as possible; a fuller account may be obtained in Dr. Stephen Smith's paper already referred to.

A man was admitted to the Bellevue Hospital for syphilis. He had a hard immovable tumour over the upper cervical vertebræ, which he stated was the result of a blow received upon the part a few months previously. Several days after admission he complained of pain in the neck and sleeplessness. Subsequently partial paralysis of all his extremities came on after sitting up suddenly in bed; next day the paralysis was more marked, and he died that evening.

Post-mortem.—The odontoid process was fractured and displaced forward with the atlas.

A labourer was admitted to the same hospital. Three months previously he had fallen from a building and received a blow on the head, which he thought had sprained his neck. He continued his work for six weeks, when he noticed a prominence in the back of his neck, and loss of power in his arms. During the following three weeks the deformity of the neck increased, and he lost all power in his left arm and leg; sensation was impaired in all the extremities. He had several severe attacks of dyspnœa, and died nearly six months after the fall.

Post-mortem.—Odontoid process fractured. The vertebral canal was reduced to $\frac{3}{8}$ in. antero-posteriorly by the luxation forward of the atlas.

In the following case, death is questionably referred to concussion of the spine. It is reported by Mr. Jeremiah M'Carthy.[1]

J. H. was admitted into the London Hospital with the history that while at work unloading a ship he had fallen head-foremost down the hold.

On admission he was perfectly sensible. There was complete motor paralysis of both upper and lower extremities; diaphragmatic breathing, priapism, and total loss of sensation below the level of the third rib. There was no perceptible irregularity of the vertebræ, but some swelling at the nape of the neck, and he complained of pain when pressure was made at this part. It was supposed that he had sustained a fracture of the spinal column about the fifth or sixth cervical vertebra. On the following day his condition as to respiration, priapism, and motor paralysis was unchanged, but he had completely regained sensibility in his upper extremities, trunk, and upper part of thighs. During the afternoon his temperature rose rapidly, he became violently delirious, and died next day.

Post-mortem.—The atlas was broken into five fragments, and

[1] Path. Soc. Trans., vol. xxv. p. 201.

the odontoid process fractured at its base. There was, however, no displacement, and no evidence of compression of the medulla or cord. "The symptoms in this case appear to have been due to concussion of the cord."

South[1] relates the case of a man who fell down-stairs. His limbs were paralysed, except the left leg, which subsequently became affected. Death took place on the fifth day.

Post-mortem.—Fracture of the odontoid process and of atlas; the cord was not, however, compressed at this point. The body of the fifth cervical vertebra was fractured, and the cord disorganised opposite to it.

Here, evidently, the fracture of the odontoid process produced no symptoms.

Mr. Hilton's[2] case is not included in the above, the fracture being almost certainly due to caries of the axis. The symptoms were precisely similar to some cases of fracture from violence. Death resulted from the pressure of an abscess sac upon the cord five months after the occurrence of the fracture. I mention this because the most careful treatment by position, &c., was adopted.

Separation of the odontoid epiphysis is said to occur in children under four years, before the process has become united by bone to the axis. I have not been able to find any recorded case, although separation of the epiphysis from disease is not rare.

A review of the foregoing cases brings us to the conclusion, that recovery from this injury is not only possible, but actually hopeful. Paralysis may be safely said to be a less frequent, and, at any rate, not so immediate a symptom as in fractures or dislocations of the spine lower down. The large amount of room allowed for the cord by the posterior arch of the atlas permits a considerable displacement before it is seriously compressed. The strong ligaments which bind the first two vertebræ together and to the occipital bone, especially the occipito-axoid ligament, take an important part in preventing dislocation; the yielding of these ligaments, however, permits a gradual displacement, but so gradual is the change, that the cord accommodates itself in a marvellous manner to the diminished space, and thus paralysis is for a time prevented.

An error seems to have crept into some surgical works of high repute, that fracture of the odontoid process and other lesions of the first two vertebræ are inevitably and immediately fatal.

Analysis of Symptoms.

Instantaneous death:—In one case.[3]

Paralysis:—Was immediate in three cases; absent for some

[1] South's Chelius, vol. i. p. 534. [2] Lancet, Oct. 1866, p. 459.
[3] Malgaigne, op. cit., tom. ii. p. 133.

time in six; absent altogether in two; in one case it was produced by a fracture lower down.

Deformity of neck:—Present in seven cases, although not immediately in one; absent in four cases; one case was not seen alive.

Loss of support to head:—Mentioned in nine cases.

Pain on movement of neck:—In five cases; not mentioned in six.

Crepitation:—In one case.

Mode of production:—Fall on head in nine cases; blow on neck in two; blow on head in one case.

I cannot here do better than quote Malgaigne's summary of the chief diagnostic points:—"If, then, by a minute examination an unusual depression is felt behind between the atlas and axis, with projection, may be behind may be to one side, of the spinous process of the axis; if the pain is seated precisely in the region of these two vertebræ and not lower; if above all rotation of the head is prevented; finally, if in carrying the finger to the top of the pharynx, the atlas is found extremely prominent; the union of all these symptoms would give to the diagnosis a probability approaching certainty, and even the coincidence of many between them lead to a presumption sufficient to justify the treatment." I would only add, that, in addition to the prominence of the atlas, a sharp projection would be felt at the base of the odontoid process.

Treatment.—One word, "rest," includes all that can be said in the way of treatment. The horizontal position should be strictly maintained, and the head supported either by sand-bags placed on either side, with a pad under the neck, or by means of a splint applied to the back and head. Of course no pillow should be allowed.

The ease with which the displacement was reduced after death, in the case first narrated, leads me to think that cautious traction on the head, with a slight inclination backward, would be justifiable in recent cases in which the cord was compressed, and also in those cases in which premonitions of paralysis had appeared from a gradually increasing displacement.

In concluding, I would offer an apology for having dealt with a subject already partially treated of by Malgaigne, and more fully by Dr. Stephen Smith; but trust that, by a careful grouping of cases and analysis of symptoms, I may have rendered this form of injury more easy of recognition, leading thus to its more efficient treatment in the future.

ON

THE FORMATION OF SYNOVIAL CYSTS IN THE LEG IN CONNECTION WITH DISEASE OF THE KNEE-JOINT.

BY

W. MORRANT BAKER.

My attention was first drawn to the diseased condition which forms the subject of the present paper by the following case, which was under the care at different times of my colleagues, Mr. Callender and Mr. Marsh, and of myself. For the notes of the case I am indebted to the records of the Surgical Registrar, Mr. Butlin.

Case I.

Large Cyst in the Calf of the Leg—Osteo-Arthritis of Knee-Joint—Amputation.

A woman (M. S.), 38 years old, was admitted into St. Bartholomew's Hospital, under the care of Mr. Howard Marsh, July 22, 1873, with a large swelling in the calf of the right leg. The right leg was about twice as large as the left, from just above the knee to the ankle. There was slight œdema, and the superficial veins looked tortuous and dilated. There was no great pain or tenderness, and no hardness or swelling could be felt in the track of the popliteal vein. The swelling was generally uniform, but especially marked in the calf, where deep-seated fluctuation could be felt. A slight pulsation was also perceptible, but was apparently only transmitted. There was also some effusion in the knee-joint.

The patient was thin, but otherwise in fair health, and complained only of numbness and very slight pain in the leg.

The history given by the patient was that five months ago the right leg began to swell, and had continued since slowly increasing. She thinks that, as she stooped one day, something cracked in the knee, and from that time it began to swell. She has had swelling of the leg after each confinement.

At a consultation which was held on the case, it was generally agreed that there was a quantity of fluid, perhaps pus, beneath the superficial calf-muscles, with probably thrombosis of the deep veins.

A day or two after the patient's admission into the Hospital, the swelling in the calf was punctured by Mr. Marsh with a very fine trocar, and several ounces of fluid were drawn off, leaving behind a considerable amount of thickening. Much to the surprise of those present, the fluid was not purulent, but apparently cystic. It was translucent, pale red, viscid, slightly turbid and alkaline. It contained a large amount of chlorides, and was almost solidified by heat and nitric acid. Microscopic examination failed to detect more than the presence of blood corpuscles; there were no pus-cells.

July 28.—The fluid has apparently collected again. The measurement of the right calf is 13⅝ inches; that of the left, 9⅝ inches. There is no enlargement of the femoral or inguinal glands. The swelling and thickening of the leg seems to be chiefly in the upper part of the gastrocnemius, especially in front of the muscle, and in its external head, and between the two heads, as well as some three or four inches lower.

The swelling below the calf is probably only œdema, on account of the pressure above.

July 31.—The swelling in the lower part of the leg is much diminished.

Since her admission the patient has been unable to retain either urine or fæces, which all pass involuntarily. This has been so, it is said, for some time past. An examination of the vagina and rectum, however, has discovered no abnormal condition, and throws no light on the condition of the leg.

August 4.—The leg is generally much smaller and less painful. Measurement of the calf is 12 inches. The condition of the knee is not changed.

August 16.—The thickening in the upper part of the calf is much less. The knee is bandaged.

September 5.—There is still some thickening in the upper part of the calf. The knee, in spite of careful and constant bandaging, is gradually increasing, apparently on account of

fluid in the joint. The leg is now abducted and slightly everted.

September 17.—The thickening in the upper part of the calf is apparently permanent, but not manifestly increasing. The knee is still enlarging. The patella is now much displaced outwards, and the leg is still more abducted and the foot everted. It seems as if there were some enlargement of the upper end of the tibia or the lower end of the femur. Measurement around the knee is $15\frac{1}{2}$ inches, and around the lower end of the femur, 16 inches.

Soon after the last note the patient left the Hospital, but was readmitted in August 1874, under the care of Mr. Callender, on account of the condition of her knee-joint. In his absence she was for a time under my care, and I had many opportunities of observing the state of her limb.

Since she had left the Hospital, the swelling of the knee had to a great extent subsided. About two months, however, before her readmission she fell down, and from that time the leg has been "out of place," and dangling loose and useless. There has not been very much pain. At the time of her readmission the right tibia was found dislocated outwards and backwards, and the leg hung loose and flail-like. It could be twisted easily in all directions, and even replaced in fair position, from which, however, it at once reverted to its mal-position when restraint was discontinued. The bones grated at the knee-joint, as if they had lost their cartilage. The synovial membrane was not now very much thickened, and there was no pain or tenderness, even on free movement.

The whole of the extremity was atrophied. No trace of the cystic disease of the calf, or even of thickening in this part, could be detected.

Attempts were made to improve the position of the dislocated bones, and to give such mechanical support as would enable the limb to be used, but without success, and amputation of the thigh was performed by Mr. Callender in January 1875.

Examination of the limb after removal.—The joint-surfaces were found in great part denuded of cartilage, smooth and eburnated, having nodules of bone growing out from their edges. Portions of the cartilage remaining were soft, vascular, and pulpy. The ligaments had been almost wholly destroyed. The synovial membrane was thickened, many of its processes standing out on its interior like small firm fibrinous nodules. A considerable quantity of viscid fluid was in the joint.

No trace of the cyst in the calf could be discovered.

On thinking over this case, it seemed to me more than prob-

able that the supposed cyst in the calf of the leg was formed really by a collection of fluid which had escaped from the interior of the knee-joint. The character of the fluid, the progress of the case as it developed, and the total disappearance of the cyst, so that even on examination of the limb after removal no trace of it could be discovered—all seemed to favour this view of its nature.

The following case, which I met with not very long afterwards, confirmed me in this idea.

Case II.

Osteo-Arthritis of Right Knee-Joint, with Cystic Tumour at Upper and Inner Part of the Calf of the Leg.

October 1875.—The patient, a man (J. S.), 52 years old, came to my Out-Patient Room at St. Bartholomew's Hospital on account of disease of the right knee. There were the usual symptoms of chronic rheumatoid arthritis, with a considerable amount of fluid in the joint; and the tissues seemed very tight, as if the fluid were under considerable pressure. The leg could be extended almost completely, but it could not be flexed beyond a right angle with the thigh. At the upper and inner part of the calf, and quite distinct from the swelling in the knee-joint, was a circumscribed oval swelling, measuring about two inches in length by three-quarters of an inch to an inch in breadth. Its long axis corresponded with that of the leg, and beginning four inches below the lower border of the patella, it extended along the inner edge of the gastrocnemius, slightly posterior to the inner border of the tibia.

Fig. *a*.

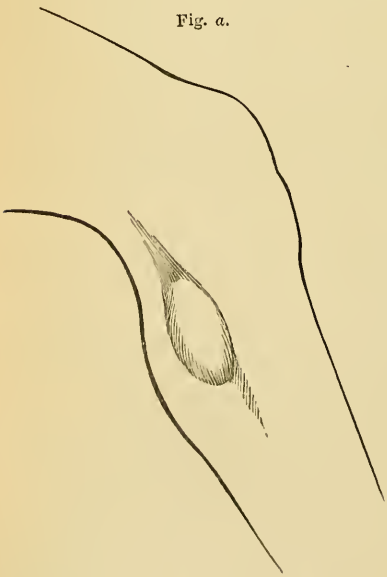


Fig. *a*.—Diagrammatic representation of the cyst in the calf of the leg, referred to in the text. The drawing is from memory only, as unfortunately no drawing or cast of the diseased part was taken at the time at which the patient was under my care.

It felt elastic, as if from the presence of fluid rather tightly confined within it, and seemed seated in the subcutaneous

tissue, the skin over it not being altered in any way. By firm pressure the fluid could be pressed apparently along what seemed a narrow prolongation of the tumour, leading off like a small canal in the direction of the knee-joint, but I could not trace this prolongation of the cyst quite to the joint, nor could I feel fluctuation distinctly transmitted by finger-pressure from the fluid within the knee-joint to that in the cyst. The contents of the cyst could not, moreover, be pressed out of it; the only effect of even firm pressure being to squeeze a certain amount of fluid along the small channel just referred to. The sensation given to the fingers was quite that of a closed cyst or cavity. Little or no pain was caused by the manipulation.

The patient first noticed a swelling of his right knee six months ago. The knee ached, especially on exertion, and has done so ever since. It was also stiff, and the stiffness has increased. About a fortnight after the first appearance of the disease, he came to the Hospital, and had a bandage applied, but did not apply for relief again until the present date. He used his leg up to this time.

About a week ago he first noticed the swelling on the inner side of the calf. It has not given him any pain or inconvenience, but has considerably increased in size.

The knee not improving under treatment in the Out-Patient Room, I admitted him for a short time into the Hospital. At this time (November 3) the right knee measured 14 inches round, and the left, 12½ inches. Under the influence of rest, strapping of the knee, and bandaging, the condition of the joint improved, and he was discharged in about a month.

December 20.—The patient came to the Out-Patient Room to-day. The knee was still swollen and rather tender; the general condition of the joint resembling that characteristic of chronic osteo-arthritis.

The cyst on the inner side of the calf was still present, but it seemed much more lax than heretofore, so that on firm pressure it could be nearly flattened out; but this seemed to occur rather from the laxity of its walls than from the pressure driving the fluid elsewhere. The popliteal space felt more full than that of the opposite leg; but I could not detect, on pressure, any fluctuation between either the popliteal space or any part of the knee-joint and the cyst.

The knee was again strapped and bandaged.

June 1876.—At this date I again saw this patient, and found that the cystic swelling of the leg had disappeared, and, he said, for a long time past.

The knee was still stiff, and the subject of chronic osteo-arthritis.

Taken in connection with Case I., I did not doubt that in this case the cystic tumour of the leg was caused by the escape of synovial fluid from the distended knee-joint.

I am indebted to my colleagues, Mr. Holden, Mr. Callender, Mr. Thomas Smith, and Mr. Willett, under whose care the following cases occurred, for permission to record them. The details of Cases III., V., and VI. are quoted from the notes of Mr. Butlin, whom I have also to thank for informing me of their admission into the Hospital.

Case III.

Cystic Tumour of Calf of Leg—Puncture—Subsequent Acute Inflammation and Suppuration of the Knee-Joint.

A man (J. H.), 53 years old, a surgical instrument-maker, was admitted into St. Bartholomew's Hospital, under the care of Mr. Holden, November 27, 1875, with the following history:—About a month ago he first noticed a swelling in the calf of the leg, which has been slowly increasing ever since. The redness, which was for some time present, has now passed off.

On admission, there was found a considerable tender swelling of the calf of the right leg, especially prominent at the upper and inner part, three or four inches below the knee-joint.

Under the impression that it was an abscess, it was punctured by the House-Surgeon, when there escaped a greasy fluid containing a number of flakes and masses of lymph, but no pus. It was thought at the time, in the absence of any other suggestion, that the disease might be a hydatid cyst; but a diligent microscopic search failed to find any evidence of this.

November 29 (two days after admission).—There is no discharge from the opening, which is surrounded by a wide area of superficial inflammation. The calf is generally much swollen, very painful, and tender.

November 30.—Suppuration has commenced within the cyst. Temperature, 101·2°. There is also now slight effusion into the knee-joint. No affection of the joint was noticed until to-day; and the patient states that there was nothing wrong with his knee before his admission into the Hospital.

December 3.—The knee-joint has been very tight during the last day or two, on account of effusion within it; but the inflammation of the calf has passed off. There is free discharge from the wound. Temperature normal.

December 6.—Pus mixed with synovia-like fluid escapes now freely, and can be made to issue from the wound in the calf by making firm pressure for a second or two on the knee-joint. The knee is rather less swollen and tense; but it cannot be straightened, nor can any attempt be made to do so without causing severe pain. The patient's general condition is good.

December 28.—Nothing worthy of special remark has occurred since the last note. It has been evident that there is a tolerably free communication between the cavity of the knee-joint, and the interior of the cystic swelling in the calf. Now there is scarcely any discharge from the opening, and what passes is like synovia. The leg has been bandaged, and is now much reduced in size, but the knee remains much swollen and somewhat flexed. A weight of 4 lb. has been applied to the leg in order to straighten the knee.

January 8, 1876.—The wound is soundly closed. Knee to be strapped with Ung. hyd. co.

January 19.—The joint is strapped as before, and the swelling is much diminished, although the knee is still contracted. The weight has been left off.

The patient was discharged from the Hospital, January 29.

I saw this patient again about a month after he left the Hospital. At this date (March 2) the knee was still somewhat swollen, but it seemed more like the swelling of rheumatoid arthritis than that of simple chronic synovitis. It was not hot or specially tender. Movements of flexion and extension could be performed over a considerable range. There was grating like that of rheumatoid arthritis when the joint-surfaces were pressed together, and some pain at the same moment. The ligaments seemed weakened or in part destroyed, so as to permit too free rotation of the tibia. The knee is still somewhat flexed. The patient said the joint was still too weak for walking, and he had not yet returned to work.

The scar of the puncture in the calf was soundly healed. It was about four inches below the knee-joint, on the inner side of the leg.

There was no swelling of the calf, nor the slightest indication of any connection of this part of the leg with the interior of the knee-joint. The integuments were also perfectly normal in every respect.

Case IV.

Cyst in Upper Part of the Calf of the Leg—Insertion of Seton—Inflammation of Knee-Joint—Amputation.

This case, it will be seen from the dates, occurred some years before either of my own; but I was not aware of the facts until Mr. Willett kindly offered me the notes for addition to those which I had previously collected.

The patient (J. M.) was a man 23 years old, who was admitted from the Out-Patient Room into St. Bartholomew's Hospital, under Mr. Willett's care, February 8, 1869, on account of a prominent swelling situate at the upper portion of the calf of the left leg, where the two heads of the gastrocnemius unite.

The history given by the patient was that two years ago he sprained the left knee, which has remained weak and slightly swollen. The popliteal swelling commenced eight months ago. It was not painful, but caused inconvenience by its size.

"On examination, the synovial membrane of the left knee-joint was found thickened, but there was no excess of fluid in its sac, and the joint was in a perfectly quiescent state. The tumour lay precisely at the lower angle of the popliteal space, overlapping the united gastrocnemius muscle, and was of the size and shape of a Tangerine orange. It was tense and fluctuated. Pressure did not reduce its size."

"A suspicion naturally arose in my mind," Mr. Willett continues, "as to a possible communication between the synovial membrane of the knee and the popliteal cyst, but it seemed negatived for these reasons—(1) By their relative positions; (2) the apparent complete isolation of the cyst; and (3) the absence of fluid in the knee-joint. The conclusion I came to was that the cyst was of a bursal character, and as a precautionary step I tapped the swelling with a trocar. A clear, slightly viscid fluid was drawn off, and the cyst was emptied so completely as to disappear. The patient kept about, the knee being unaffected; but at the end of ten days the swelling was nearly as large as before. I therefore resolved to attempt a radical cure, and with this object introduced, as a seton, a couple of silk threads. This measure was almost immediately followed by rapid synovitis of the knee-joint, with acute local inflammatory symptoms, and violent constitutional disturbance, excessive pyrexia, and delirium. The threads were removed at the end of twenty-four hours; but the febrile symptoms did not abate until the cyst was laid open, while diffuse suppuration in the calf and popliteal space followed, with, in the end, complete disorganisation of the

knee-joint. Amputation through the thigh was performed on the 14th of April, and the patient made a good recovery.

"The limb was carefully dissected, but it was impossible to trace the track of the communication, which evidently must have existed, between the knee-joint and the cyst, on account of the disorganisation of all the structures concerned."

Case V.

Osteo-Arthritis of Right Knee-Joint, with Large Cystic Swelling of the Calf of the Leg—Amputation.

A man (G. R), 56 years old, was admitted into St. Bartholomew's Hospital, under the care of Mr. Thomas Smith, June 2, 1876, on account of disease of the knee of three years' duration. The knee had become much worse during the last three or four months. The calf of the leg also has become swollen, and the swelling has extended to the foot.

At the time of admission into the Hospital, it was evident that there was extensive disease of the right knee-joint. The leg lay bent almost to a right angle with the thigh, and could not be straightened, and only slightly flexed. All movement was painful. The patella was scarcely moveable. There was evidently effusion into the joint, with thickening of the synovial membrane. Fluctuation was distinct on each side of the patella, and there was a large bulging and fluctuating swelling above the patella, on the outer and front aspect of the thigh. Fluctuation was distinct, extending from this part across to the front and inner aspect of the joint, beneath the common extensor tendon. There was some tenderness about the joint, but no marked increase of temperature.

Fig. *b*.

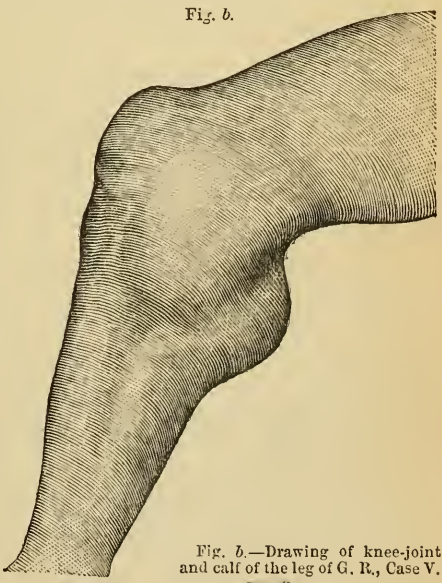


Fig. *b*.—Drawing of knee-joint and calf of the leg of G. R., Case V.

In the upper part of the calf of the leg, and in the popliteal space, projecting markedly on the inner side, was a large, tender, irregular, and fluctuating swelling (fig. *b*). Fluctuation could not, however, be communicated to the fluid in the knee-joint by pressure on this swelling in the calf. The lower part of the leg and foot were also much swollen and œdematous. The tibia was a good deal displaced backwards, but could be drawn forwards, with the occurrence, at the moment, of a sharp jerk or snap, as if the ligaments had been destroyed.

The patient's health was but little, if at all, interfered with.

At a consultation which was held on the case, it was generally agreed that the only hope of relieving the patient lay in amputation of the thigh, and this operation was performed by Mr. Smith, July 1st.

Examination of the limb.—On examining the leg after removal, the cavity of the knee-joint was found much increased in size, and containing several ounces of curdy purulent fluid. The cartilage was ulcerated, especially over the head of the tibia. The synovial membrane was thickened and pulpy. The bones were slightly softened beneath the diseased part of the cartilage. One or two small excrescences, similar to those usually found in cases of osteo-arthritis, were discovered; and there was eburnation of the articular surface of the femur, characteristic of the same disease.

The fluctuating tumour of the calf of the leg, which lay beneath the gastrocnemius, was formed by a large cavity, containing a precisely similar fluid to that which distended the knee-joint. This cavity communicated with the joint through a narrow sinus, which extended upwards along the back of the tibia.

Case VI.

Chronic Disease of the Knee-Joint—Large Cyst in the Calf.

January 6, 1877.—A woman (S. A. S.), 47 years old, was admitted into St. Bartholomew's Hospital to-day, under the care of Mr. Callender, with the following history:—

She has suffered from slight disease of the left knee-joint for the last five years; but although it has been sometimes better, sometimes worse, she has been able to get about without much discomfort, or even a stiff knee. Five weeks ago she was attacked with rheumatism in the right shoulder, then in the left, and in the left knee. Since that time the affected knee has been much swollen and painful.

Three weeks ago a swelling began to form in the left calf, and

has been very painful, but without any corresponding change for the better or worse in the condition of the knee-joint.

She has been "bodily ill" during the last five weeks.

There is now swelling of the left knee-joint, which is apparently the seat of old-standing chronic inflammation. The synovial membrane is thickened. The knee is slightly flexed, and cannot be extended. There is pain on movement, but not at other times. There is some increase of temperature of the affected joint, and tenderness on firm pressure.

There is a large fluctuating swelling in the calf of the leg; the fluid seeming, on palpation, to be just beneath the integument. The swelling is not more prominent on one side of the limb than the other, and is not painful or tender. On pressure, no fluctuation can be felt extending from this swelling to the knee-joint, or *vice versâ*.

The patient is sallow, and evidently much out of health. Tongue coated; breath foul; acid perspirations.

January 12.—To-day the calf was punctured with a grooved needle, and some clear viscid fluid, apparently synovia, was let out. There was no pus.

January 13.—There is some pain and swelling about the right ankle.

January 16.—The patient's general condition is much worse than the condition of the knee and calf of the leg seem to warrant. Her breathing is quick and jerky; there is frequent cough, and great prostration.

On examination of the chest, Dr. Southey found signs of slight lobular pneumonia in the subscapular region, with some impaired resonance beneath the left clavicle. T. 100·6°.

January 19.—An incision was made in the calf, and the cavity washed out with a lotion of carbolic acid (1 to 30). The fluid which escaped on making the incision consisted apparently of synovia, mixed with a little thin pus, and some curdy matter. The injection did not apparently pass into the interior of the knee-joint. Temperature before the operation, 100·2°.

January 20.—T. 99°.

January 25.—No discharge of fluid takes place from the cavity in the calf of the leg, but an ounce of carbolised lotion can be injected into it.

January 29.—The cavity is again discharging.

February 9.—The quality of the discharge, which still continues, varies from time to time, being sometimes purulent, sometimes clear.

February 15.—The patient has been worse to-day, and has suffered much pain in the knee during the last few days. Tem-

perature in the morning 99·8°; in the afternoon, 102·6° An erysipelatous blush was noticed over the calf and back of the knee.

After a sharp attack of erysipelas, which nearly proved fatal, the patient is reported (March 1st) to be better; the integuments of the leg wrinkling, and losing their red colour.

March 12.—All signs of erysipelas have now passed off.

March 21.—During the last few days the patient has had some ascites. The opening in the calf is now quite closed. The knee is in much the same condition as at the time of her admission into the Hospital.

On March 26 the patient was transferred to a medical ward.

November 5, 1877.—I saw the patient again at this date. She was looking healthy and strong, and was able to walk well with the aid of one stick or crutch. The knee-joint was still somewhat swollen, bulging as if from thickened synovial membrane rather than from much fluid within. The leg could be flexed and extended, but a good deal of creaking and fine crepitus could be felt by the hand at the same time. The joint seemed, the patient said, to "go in and out."

Except some slight firmness in the calf, as if the tissues were condensed by past inflammation, no trace of the large cystic swelling could be found. All seemed soundly healed, and only a small scar remained to show where the puncture had been made.

Case VII.

Effusion into the Knee-Joint with Fluctuating (Synovial?) Tumour in the Popliteal Space and Calf of the Leg.

A man (W. M.), aged 49, came to my Out-Patient Room; December 4, 1876, with an enlarged and very tense knee-joint, from thickening of the synovial membrane, with effusion. The popliteal space seemed also tense and swollen, and the calf of the leg was affected in a like manner. When pressure was made on the calf, there was a sense of fluctuation, at the same time that a marked swelling on the inner side was produced, resembling that which was present in some of the cases previously narrated.

The disease in the knee began about three and a half years ago, from no assignable cause, the joint reaching its present size in about a year and a half, and not altering much since that date. The calf had been swollen about twelve months.

October 1877.—I did not see this patient again; and on inquiry at his address at this date, found that he had died of "general debility" in June or July last. Since attending at St. Bartholomew's Hospital, he had been admitted into

Guy's Hospital, under the care of Mr. Howse; and to him and to Mr. Frederic Durham I am indebted for the following additional particulars of his case.

"He was in Guy's Hospital from April 28 to May 23. At this time there was an enormous amount of effusion into the knee-joint, extending for some distance (nearly half-way) up the thigh. It was tapped twice by Mr. Howse, 12 oz. and 8½ oz. of fluid being drawn off. The fluid contained flakes of caseous material.

"There was also a large fluctuating swelling below the knee, extending more especially over the inner surface of the tibia, and later on down the inner side of the calf.

"The case was looked upon as one of very advanced chronic osteo-arthritis."

Case VIII.

Cyst in the Leg of Uncertain Nature.

The following case, which I have found among my notes, may be here inserted for the sake of the accompanying drawing, which was made at the date of my seeing the patient. It doubtless belongs to the present group of cases; but the note is too fragmentary to be of much service. So far as I can remember, or gather from my notes, the patient was seen only once; and nothing is stated regarding the condition of the knee-joint.

Fig. *c*.

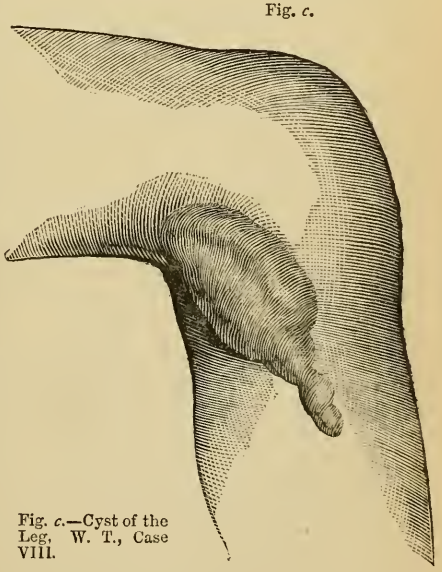


Fig. *c*.—Cyst of the Leg, W. T., Case VIII.

W. T., aged 62, came to my out-patient room November 4, 1872, with pain in the outer part of the thigh. While examining his leg my attention was accidentally drawn to a cyst-like swelling at the upper part of the calf, just below the knee. It was tense, and evidently contained fluid, and from its lower part was a short and slightly tortuous cord,

somewhat like an obliterated varicose vein. The skin over the parts was not at all altered in colour. The patient had noticed the swelling for above ten weeks.

The route which is taken by the fluid, when making its way out of the knee-joint to form an artificial synovial cyst in neighbouring tissues, is probably one determined by definite anatomical conditions. What anatomical arrangement is, however, most often concerned in guiding the fluid on its way, I cannot at present say.

In the only one of the cases already related in which there was an opportunity of examining with reference to this point, Mr. Butlin could only find a sinus running up to the back of the joint, and could not determine precisely its relations.

In the case of a little girl, whose leg was amputated by Mr. Callender in December 1875, on account of acute inflammation and suppuration within the knee-joint, and in which a large abscess had formed in the upper part of the calf, I found on subsequent careful examination of the limb that the abscess-cavity in the leg communicated with the interior of the joint by a narrow channel, which seemed to track by way of the tendon of the semi-membranosus muscle and its bursa; and there can be little doubt that the abscess, like the synovial cysts here described, owed its origin to fluid, doubtless in this instance puriform, which had escaped from the joint.

The late Mr. Wormald taught that the synovial membrane of the knee-joint was thinnest, and, therefore, most likely to give way under distension at the spot at which it partially encircles the tendon of the popliteus muscle; and Mr. Holden[1] states that there is a bursa under this tendon which generally communicates with the interior of the joint. It is quite possible, therefore, that the tendon of the popliteus may form sometimes a guide for fluid which is making its way out of the knee-joint; but further experience is necessary before a positive opinion could be expressed on this point.

In some cases, probably, there is a communication between the interior of the knee-joint and a bursa, the walls of which, having been first distended from the joint as far as they can bear, give way and permit their fluid contents to track along the limb.

It is somewhat curious, however, that in neither of the cases here related has the position of the cyst corresponded exactly with that of any normal bursa. It might have been expected that, at least in some instances, there would have been a special enlargement of one of those connected with the tendon of the

[1] Manual of Dissection, 3d ed., p. 490.

semi-membranosus muscle; but such a condition was not observed, unless Case VIII. be an exception; a swelling in the situation of the bursa being either not perceptible, or, if present, being merged in a much more extensive swelling, which involved neighbouring parts also. At an earlier stage of the disease it is quite possible that a noticeable enlargement of one of the normal bursæ might have been found to precede the larger or more distant artificial synovial cyst, which alone was manifest when the patients came under observation.

What connection, if any, the cases here related may have with the herniæ of the synovial membrane, described by some authors,[1] it is impossible to say. It may be assumed, perhaps, that the production of synovial herniæ may sometimes be the first step in the production of artificial synovial cysts of the leg, and that the fluid which tracks from the joint may escape by rupture of one of these hernial sacs, as it might from the ruptured wall of an over-distended normal bursa. But this is a mere suggestion. I have at present no facts wherewith to support it.

In an interesting article on popliteal cysts,[2] which I had overlooked until this paper was in type, M. Foucher refers to the occasional co-existence of hydrarthrosis of the knee with enlarged popliteal bursæ, and records six examples. In one of these the enlarged bursa was on the outer side of the popliteal space; in three on the inner side; and in two the position was median. The description of one of his cases corresponds to some extent with Case VIII. here related. M. Foucher considers it to have been an instance of enlargement of the bursa, common to the semi-membranosus and inner head of the gastrocnemius muscles. He refers to one example only of escape of fluid from a bursa into the tissues of the limb. The case was that of an officer, who first noticed a small swelling in the inner side of the popliteal space, three days after a forced march on a rough road. The tumour only very gradually increased. About eighteen months after its first appearance a sudden effort at extending the leg caused a rupture of the wall of the cyst; the tumour disappearing at the moment, at the same time that the calf of the leg began to swell. A bandage was applied, but the patient was not laid up. Two years afterwards the cyst was larger than ever; and for a short time the patient was obliged to lay up, as a part of the fluid contents of the cyst, after a tight bandaging, had extended on both sides of the knee.

[1] See Bilroth's Surg. Patholog., transl. by C. E. Hackley, M.D., 1874, p. 499, where a drawing is given after W. Gruber.

[2] Archives Générales de Médecine, 1856, vol. ii. ser. v. tome 8.

Ultimately the disease disappeared. The knee-joint appears to have been unaffected; and the case therefore has only an indirect bearing on the subject of the present paper.

M. Foucher appears not to have met with any instance of extension of fluid from the knee-joint beyond the limits of distended bursæ, and, indeed, seems to doubt the existence of any close relationship, in regard to cause and effect, between hydrarthrosis and popliteal cysts, in the examples of the combination of the diseased conditions which he records. "Quant à l'hydrarthrose, considerée comme cause efficiente," he is speaking of the etiology of popliteal bursæ, "nous ne lui accordons pas la moindre importance." In two of the six cases, moreover, which he relates, the enlargement of the bursa preceded that of the knee; and in the remaining four the articular synovial membrane was but slightly distended. "We must therefore conclude," he adds, "that the presence of hydrarthrosis is but a coincidence, although its appearance preceding that of the tumour lends some little probability to the opinion that a synovial hernial cyst may occasionally be present."

The passage of fluid from the interior of the knee-joint into the bursa beneath the inner head of the gastrocnemius muscle, in cases of chronic osteo-arthritis, is referred to by Athol A. Johnstone.[1]

Abscesses in the calf, as the result of the escape of pus from the knee-joint are much less rare than artificial synovial cysts. The comparative rarity, however, of their occurrence in connection with disease of the knee makes the following note, kindly written to me by my colleague, Mr. Howard Marsh, especially interesting in connection with the cases which are more particularly the subject of the present paper:—

"*December* 15, 1875.—There is a child now under my care, who, I believe, is an example of the class of cases you were mentioning the other day, in which fluid, originally formed in a joint, tracks away, and seems unconnected with the articulation. The child is about three years old. Some few weeks ago the knee was distinctly swollen, and hot, and lame, and a doctor who was consulted said it was the seat of disease. I saw it a few days ago, and then it had a distinctly circumscribed chronic abscess in the calf, just below the insertion of the Sartorius and its neighbouring tendons, with no sign of joint-mischief except a slight degree of puffiness, and very slight heat. I opened the abscess, and now the joint seems quite normal."

[1] Holmes's System of Surgery, 1870, vol. iv. p. 92.

The following are the conclusions deducible from the foregoing cases:—

1. That in cases of effusion into the knee-joint, and especially in those in which the primary disease is osteo-arthritis, the fluid secreted may make its way out of the joint, and form by distension of neighbouring parts a synovial cyst of large or small size.

2. That the synovial cyst so produced may occupy (*a*) the popliteal space and upper part of the calf of the leg, or may (*b*) be evident in the calf of the leg only, projecting most, as a rule, on the inner aspect of the leg, or (*c*) may be perceptible only at the upper and inner part of the leg as a small defined swelling, not approaching within three or four inches of any part of the knee-joint.

3. That however large the synovial cyst may be, fluctuation may not be communicable from it to the interior of the knee-joint; but the absence of such fluctuation must not be taken to contra-indicate the existence of a connection between the joint and the cyst.

4. That the synovial cyst may be expected to disappear after a longer or shorter period, without leaving traces of its existence, even on dissection of the limb.

5. That the cyst should not be punctured or otherwise subjected to operation, unless there appear strong reasons for so doing; inasmuch as interference may lead to acute inflammation and suppuration of the knee-joint.

6. That most often the disease in the knee-joint will be found to have begun some time before the appearance of the secondary synovial cyst; but sometimes the patient's attention may be first drawn to the latter, or the cyst may seem for a long period the more important part of the disease.

ON

THE ÆTIOLOGY OF MITRAL STENOSIS.

BY

DYCE DUCKWORTH, M.D.

It is still commonly taught and believed, that of the several forms of valvular disease of the heart, no one less often owns rheumatic inflammation for its cause than stenosis or contraction of the mitral valve. If this indeed be a fact, it is certainly a remarkable one, and well merits an explanation.

In this paper I have to record the results of a special inquiry into the connection between rheumatic endocarditis and a resultant mitral stenosis.

The cases furnishing the basis for this inquiry are eighty in number, and all of them came under my immediate observation either in the Out-Patient department or in the wards of the Hospital.

It is right to state here that I have been collecting notes of these cases for the last four years, and on two occasions during this period have drawn up for my own information statistics respecting certain points in them. In Dr. Hayden's classical book on "Diseases of the Heart," published in 1875, I found the little task I had proposed to myself elaborately carried out, and it was a matter of much interest to me to learn that his large number of cases, together with those recorded by Dr. Hilton Fagge, bore out with singular exactness the points I had ascertained for myself in the same direction.

As I believe it to be always of use in practical medicine to corroborate facts, I make no apology for adding my cases and deductions to the literature of this subject, which cannot as yet be affirmed to be extensive.

Table I.

No.	Name.	Age.	Sex.	Rheumatic Antecedents.	Remarks.
1	G. Miles	28	Male	Rheumatic fever 4 years previously in Mark Ward	A painter
2	E. Cooper	50	Female	Do. do. three times	
3	C. Thomas	18	Male	Do. do. 3 years previously	Hepatic enlargement (?). Præsystolic murmur heard behind
4	James Morris	39	Male	Had "rheumatic gout" 11 years ago in St. Thomas's Hospital; not very ill with it	Alleged to have had palpitation of heart when 3 or 4 years old
5	M. Plaws	47	Female	Two attacks of rheumatic fever	
6	Anna Cody	18	Female	None	Occasional hæmoptysis 12 months ago
7	M. A. Chambers	35	Female	One attack of rheumatic fever when 13 years old in Hope Ward	Signs of phthisis in both lungs; hæmoptysis; phthisical history on both sides of family
8	H. Munslow	18	Female	None	Palpitation for 18 months; sister died cyanotic æt. 3 years
9	M. A. Woodall	37	Female	None	Dyspnœa and palpitation for 2 years
10	Eliza Burton	46	Female	None	Cough and occasional hæmoptysis for 2 years
11	Nathan Orton	63	Male	None	Had gout; is a brewer's labourer; bronchitis for 3 yrs. past
12	Charles Thomas	21	Male	Rheumatic fever 4 years ago after immersion	
13	Elizabeth Farthing	28	Female	Uncertain	Symptoms date back 6 years
14	John Sims	46	Male	Rheumatic fever 5 years ago	An old soldier; had syphilis 20 years ago; also fever in India
15	John Rentmore	55	Male	None	Cupped and bled at Guy's Hospital 26 years ago for palpitation
16	Martha Besley	50	Female	Seven attacks of rheumatic fever in this Hospital at different periods	General bronchitis, with emphysema
17	Elizabeth Playfair	38	Female	None	"Short-winded as a little girl"
18	Emma Hill	46	Female	None	
19	Ellen White	46	Female	None	Palpitation on exertion "all her life" (Irishwoman)
20	Sarah Costello	35	Female	None	Palpitation for 6 years
21	Mary J. Rowland	20	Female	None, but had six attacks of chorea	
22	Louisa Freshwater	32	Female	Rheumatic fever 7 years ago	Embolismic hemiplegia (left side)
23	Lucy Westcott	19	Female	Rheumatic fever 6 years previously; admitted with second attack	Aortic regurgitation supervened
24	Solomon Barton	47	Male	Severe attack of rheumatic fever 15 years before	
25	William Cook	23	Male	Seven attacks of rheumatic fever	
26	William Waterman	25	Male	Four attacks do. do.	Tricuspid regurgitation ensued
27	Caroline Tanner	23	Female	Two attacks do. Chorea after first attack	
28	Clara Peel	37	Female	None recorded in notes	
29	Emma Steer	17	Female	None	Dyspnœa for 2 years; palpitation for 6 months
30	Catherine Deverell	27	Female	Two attacks of rheumatic fever	
31	Selina Newman	26	Female	Rheumatic pains for 6 months past; no history of rheumatic fever	Has aortic regurgitation also; patient of Dr. Hensley's
32	Ann Sell	36	Female	Two attacks of rheumatic fever	
33	Sarah Ann Wharry	36	Female	Rheumatic pains	Embolismic hemiplegia (right side); aortic incompetence
34	Alice Childs	15	Female	Rheumatic fever 4 years previously	
35	L. T.	21	Female	No history of rheumatic fever, but admitted with articular pains and effusion	
36	J. L.	23	Female	Rheumatic fever 16 years previously	Death, autopsy. Aortic disease as well as mitral
37	——	21	Female	Two attacks of rheumatic fever	

38	Robert Mackay	35	Male	None	Late in Royal Engineers; palpitation for 15 years
39	———	32	Male	Rheumatic fever 20 years ago, treated in this Hospital	
40	S. B.	22	Female	None	Mitral regurgitation supervened while in Hospital
41	M. de S.	24	Female	None recorded	Aortic regurgitation also. Præsystolic murmur heard at lower angle of left scapula
42	S. R.	36	Female	Rheumatic fever 3 years previously	Tricuspid regurgitation also
43	Eliza Doran	20	Female	Two attacks of rheumatic fever	
44	Louisa Tobias	26	Female	Rheumatic fever 8 years previously	
45	Ann Cell	38	Female	Two attacks of rheumatic fever	
46	James Streeter	15	Male	One attack do. do. 1 year previously	
47	Emma Edwards	40	Female	None	
8	Jemima Labern	32	Female	Rheumatic fever 17 years previously	Embolismic hemiplegia with aphasia (right side)
49	Lucy Elston	20	Female	Two attacks of rheumatic fever in this Hospital	
50	E. A. Gutteridge	39	Female	Rheumatic fever 5 years previously	
51	M. A. Armstrong	27	Female	None	Pulmonary symptoms and hæmoptysis for 2 years
52	Ann Holloway	49	Female	None	Tricuspid regurgitation; "nutmeg" liver
53	W. H. J.	19	Male	Rheumatic fever 13 months previously	Palpitation and dyspnœa for yrs. before he had rheumatic fever; præsystolic murmur audible at lower angle of left scapula
54	M. A. G.	26	Female	None	Palpitation for 14 months
55	E. M. French	36	Female	Two attacks of rheumatic fever	
56	M. A. Lynch	40	Female	None	Ailing 4 or 5 years; illness began with "faints"
57	Lydia Hale	35	Female	Two attacks of rheumatic fever	
58	F. Balls	19	Female	None, but had three attacks of chorea	Aortic regurgitation also
59	Jno. Busson	15	Male	None	Scarlet fever some yrs. previously; probably adherent pericardium
60	Emma Stapleton	25	Female	Rheumatic fever 13 or 14 years previously	Aortic regurgitation also; epileptiform attacks
61	Louisa Tilford	21	Female	No report	
62	M. A. B.	53	Female	Rheumatic fever 15 years previously	
63	Anne Ritallick	25	Female	None	
64	Sophia Payne	46	Female	Rheumatic pains only	Symptoms developed suddenly
65	Sarah Cartwright	23	Female	Rheumatic pains a few weeks before admission	Embolismic hemiplegia (left side)
66	—— Honeylove	47	Female	Rheumatic fever 14 years previously	
67	Mary Smart	55	Female	Do. do. 3 times do.	
68	Harriet Painter	33	Female	Do. do. 21 years before	Epileptic
69	Mary Ann Tuck	39	Female	Do. do. 8 or 9 years before	
70	Anne Embleton	18	Female	Do. do. 4 yrs. ago, admitted with second attack	Aortic stenosis and incompetence ensued
71	Elizabeth Purger	25	Female	Rheumatic fever 18 years before	
72	Elizabeth Guntriss	36	Female	No history of rheumatic fever; hands affected with osteoid arthritis	Palpitation for 20 years; a brother has "chalky" gout
73	Henrietta Hall	14	Female	None, but chorea at 6 years old, several attacks since	
74	Alfred Husband	15	Male	None	
75	Anne Geoghegan	31	Female	Rheumatic fever 17 years previously	
76	John M'Ord	35	Male	Do. do. 3 years ago	
77	Amelia Coyne	41	Female	None, but chorea 23 years ago	Palpitation first came on during an attack of left pleurisy one month ago in London Hospital
78	Emily Orton	32	Female	Rheumatic fever 6 years previously	
79	Ann Thorpe	44	Female	Three attacks of rheumatic fever	
80	Caroline Perry	18	Female	None, but three attacks of chorea	Aortic disease in addition (double murmur)

I believe that, with the amount of material now accumulated, it is possible to draw with large measure of exactness some conclusions which are both important and interesting in respect of the ætiology of mitral stenosis.

It should be remarked that the diagnosis in my cases has been arrived at for the most part after repeated examinations; that the patients were all seen in hospital practice, and made the subjects of instruction to numerous pupils.

I might add, that I had the advantage of learning to recognise the auscultatory and other phænomena of mitral stenosis in my student-days, at a period when the subject attracted a good deal of attention and discussion at the hands of the best authorities.

Some of the cases in the above table were under the care of Dr. Andrew, and therefore have the additional security of his diagnosis attached to them.

In the eighty cases above tabulated (Table I.), only seventeen occurred in the persons of males. The average age of the eighty cases is 31·77 years; of the sixty-three females, 31·79 years; of the seventeen males, 31·70 years.

Of the seventeen males, eleven had history of rheumatic fever or rheumatism; in one there had been "rheumatic gout;" and no rheumatic affection could be traced in five cases.

Of the sixty-three females, thirty-eight had history of rheumatic fever or rheumatism; five had had chorea, and this affection followed on one of the cases of rheumatic fever; one had osteoid arthritis. In fifteen cases there were no rheumatic antecedents discoverable, and in four the report was incomplete on this point. There is no history of chorea in any of the males. I have deemed it fair to put all the cases together in which rheumatic antecedents were noted, and have included the instances of chorea in this category for obvious reasons.

An examination of Table II. shows that in fifty-six out of eighty cases, or 70 per cent., there was a certain or strong presumption of rheumatic antecedents, and it is not too much to say that a more careful sifting of the histories might have led to a discovery of these in a somewhat larger number. For it must be remarked that it is very difficult to procure history of rheumatic affections in many patients. The phænomena of acute rheumatism in the young are markedly different from those observed in the adult; and many an endo-, myo-, and pericarditis run their courses unobserved and unnoted. Hence, I believe, it might fairly be said that, if the whole truth was known, two-thirds of all the cases of mitral stenosis own a rheumatic origin. In my cases there were twenty-one devoid of rheumatic antecedents, and the record is uncertain upon this point in four cases.

Certainly no other morbid condition can be charged to the same extent with the induction of mitral stenosis.

TABLE II.

No.	Physicians.	No. of Cases.	SEX.				Avge. Age.	Rheumatic or Choeric Antecedents.	
			M.	Pr. ct.	F.	Pr. ct.		Cases.	Pr. ct.
1	Dr. Hayden,* . .	81	27	33·3	54	66·7	(Of 77 pts.) 29·63	40	49·3
2	Dr. Fagge,† . . .	66	28	43·1	37	56·9	(Of 61 pts.) 32·77	33	50
3	Professor Gairdner, ‡	7	3	42·85	4	57·14	...	Not recorded	...
4	Dr. Hyde Salter, §	7	2	28·57	5	71·42	24·8	5	71·4
5	Dr. Dyce Duckworth,	80	17	21·25	63	78·75	31·77	56 { 12 males 44 fems. } (Chorea recorded in six cases, in one of which it followed rheumatic fever. In my earlier cases chorea was not carefully inquired for.)	70
6	Sundry cases collected from St. Barth. Hospl. post-mortem records, Path. Socy.'s Trans., &c., &c. ‖	23	9	39·13	13	56·52	31·5	7 of 11 reported cases.	63·63
		264	86	34·64 = m. avge.	177	64·33 = m. avge.	30 = mean average.	141	60·8 = m. avge.

Gout does not figure in the ætiology of mitral stenosis to any noteworthy extent. In one case only, and that in the oldest man who presented himself with this lesion, was there clear history of gout. In Case 72, a woman had her hands affected with typical osteoid arthritis, and related that her brother suffered from "chalky" gout. One man brought a history of "rheumatic gout," and these were the only cases in which osteoid arthritis was noted. The man's case, indeed, is not to be reckoned as an absolutely certain one. As he was in Hospital eleven years previously, "but not very ill," on account of this ailment, at the age of twenty-eight, it is not likely that the case was one either of gout or of osteoid arthritis, and it was most probably one of mild rheumatic fever, perhaps not the first attack in his life, for he stated that he had had palpitation of the heart at an early age.

Dr. Fagge has hazarded the conjecture that scarlet-fever or

* Diseases of the Heart and the Aorta, Dublin, 1875, p. 964, et cæt.
† Guy's Hospital Reports, Ser. iii. vol. xii. Sex not recorded in one case.
‡ Clinical Medicine, Edinburgh, 1862.
§ Lancet, vol. ii. 1869.
‖ Sex not recorded in one case.

diphtheria may sometimes figure in the ætiology of this affection.[1] My cases lend no particular support to this view.

I think Dr. Fagge has fully disposed of the hypothesis that some of those cases are due to malformation of congenital (*i.e.*, intra-uterine) origin. At all events, such cases do not, as a rule, come before one in the persons of infants, or in very young children, and the majority of sufferers seldom date back their troubles beyond a few years. The sudden onset of cardiac symptoms is noteworthy in many of these cases. This is no doubt significant of slowly progressing disease of the mitral valve, and of some special strain thrown upon the heart or circulation at the particular time the distress was remarked. Dr. Clifford Allbut of Leeds related at the Clinical Society of London two or three years ago the histories of two cases of mitral stenosis which had apparently followed upon injuries to the wall of the chest, and in which there was no history of rheumatism.

I have made inquiries upon this point in a number of my cases, and have found no important facts to support the view that direct injury to the thoracic wall is a common factor in their production.

In one of my cases, a woman with no rheumatic history had often been struck on the chest by her husband; in another, the first symptoms followed a strain on lifting a heavy basket. In Dr. Bright's case, with no rheumatic history, the cardiac trouble began after unusual exertion in running.

Dr. Fagge records one case which dated from a fright in connection with a fire in a house, another in which a hurt of the chest was sustained by a fall from a railway carriage, and a third which was attributed to a strain in moving a piano. In all these instances one would be prepared to believe that pre-existent disease of the valve only awaited some special cause to elicit distinct or aggravated manifestation of symptoms. Such a view is at all events held respecting similar affections of the aortic valves, and there seems to be no reason to argue differently in these two cases.

Syphilis has been supposed to account for some few instances in which no rheumatic or other antecedents were noted, but no plainly recorded facts that I know of warrant this view.

Dr. Fagge has offered an opinion to the effect that in children and young subjects the mitral valve is especially prone to undergo changes akin to slow or low forms of chronic inflammation, leading to the condition of stenosis. Such changes, he holds, must be considered distinct from those produced by rheumatic or choreic disease, and he further thinks it not unlikely that the

[1] Reynold's System of Medicine, vol. iv., 1877.

same affection explains like conditions of the aortic and tricuspid valves.

We may, I believe, accept this position, as a temporary one at all events, and invoke such a hypothesis to include such cases as cannot in any fairness be attributed either to rheumatic or to degenerative valvular affection of more specific character.

How it comes to pass that females are so much more liable to this form of mitral disease than males, I am unable to explain. The fact remains, and is in accordance with our knowledge as to the greater general frequency of mitral than of aortic disease in the same sex. The ratio of frequency in aortic disease in the two sexes is twenty-one men to five women (Hayden).

Dr. Hayden remarks, at page 893 of his work, that mitral stenosis is most frequently met with in children. I can hardly agree to this statement. A glance at the statistics collected by himself, by Dr. Hilton Fagge, or by myself, shows that the largest proportion of cases clinically met with and recognised occur in adults between the ages of twenty and thirty-five years, the mean average being thirty years.

The youngest of my patients was fourteen. Dr. Hayden met with two at seven years of age. Dr. Fagge had no patients under ten; and in the whole of my eighty cases there were only four patients between the ages of fourteen and seventeen. Large numbers of children are brought to St. Bartholomew's and Guy's Hospitals, and should furnish examples of this affection if it was especially to be found amongst them. My oldest patient was a man aged sixty-three, with no rheumatic antecedents, but he was gouty. Dr. Fagge also met with two cases in persons over sixty years of age.

It is not easy to offer an explanation of the fact brought out by the statistics that the most usual age to meet with cases of mitral stenosis lies early in the third decade. There is often, as has been shown, an antecedent history, extending in many cases over years, but yet no urgent symptoms had led to advice being sought. We must suppose that the difficulties falling upon the circulation were gradually imposed, and there is every reason to believe that the full measure of stenosis is reached by progressive indurating changes in the ring or curtains of the mitral valve. That stenosis itself is not always the final step attained in some cases, is proved by the fact that further changes progress in the valve, which comes at last to permit of regurgitation. At all events, the clinical phenomena in certain instances distinctly point to this.

I am not disposed to lay any stress upon the fact that most cases of stenosis occur about the age of thirty, for I imagine

that a statistical record of cases of mitral disease of all forms would show much the same result. Dr. Hayden found the average age of twenty-five cases in both sexes of mitral regurgitation to be thirty-one years and four months, and he likewise found that more than half the cases of aortic disease in females occurred in the third decade.

In two cases the præsystolic murmur was audible at the lower angle of the left scapula. These patients were both under Dr. Andrew's care, and he called my attention to this unusual clinical fact. The *rhythm* of præsystolic murmurs is often detectible in this situation in thin persons, but the *murmur* is most rarely audible here. In Case 3 I also thought I heard the same.

I purposely withhold all comments upon this and other clinical points in the present paper, the scope of which does not admit of such discussion.

The deductions from the statistics I have drawn up and collected are mainly as follows (264 cases):—

(1.) That mitral stenosis owns a *rheumatic* origin in the majority of instances, at least in 60 per cent., the term being employed in a comprehensive, but not inexact, sense, and it would probably not be wrong to believe that two-thirds of all cases are the result of rheumatic disease.

(2.) That in the present state of knowledge upon the subject, no other ætiological cause can be so distinctly connected with this lesion.

(3.) That there appears reason to believe that the mitral valve is sometimes affected in children in the course of the exanthemata, or may be prone in young persons to slow degenerative changes resulting in stenosis (Fagge).

(4.) That direct injury to the thorax, or the effects of strain, can hardly be accepted as ætiological factors in the disease.

(5.) That congenital forms of endocarditis do not as a rule lead to this lesion, at all events in an uncomplicated form.

(6.) That females suffer more frequently than males, almost in the ratio of two to one. (My own cases give a much larger proportion than this, viz., 78 per cent. against 21 per cent.)

(7.) That clinically the majority of instances present themselves between the ages of twenty and thirty-five, and that they are not commonly met with after forty-five.

A CASE

OF

GREAT ENLARGEMENT OF THE PONS, CRURA CEREBRI, AND MEDULLA.

BY

P. KIDD.

I have to thank Dr. Gee for permission to publish the following case, which was under his care in the Children's Hospital.

Florence B., aged 6½ years, was admited on April 26.

Her condition on admission is thus described:—The child is well nourished. Eyes prominent, with convergent strabismus of both eyes, right more than left. Paralysis of both sixth nerves is complete. Slight ptosis of right eyelid. Right side of face flattened; mobility unimpaired; sensation natural. Food lodges between teeth and cheeks. Deglutition slow; cannot swallow liquids without coughing. Speech deliberate but distinct. No paralysis of arms. When put on her feet, sways about, and is apt to fall backwards; walks with a tottering gait. Legs muscular and well nourished. Forehead prominent; head otherwise natural. Sight and hearing unaffected. Double optic neuritis.

The mother's account is that the child has never been strong, and was always weak on her legs. Never had any chest affection. Four months ago she commenced to vomit. The vomiting was accompanied by frontal headache, and sometimes continued the whole day. At the same time the squint appeared, and has gradually increased since. There has been no loss of intelligence, but the child has been drowsy the last four weeks. Her walk has been unsteady the last six weeks, and she has frequently fallen forwards. There has been no wasting. Has complained of pain in the lumbar region the last six weeks.

Swallowing has been slow and difficult. The vomiting still continues.

The point whether the staggering were paralytic or vertiginous could not be determined, on account of the child not understanding the meaning of the word "giddy." The diagnosis consequently lay between cerebellar and pons disease.

For the next three or four days there was no material change in her condition.

May 1.—Caught cold and has a cough. Intellect weak. Mucus hangs about her mouth as if she could not cough it out.

May 7.—Swallows liquids with considerable difficulty and coughing; none returns through the nose. Saliva runs from her mouth. Has become more drowsy and stupid. Takes no interest in anything around her. Respiration snoring. Makes no attempt to expectorate, mucus has to be wiped out of the throat. Has only spoken once to-day. No paralysis of limbs. Extreme cyanosis has appeared. She lies in a condition of general nervous failure. Death occurred in the afternoon from gradual asphyxia.

Examination twenty-one hours after death.—Body well nourished. All the organs healthy except the brain. Brain: membranes and sinuses natural. Ventricles filled with clear serum, causing flattening of convolutions. Sixth nerves clearly compressed between pons and basilar process. Both lobes of the pons greatly enlarged, right half more than left. Greatest transverse measurement of pons = $2\frac{1}{4}$ inches, antero-posterior = $1\frac{3}{4}$ inch, depth = $1\frac{1}{2}$ inch. The swelling is very translucent and colourless, involving the posterior part of both crura cerebri, and also the right half of the medulla down to the lowest part of the decussation; left half seems less affected. Right seventh nerve compressed by the swollen medulla. The membranes and blood vessels at these parts are much attenuated, but are otherwise natural. No softening of central parts of brain. The lining membrane of the ventricles seems natural. Corpora striata and thalami optici look healthy. Corpora quadrigemina remarkably flattened out. The floor of the fourth ventricle is spread out and enlarged, and is so much pushed backwards that the root of the velum interpositum is undoubtedly compressed. The enlargement of the parts mentioned is uniform and has not the appearance of a tumour. Cerebellum and remainder of brain natural.

Microscopical examination.—Different parts of the pons, medulla, and crura cerebri were examined, but as they presented the same characters, varying only in degree, one description may suffice for all. Chromic acid $\frac{1}{6}$ p. c. was used for harden-

ing. Some sections were stained with hæmatoxylin, others with carmine. To begin with the nerve tissue, the most obvious pathological change in every case was noticed in the case of the nerve fibres. The medullary sheath was absent in almost all the fibres, and was replaced by a clear round space marked out by the surrounding neuroglia. The contents of these spaces were not stained by either hæmatoxylin or carmine. This change in the nerve fibres was most marked in the pons and crura, less so in the medulla. In the pons and crura not one normal medullated nerve fibre was detected; whereas in the medulla several normal fibres were to be seen scattered among the affected fibres. In some places in the pons and crura, naked axis cylinders were seen lying in the meshwork of the neuroglia, but in nearly all cases the axis cylinders had disappeared together with their medullary sheaths. The ganglion cells did not seem to be much affected, excepting that they were deeply pigmented, and did not show well-marked processes. The blood-vessels themselves appeared healthy in structure, but a very remarkable fact was noticed in connection with the perivascular lymphatic sheaths of the capillaries and smallest veins. Outside the vascular wall irregular bulgings were seen following the course of the vessels. These bulgings were filled with clear fluid. In some cases they looked like large vesicles surrounded by a fine membrane, attached to the wall of the vein or capillary. In other places they followed the course of the vessel for some distance as irregular tubular sheaths. The existence of these perivascular lymphatics in the normal brain has been lately called in question by some authors, from the fact that they cannot be artificially injected. But I think there can be little doubt that the appearances above described were due to œdema or exudation from the small veins and capillaries into the surrounding perivascular sheaths. It should be mentioned that, in some cases, the neighbourhood of the smaller vessels was marked out by irregular strings of small cells, probably leucocytes. No accumulation of leucocytes was found in the perivascular sheaths. The neuroglia in every case had undergone a change. Instead of the finely granulated matrix found in the brain and spinal cord in the normal condition, there was seen here a distinct meshwork of delicate fibres, containing the clear spaces which represented the nerve fibres. The neuroglia was evidently swollen and abnormally distinct. In some sections the only obvious change was a small celled infiltration replacing the nerve tissue. The number of small cells varied in different sections. In some parts of a section nothing could be made out except small cells, with here and there large ganglion cells interspersed. In other parts the

small cells were less numerous, and spaces could be seen between them representing the nerve fibres. This small celled infiltration was not present in any one part exclusively, but seemed to be irregularly disseminated in small foci throughout the pons, crura, and medulla. This infiltration was most marked in the pons. No other pathological change could be detected in any of the parts.

It remains now to find the proximate cause of the great enlargement of these parts, and here there is considerable difficulty. It is obvious that the increase in size must have been caused either by hypertrophy of the nerve tissue, or by enlargement of the interstitial tissue or neuroglia. The nerve fibres have been shown to have undergone some extensive degeneration, so that we must look elsewhere for the cause of this enlargement. The neuroglia was undoubtedly more prominent and more distinctly fibrillated than it should be, and in some places seemed to have encroached on the nerve tissue.

But although this change was distinct enough, it is not sufficient to account for the amount of enlargement present. Considering the condition of the perivascular sheaths, it seems evident that considerable interstitial œdema was present. This would account for a certain amount of enlargement, though it is doubtful if it would explain it entirely.

Again, the small celled infiltration was not extensive enough to produce such swelling of the parts. This growth would seem to suggest something of the nature of a chronic inflammation, but what part it took in the disease must remain doubtful. However, from its limited extent it would seem to have been rather a secondary than the primary change; possibly it had some influence in the production of the œdema mentioned. One is almost driven to seek for the primary change in the nerve fibres themselves, and yet anything of the nature of a metamorphosis or infiltration of nerve fibres other than a fatty change, appears to be unknown.

It is certain that the enlargement was not caused by a new growth, such as a myxoma, but was due to a uniform and general swelling of the parts.

It is much to be regretted that more light cannot be thrown on the pathological anatomy of this very interesting case.

A CASE

OF

SPASMODIC MUSCULAR RIGIDITY WITH IDIOCY.

BY

JOHN ABERCROMBIE, M.B.

M. E., aged 3, admitted into Hope Ward, August 18, 1877, under the care of Dr. Black. The child is small for her age, and decidedly thin; there is no subcutaneous fat about her body anywhere. She cannot sit up at all, and on attempting to make her do so, her head falls to one or other side as if too heavy for her, and she sits just like an idiot child, as if there was no strength in her back. She does not make her wants known in any way. She is constantly agitated by spasmodic rigidity of one or more groups of muscles. Thus at one time the whole of one side of the body will be affected, the leg being flexed upon the thigh, and the toes flexed upon the sole of the foot, the arm at the same time being rigidly extended with outstretched fingers, or the forearm flexed on the arm, with the fist clenched, thumb innermost, the head being thrown back on the neck, and the mouth drawn to the affected side.

This state of tonic spasm will last perhaps a quarter of a minute, and then pass off completely as suddenly as it came. This hemiplegic variety is not common, and much more often the rigidity is confined to one extremity or to one side of the face only, or there may be opisthotonic contraction of the head upon the neck alone, the rest of the body being quite flaccid. When this happens there is generally an expression of distress. Sometimes both arms are affected alone, or sometimes both legs

are affected, and are then in a state of rigid extension, with the feet in the position of equino-varus, exhibiting a marked tendency to cross, quite similarly to the cases recorded by Dr. Gee in another part of this volume. On making the child attempt to stand, this rigid extension of the legs with a tendency to cross is very marked. There is no spinal epilepsy. The rigidity can always be overcome by the exercise of a little force. The period at which these spasms succeed each other is very variable, sometimes scarcely half a minute elapses between the termination of one spasm and the commencement of another; at others there may be an interval of even a quarter of an hour. In the intervals between the spasms the muscles are all quite flaccid, nor can a spasm be brought on by manipulation. The muscles are very well developed all over the body, and this is particularly noticeable owing to the absence of subcutaneous fat. During sleep and under the influence of chloroform the muscles are everywhere quite flaccid. The muscles all act well to the induced current, none of them are markedly sluggish, nor on the other hand do any of them seem too irritable.

Her head is not small, and the anterior fontanelle is not quite closed. Her pupils are equal and of natural size; there is no strabismus or ptosis; she appears to be able to see well, and can follow a rapidly moving object with her eyes. The bridge of her nose is not flat. There are no scars about her mouth; she has all her teeth, and they are regular and good. The arch of her palate is rather high. She does not make any attempt to speak. There is no otorrhœa, it seems as if she could hear when her name is called out, but she shows no sign of understanding what is said to her. The cutaneous sensibility appears to be good. The functions of circulation and respiration are naturally performed. Physical examination of the chest and abdomen reveals nothing amiss. The ribs are not beaded, nor are the ends of the long bones enlarged.

This child was born at the full time, and was considered to be a fine child. She never evinced any sign of syphilitic taint. She was brought up by hand; the mother never tried to suckle her owing to some affection of her breasts. Up to the age of nine months she was considered a fine child, but at this age the mother first noticed she could not sit up, and after this date she observed the rigidity of her muscles already described. At about the age of eleven months she had a fit, and continued to have fits after this nearly every day during the whole period of dentition. She cut her first tooth at thirteen months old; since she finished cutting her teeth she has had no more fits. She has never been able to take solid food, and is always fed with a spoon.

Her mother has had two other children, both older than this one, and both are alive and well. She has had no miscarriages. The mother's father was subject to epileptic fits; there is no other family history of nervous affection. No history of phthisis.

This case is brought forward more with a view to putting on record a group of clinical symptoms of certainly very rare occurrence, than with any hope of adding anything to the physiology or pathology of the nerve centres. Notwithstanding the great advances that have been made during the last few years as to the pathogony of the nervous system, I am not aware of any published observation that would throw light on this case.

In the absence, then, of any direct knowledge, it would be premature to speculate much on the cause of these muscular spasms. I am inclined, however, to the belief that the symptoms are due to some congenital defect of the brain, notwithstanding the fact of the mother's assertion that the child was quite well up to the age of nine months. The absence of microcephalus will doubtless be held to militate against this view. But this point must not be too much insisted upon, for there can be no question as to the child's idiocy. Does it seem unreasonable to believe that the arrest of development, or the want of it, which has resulted in this state of idiocy, may also be the cause of some interruption between the brain proper (meaning that portion of the cerebral hemispheres wherein lies the physical representative of the will), and the motor nerve centres, resulting in a total loss of power over the voluntary muscles, and giving rise instead to irregular spasmodic discharges of nerve force?

The only other feature in the case which calls for observation is that, notwithstanding the loss of power over all the voluntary muscles of the body, the muscles of the eyeballs, of expression, and the depressors of the lower jaw are still under the control of the patient. These muscles are, however, the ones which almost always do escape, and in asylums it is by no means rare to find patients who, though paralysed in all other respects, are yet able to put in action all the muscles of the eyeball and those of expression, and to open their mouths for their food, though they have lost all power over the muscles of mastication, and so can only perform the automatic movements of deglutition.

A CASE

OF

PERFORATING ULCER OF THE SCLEROTIC.

BY

S. D. DARBISHIRE, M.B.

Florence A., aged 5 years, was admitted into Radcliffe Ward, under Dr. Southey, on May 28, 1877, with the diagnosis of diphtheria (?), scarlatina anginosa (?).

She was an ill-nourished child, ill-cared for and dirty. She had a purulent discharge from both eyes, with great photophobia; both conjunctivæ were injected, and there was an ulcer on the left cornea. A copious discharge flowed from the nostrils, which were nearly blocked up, and the lips were cracked and excoriated. There was no sore throat, but the fauces and pharynx were dirty with adherent mucus. She had no particular cough, but had frequent sneezing.

She had a pustular rash scattered irregularly over her head, face, chest, and upper extremities. The pustules on the head resembled those of impetigo; on the chest, face, and arms they were larger, and gave place to ulcers like those after a burn or a pemphigus bleb. Besides several of these ulcers on the arms, chest, and face, of the size of a threepenny bit, there was a large one an inch in diameter on the anterior fold of the left axilla, and another on the palmar surface of the second finger of the right hand, exposing the flexor tendons, and reaching from the first to the second fold. Both these ulcers were foul, with grey and indolent bases; the edges of the one on the axillary fold were sharply cut and excavated. Temp. 3 P.M., 102·5°; at 10 P.M., 102·8°.

The history which the mother gave was that the child had had whooping-cough at the end of March, which left persistent bronchial catarrh behind it; that the sores appeared four weeks

ago, and the eyes became affected two weeks before admission. There were ten other children in the family, none in good health; one, the mother said, was in the same condition as the patient in the Hospital. The child's general condition was wretched; she looked as if she had suffered from want of food, fresh air, light, and cleanliness for many weeks.

She was ordered dietum lactis, with a pint of beef-tea, half a pint of essence of beef and two eggs, four ounces of wine, and as much extra milk as she cared to drink. Her eyes were to be washed out frequently with tepid water; Lotio boracis c. pot. chlorat. was ordered for her mouth; and a draught containing Pot. chlorat. and sod. biborat., five grains of each, every four hours, was prescribed. The ulcers on the chest, face, and finger to be poulticed.

May 29.—Temp. 102°. Had passed a good night; takes her food well. Urine dirty amber; acid; 1025 sp. gr.; no albumen. Bowels not opened; tongue very furred.

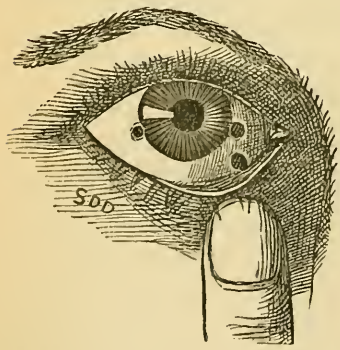
May 30.—Temp. 102·4°. Pulse, 140. Respiration, 36.

May 31.—Temp. 103°. Bowels open.

June 1.—Temp. 103°. Ordered Quin. sulphat., gr. i.; tr. ferri sesquichlor. ♏ x.; syrupi, ℥i.; aquæ distillat., ℥i., three times a day. The poultices to be left off, and the ulcers to be dressed with lint soaked in zinc lotion. The finger was supported by a splint.

The child improved in general condition under the treatment of good nourishment and cleanliness, so that by June 9 her temperature was normal, the ulcers were healing rapidly, no more pustules had appeared on the head or elsewhere, and she was gaining flesh.

The left eye, however, did not improve; the ulcer of the cornea, noticed on admission, had increased in size and depth; it now extended from the centre to the circumference, occupying a full quadrant. The right eye was better.



On June 13, attention was drawn to the right eye by some increase of lachrymation and photophobia, when for the first time three perforations of the sclerotic were noticed, situated as in the woodcut. The one on the equator to the outer side encroached slightly on the cornea, and the edges shelved down to the base, which, as in the others, was of a slaty-grey colour. In the lower one,

on the inner side, which was covered by the lower lid, the edges were very sharply cut, and the dark choroid bulged distinctly through the opening in the sclerotic.

The after history of the case may be told in a few words. The left cornea gave way on June 20; after the application of pressure with a pad and bandage for seven days, paracentesis of the anterior chamber was performed on June 28, and the pressure continued. The perforating ulcers of the right sclerotic glazed over and contracted slightly.

When discharged on August 11, she was in very good general health; the functions of the right eye were in all respects normal, but the left eye was inflamed and irritable, and threatened to give rise to sympathetic ophthalmia of the right. On leaving Radcliffe Ward, she was admitted into Alexandra Ward, under Mr. Vernon, who performed abscission of the left eye. She finally left the Hospital with a good moveable stump in place of her left eye; the right, which still showed the scars of the sclerotic ulcers, remaining perfectly quiet.

The interest of the case depends upon these perforations of the sclerotic, which were first observed on June 13, a fortnight after admission. From the appearances which they presented, it is probable that they had existed for some days before they were noticed, for there was nothing active in the way of ulceration going on in them; and to judge from the subsequent course pursued by them while under observation, it was evident that the outer ulcer with the shelving edges was already undergoing a reparative process when seen. Their general appearance suggested a suspicion of their having been caused by the application of some caustic in the solid form; but on inquiry, both the mother and the medical attendant who had seen the child before admission into the Hospital gave their assurance that the only application used for her eyes had been a lotion of sulphate of zinc, gr. ii. to the ounce of water.

Were they, then, a local manifestation in an unusual situation of the general pemphigoid rash?

ON THE

ALBUMINOUS SUBSTANCES WHICH OCCUR IN THE URINE IN ALBUMINURIA.

BY

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

AND

D'ARCY POWER.

The advances made by physiological chemistry during late years have been so rapid that it has been exceedingly difficult, indeed almost impossible, for any one busily engaged in practice to keep up with them. As the subject of albuminuria is one of great practical importance, and the discrimination of its different varieties may greatly modify the prognosis and treatment in any given case, we consider it advisable to begin this paper by a few words on the general chemistry of albuminous substances before proceeding to the special subject of the paper. For some readers this may be useful; for others it may be superfluous, but it is easy for these to pass it over. White of egg, or albumen, has given its name to the substances which more or less resemble it in their chemical reactions, and they are therefore termed albuminous. But white of egg contains other substances besides the constituent which gives its characteristic properties to it and forms the chief part of its bulk. To distinguish between the crude white of egg, or albumen, and its chief constituent, egg-albumin, the former is spelt with an *e*, albumen, and the later with an *i*, albumin.

The class of albuminous substances contains bodies which differ so much from each other that it has been found necessary to subdivide it into several groups. Hoppe-Seyler gives eight groups, which are—I. Albumins, II. Globulins, III. Fibrins,

IV. Albuminates, V. Acid Albumins or Syntonin, VI. Amyloid, VII. Coagulated Albuminous Bodies, VIII. Peptones.

Two of these groups, viz., fibrins and amyloid substance, are only met with in the solid form, so they do not concern us in an inquiry into the nature of the albuminous substances which occur in solution in the urine. A third group, viz., coagulated albuminous bodies, also occurs only in the solid form. Five groups, therefore, remain which occur in solution, and may consequently appear in the urine. These are albumins, globulins, albuminates, acid-albumins, and peptones. We may further classify these five groups into three divisions. *First,* Albuminous bodies in what, for convenience' sake, although not perhaps strictly accurately, we may term their natural condition, in which they are coagulated by boiling. This division contains albumins and globulins.

Second, Albuminous bodies in combination with acids and alkalis, and not coagulated by boiling. Albumins and globulins both combine with mineral acids and alkalis to form acid-albumins and alkali-albumins, or alkali-albuminates, as they are generally called. Thus, if we take a little white of egg dissolved in a quantity of water, we get a solution which is coagulated by boiling. But if we first add to it some very dilute nitric or hydrochloric acid (*e.g.*, its own bulk of four parts commercial acid in 1000 of water), and then heat it, we may boil it as much as we please, but no coagulum will form. The albumin has combined with the acid and formed acid-albumin, which is not coagulated by heat. This is the reason why carelessness in washing out test-tubes sometimes causes the presence of albumin in urine to be overlooked. Let us suppose that a man tests urine in the usual way, either by boiling and adding nitric acid afterwards, or by nitric acid alone, and afterwards throws out the mixture of urine and acid. He then pours some fresh urine into the tube without washing it and proceeds to boil. The urine remains clear, and he supposes it to be free from albumin, yet it may be highly albuminous. For the acid diluted by the urine first tested clings to the side of the tube, and being thus heated with the second urine, gradually converts it into acid-albumin; and by the time the coagulating point of the unchanged albumin is reached, there is no longer any to coagulate, the whole having been changed into acid-albumin. By boiling a solution of white of egg or some albuminous urine with liquor potassæ, instead of dilute acid, the albumin in either solution will be converted into alkali-albumin, or alkali-albuminate, and will not be coagulated by boiling. By its conversion into acid-albumin or alkali-albumin, ordinary albumin undergoes another change besides the loss of its coagulability on boiling, for it also loses its solubility

in water. White of egg, or the dried albumin from serum or urine, may be dissolved in water and give a neutral solution, but after it has been changed into acid-albumin or alkali-albumin it becomes insoluble in water, and is therefore precipitated from its acid or alkaline solutions by neutralising them. When the point of neutralisation is passed, and the solutions rendered alkaline by alkalis or their carbonates, or acid by mineral acids, the precipitate is redissolved, but will again be thrown down by neutralising. But if acetic acid be used instead of a mineral acid to neutralise a solution of alkali-albuminate, the precipitate is not dissolved by a slight excess of acid. Indeed, when sodium phosphate is present in the solution, alkali-albumin is not precipitated by exact neutralisation, and the precipitate only falls after the liquid has been rendered acid. When we wish, therefore, to separate alkali-albumin from a liquid, we acidulate with acetic acid. By then boiling we can precipitate both the ordinary albumin and the alkali-albumin from a fluid which contains them, while if we boiled without previously adding acetic acid, the ordinary albumin only would be coagulated, and on removing it by filtration the fluid would be found to contain alkali-albuminate. But besides its use in precipitating alkali-albuminate, acetic acid possesses the power of causing ordinary albumin to coagulate more readily on the application of heat, and its addition to any fluid from which we wish to separate albumin thus serves a double purpose.

The *third* section into which we have divided soluble albuminous bodies contains only one group, that of peptones. These are albuminous bodies so much altered by the process of digestion that they are neither coagulated by heat nor precipitated by neutralisation. They are, however, precipitated by alcohol.

The presence of albumin in the urine is universally acknowledged to be a morbid condition, but its causation and significance are very varied. At one time it is a symptom of the gravest importance, at another it may be of very little consequence. This fact is of itself sufficient to show that under the general term albuminuria many dissimilar conditions are grouped, Some of these have already been dissociated, and we recognise the distinction between the albuminuria of Bright's disease and that dependent on cardiac lesions. But besides these there are probably other forms of albuminuria, less common and less important, but yet deserving of more attention than they have hitherto received in respect to diagnosis, treatment, and prognosis. It was with the view of attempting to distinguish these, as well as with the hope of gaining some new insight into the ordinary forms of albuminuria, that we began our present

research. The great differences which are observed in the behaviour of albuminous urine when boiled or treated with nitric acid have led medical men to recognise that the albuminous bodies occurring in urine are not always the same; that two or more kinds of albuminous bodies may sometimes be present in the urine at once. Lehmann showed that paraglobulin is generally present in albuminous urine along with serum-albumin, and his results were confirmed and extended by Edlefsen and Senator. These observers dealt chiefly, however, with the albuminous bodies normally present in the blood, although Senator observed the presence of peptones. It is to the classic researches of Stockvis that we owe the first clear demonstration that albuminous bodies may be absorbed from the stomach and intestines, and excreted unchanged in the urine. Numerous experiments showed him that the serum of blood and the albumen usually excreted by the kidneys in Bright's disease were identical, and that when either of them was injected under the skin, or directly into the vessels of the animal, provided that the experiment was conducted in such a manner as not to disturb the circulation, the urine remained quite free from albumin, the healthy kidneys apparently refusing to let the albumin pass through them. But when egg-albumin, or Bence-Jones's albumin (a curious kind of albumin obtained from the urine in cases of osteo-malachia) was used, the result was very different, for both of these seemed to pass readily through the kidneys, and appeared again apparently unchanged in the urine. When taken into the stomach, raw eggs, as every one knows, are digested, and egg-albumin does not appear in the urine as a rule; but if the quantity of eggs has been too great for the digestive powers, the albumin is absorbed, and appears in the urine. Both egg-albumin and Bence-Jones's albumin are absorbed from the rectum, and appear in the urine. Nor is it only undigested albuminous substances which are thus absorbed and excreted. Claude Bernard observed that after partaking of a quantity of *cooked* eggs, his urine became albuminous. The coagulated albumen of cooked eggs could obviously not be absorbed without undergoing some previous change. But Kühne has found that the pancreatic juice, before converting coagulated albuminous bodies into peptones, seems to change coagulated albuminous bodies into something resembling their raw condition before converting them into peptones. In Bernard's observation the cooked eggs which he swallowed seem to have undergone this change, and then been absorbed in the same way as raw eggs would have been. The fact that absorption of albuminous substances does take place from the intestine makes

it appear extraordinary that albumin is not more frequently found in the urine during digestion, and one can only suppose the reason to be that it is only when the digestive powers are overtaxed, as by swallowing many raw eggs together, or deranged so as to digest the food partially but not completely, that such an event occurs. In a clinical lecture, published in the "Medical Times and Gazette" for April 10, 1852, the late Dr. Parkes noticed that in cases of albuminuria the albumin was much increased after meals, and he ventured the hypothesis that the albumin was of a different quality, as well as increased in quantity. In the same journal, April 22, 1854, he discussed the origin of this increase, and distinguished it by the name of food-albuminuria. This food-albuminuria he considered was not due to congestion of the kidneys during digestion, for the water of the urine is often diminished, and the solids do not increase in proportion to the albumin. He therefore thought it might be due to albumin not being altered in the stomach and liver, and therefore being eliminated like white of egg. He called attention to the fact that the antecedents of Bright's disease are often such as to impair the functions of the stomach and liver, and that dyspeptic symptoms often appear before renal. In his work on the urine he also quotes the case described by Christison of a young man in whom cheese always produced temporary albuminuria, and who afterwards died of Bright's disease.

Similar observations to those of Parkes were made about the same time as his by Gubler, who communicated to the Société de Biologie, August 6, 1853, his observation that the amount of albumin was increased during digestion. He proceeded to try the effects of various diets on the patients, and found that, with an exclusively vegetable diet, the albumin sank to a minimum ; that it reached its maximum on an exclusively albuminous diet, and was intermediate in quantity when the diet was mixed (article in Dictionnaire Encyclopédie des Sciences Médicales, 1865, tom. ii. p. 447).

This subject was again taken up by Dr. Pavy, who confirmed Parkes' results regarding the increase of albumin in the urine during digestion, and tried to ascertain experimentally whether or not Parkes' supposition were correct that the albumin found in the urine varied in character as well as in quantity at different times. It occurred to him that its occasional presence in the urine might be due to its diffusibility being greater at one time than another. He therefore employed a dialysing apparatus to distinguish between the albumins, with the result of showing that very considerable differences exist in the readiness with

which different specimens of albuminous urine pass through animal membranes. We quote the following passage from his Gulstonian Lectures for 1862 (Lancet, May 23, 1863): "On submitting some specimens of albuminous urine to dialysis, I encountered one where the albumin passed in considerable quantity. The urine was derived from a patient affected with phthisis, who never had experienced any symptom of dropsy. It was highly charged with albumin, and on being submitted to dialysis, using vegetable parchment as a septum, the albumen passed to such an extent that the diffusate, after twenty-four hours (distilled water had been placed on the other side of the membrane), gave a pretty copious precipitate with heat and nitric acid, as also with the yellow prussiate of potash and acetic acid test. It was noticed, as happens with some specimens of albuminous urine, that the precipitate produced in the diffusate, by the addition of a small quantity of nitric acid, was redissolved on agitation. A considerable excess of nitric acid was required to throw down a permanent precipitate. The same result was obtained on several occasions, and no idea could be entertained as to any imperfection in the dialyser, because the same one was used for blood and other specimens of albuminous urine, but albumin in neither case passed in twenty-four hours to any sensible extent. The patient left the hospital, and was re-admitted six months later almost in a moribund state. His urine was still highly albuminous, but, curiously enough, now gave scarcely any positive result on being submitted to diffusion. Dialysed for forty-eight hours with a septum of vegetable parchment, the diffusate yielded only the slightest turbidity on being tested for albumen. Although I have met," he continues, "with differences in other cases, still with the specimens of urine I have as yet examined I have never encountered one where the albumin diffused to an extent at all comparable to that in the case I have just mentioned." Interesting as these observations of Pavy were, they do not seem to have been taken up and extended, probably because the method of determining the nature of the albumin was too troublesome to allow of its being applied readily in the sick chamber or hospital ward. In our present research we have endeavoured to distinguish the different albumins in urine by determining their coagulating points, because we wished to employ a method which could be used at the bedside with such ease that, if it gave any useful indication at all, it might be universally employed.

The method simply consists in holding a thermometer in the urine while it is being very gently heated in a test-tube over a spirit lamp, and noting the temperature at which the urine

begins to grow milky from the commencing coagulation. Instead of holding the thermometer with the fingers, it may be fixed in the test-tube by means of a conical india-rubber stopper, with a hole in the middle through which the thermometer passes. As one is apt to break the thermometer in pushing it into and drawing it out of the hole, it is well to split the stopper to its middle along its whole length. By opening the slit the thermometer can be put in and taken out, or its position altered, with the greatest ease, and the conical form of the stopper makes it fit a test-tube of any size. The apparatus we employed was somewhat more complicated, for instead of heating the urine in a test-tube directly over a flame, we suspended the test-tube in which it was contained in a beaker of water over a lamp, so that the urine was thus very equally and gradually warmed, and the temperature of coagulation exactly ascertained.

The first question which we tried to answer by this method was, Does the coagulating point of the albumin in albuminuria vary much?

From the following examples it will be seen that it does.

I. R. A., case of intermittent albuminuria. Coagulating point of one specimen of urine, 173° F.

II. J. V., albuminuria from pregnancy. One specimen coagulated at 150° F.

III. H. H., waxy kidney. One specimen coagulated at 164° F.

IV. Chronic Bright's disease. One specimen coagulated at 180° F.

V. Chronic Bright's disease. One specimen coagulated at 144° F.

But the coagulating point varies not only in different patients but at different times in the same patient. Thus in the case of H. H., although the coagulating point was usually about 164° F., it sank to 152° on one occasion, and rose to 171° F. on another. It is well known that variations in the acidity and in the amount of neutral salts of any albuminous solution greatly alter the coagulating point of the albumin. Besides this, however, it seemed to us that the proportion of urea in the urine might influence the coagulating point of albumin, and the following experiments were tried to deterimne this point as well as the influence of uric acid.

Influence of Urea on the Coagulating Point of Serum-Albumin.

A solution was made of the dried serum of blood (serum exsiccatum), prepared by the evaporation of fresh serum at a temperature of 130° to 140° F. by Mr. Fore of Birkenhead at the

suggestion of Dr. Vacher.[1] One part of this was dissolved in 2000 of water, and portions of the solution were mixed with their own bulk of solutions of urea of different strengths. The coagulating point was then noted.

The effect of the urea in altering the temperature of coagulation is apparent from the following table:—

Influence of Urea on the Coagulating Point of Serum-Albumin.

Strength of Solution of Serum-Albumin.	Strength of Solution of Urea.	Temperature of Coagulation 154°–158° F. without admixture.
1 in 2000.	50 per cent.	No coagulation.
Do.	20 ,,	168°–172°
Do.	15 ,,	166°–172°
Do.	10 ,,	166°–170°
Do.	5 ,,	162°–168°
Do.	1 ,,	168°
Do.	$\frac{4}{5}$,,	164°–166°
Do.	$\frac{1}{2}$,,	162°
Do.	$\frac{2}{5}$,,	160°
Do.	$\frac{1}{5}$,,	160°

Influence of Urea on the Coagulation Point of Egg-Albumin.

Strength of Solution of Egg-Albumin.	Strength of Solution of Urea.	Temperature of Coagulation 136°–140° F. without admixture.
1 in 20.	50 per cent.	No coagulation.
Do.	20 ,,	140°
Do.	10 ,,	140°
Do.	5 ,,	132°–134°

In another solution of egg-albumin of similar strength the coagulating point was 138°–142° F., and admixture with an equal volume of 5 per cent. urea solution did not alter it.

It is evident from a consideration of the figures just given that although variations of a few degrees may occur in the observations without any apparent cause, except imperfection of the method of experiment, yet as a general rule the presence of urea in solution raises in a very marked degree the coagulating point of albumin, and when the quantity of urea reaches 25 per cent. of the solution (*i.e.*, of the mixed solutions employed), the coagu-

[1] Serum Sanguinis Exsiccatum, by F. Vacher. "Practitioner," vol. xvii. p. 433.

lation both of serum-albumin and egg-albumin is prevented. Egg-albumin was much less affected than serum-albumin, but this might be due to the different strength of the solution, and not to any difference in the reaction of the albuminous substances themselves.

The effect of uric acid on the coagulation of serum-albumin and egg-albumin was tried in a similar manner. We here give the results—

Influence of Uric Acid on the Coagulation of Serum-Albumin.

Strength of Solution of Serum-Albumin.	Strength of Solution of Uric Acid.	Temperature of Coagulation 160° F. without admixture.
1 in 2000.	Saturated.	144°
Do.	Do.	146°–150°

Uric acid is thus seen to have a depressing effect on the temperature of coagulation of serum-albumin.

Similar experiments made with egg-albumin seemed to show that it raised instead of lowered the temperature of coagulation, but this seems so doubtful that we must repeat our experiments on this point.

We may sum up shortly the results of this series of experiments, at least as far as regards serum-albumin, and also the results of previous observations on the effect of acids and salts, by saying that—

The coagulating point is raised by urea.

The coagulating point is lowered by uric acid, other acids, and neutral salts.

It is therefore evident that the urea will more or less counteract the effect of the acid and salts in the urine upon the coagulating point of the albumin it contains and *vice versa*, so that at one time they may completely neutralise one another, and the temperature of coagulation will then be the same as if neither were present, while at another the temperature may be either considerably above or considerably below that at which the albumin would be precipitated from a purely aqueous solution.

Effect of Dilution on the Coagulation of Albuminous Urine.

In order to get rid, to some extent at least, of the effect of the salts and of the urea on the coagulating point, we diluted the urine with water until it had a specific gravity of 1005. This altered the coagulating point, sometimes raising it, at other times

depressing it, but it did not render the coagulating point constant, as will be evident from the following table:—

Case.	Ward.	Date.	Before Dilution.			After Dilution.	
			Sp. Gr.	Reaction.	Coagulating Point.	Sp. Gr.	Coagulating Point.
Gollop,	John	Sept. 5	1019	...	131°–140°	1005	132°–137°
Chronic		,, 7	1020	...	124°–126°	1005	130°–135°
Bright's disease.		,, 8	1021	...	128°–132°	1005	132°
Hill,	John	,, 5	1016	neutral	164°–170°	1005	164°–170°
Waxy		,, 8	1015	acid	160°–166°	1005	166°–173°
kidney.		,, 20	1015	acid	162°–166°	1005	154°
Juniper,	Mark		1015	alkaline	162°	1005	152°–160°
Nephritis							
Hunt,	Luke		1019	acid	148°	1005	136°
Nephritis							
Slater,	Matthew		1032	acid	142°–148°	1005	142°
Bright's disease.							
Cane,	Matthew		1017	acid	134°–138°	1005	148°–150°
Bright's disease.							

Experiments on the Coagulating Point of the Products of Pancreatic Digestion.

As the coagulating point of the albumin in urine varied considerably notwithstanding our efforts to get rid of the effect of urea and neutral salts by dilution, it occurred to us that it would be advisable to ascertain whether the coagulating point of the soluble albuminous substances produced by the action of pancreatic juice upon solid insoluble albuminous bodies varied at different stages of the digestive process. For it is evident that if this should be the case, and these soluble albuminous bodies should be absorbed into the blood or excreted by the kidneys in the same way as raw white of egg, they would cause the temperature at which coagulation occurred in the urine to vary, apart from any influence of urea, uric acid, or salts, contained in it. The products of pancreatic digestion are in this respect more important than those of gastric digestion, inasmuch as the gastric juice can only act so long as the food continues in the stomach, its power being destroyed by admixture with the bile in the duodenum, while the pancreatic digestion may continue all the way down the intestine, where the greatest facilities for absorption are to be found, much greater than in the stomach. In order to ascertain this point, fibrin obtained from pig's blood was digested with water and some pancreatine (prepared by

Messrs Savory and Moore, and previously ascertained to be active), in a water-bath for about three hours at a temperature of 100°–105° F., until the fibrin had disappeared. The solution thus obtained was filtered, and after standing twenty-four hours, the temperature of coagulation was observed.

Undiluted the solution coagulated at 142°, 138°–142°, 128°–134°, 134°, varying on each separate occasion. The true point appears to be 128°–134° F.

When the solution was diluted with its own bulk of water, coagulation occurred at 136°–140° F. and at 144° F.

The cause of this variability in the coagulating point probably is that during the process of heating more or less digestion of the albumin by the pancreatine occurs, so that the coagulating point is more or less raised in the same way as occurs when the solution stands for a time at ordinary temperatures. When this solution was examined after standing forty-eight hours, indol had been produced to a considerable extent, and the coagulating point of the filtered solution had risen to 168° F.

Another portion of fibrin was digested with pancreatine, and a portion of the solution tested after an hour's digestion in the water-bath. Coagulation occurred at 146°–150° F. A portion was again tested after two hours and fifty minutes' digestion. The coagulating point had fallen to 138°–140° F.

Effect of Urea and Uric Acid on the Coagulation of the Products of Pancreatic Digestion.

The first solution already mentioned, when mixed with its own bulk of water, coagulated at 136°–140°; with its own bulk of 25 per cent. solution of urea, at 160°–165°; with its own bulk of 10 per cent. solution of urea, at 140°–148°; with its own bulk of 5 per cent. solution of urea, at 138°–144°.

When mixed with its own bulk of a saturated solution of uric acid the coagulating point was lowered to 128°–130° F. The second solution, made by digesting fibrin for three hours, only when diluted with its own bulk of water coagulated at 138°–142° F.; with its own bulk of a 5 per cent. urea solution, at 138°–142°; with its own bulk of a half saturated solution of uric acid, at 130°–134° F. The effect of urea and uric acid upon the coagulating point of the products of pancreatic digestion is thus seen to be similar to that in serum- and egg-albumin. But besides this, we notice that the coagulating point of these products is much lower than that of serum-albumin (about 160° F.). On looking back to the table already given of the temperature of coagulation after diluting, we observe that the numbers in it may be arranged in three groups; the lower

ranging from about 130° to 137° F., or about the coagulating point of the products of digestion; the upper from about 160° to 173° F., or about the coagulating point of serum-albumin; and the other containing intermediate numbers. In the case of Gollop (*vide* p. 304), the coagulating points are seen to belong to the lower, and in that of Hill (*vide* p. 308) to the upper group.

This fact seems to point to the existence of two separate albuminous bodies or classes of bodies in these urines, the albuminous products of digestion and serum-albumin, or a mixture of these. It must be remembered also that although the earlier products of pancreatic digestion coagulate about 130° F., yet after standing the coagulating point rose to 168° F. This corresponds closely with the coagulating point of the albumin in the urine of a patient in whom the appearance of albumin and disturbances of digestion were so closely associated that it seemed probable that the former was in great part at least due to the latter. In this case, which has been already fully described by one of us in another place,[1] the coagulating point was 173° F., and on diluting the urine with its own bulk of water, the coagulating point fell to 171° F.

Effect of Food in causing Albumin to appear in the Urine.

In two cases albumin was observed in the urine after food, while it was absent during fasting. In one of these cases its coagulating point was 148°–152°, in the other it was 171°–174°. In both of these urines no coagulation occurred when they were diluted to sp. gr. 1005, and then heated (*vide* table on next page).

We here append short notes of Mayhew's case.

Ambrose Mayhew, aged 28, admitted September 12, 1877. He was in India in the army, and was invalided home in April last.

Since October 1876 he has suffered from frequent desire to stool with tenesmus, and almost always passes a little blood. For the first five months he had pain and tenderness in the hepatic region, but never was jaundiced.

Since he was first taken ill he has never got rid of this complaint.

On admission he was much emaciated, but there were no physical signs of disease, except slight tenderness in the region of the liver, which can just be felt below the ribs.

His bowels were opened several times in the twenty-four hours; motions scanty, dark, containing a little blood and mucus. He was kept in bed, and put upon milk diet, with 8 grs. of

[1] Arsenic in Albuminuria, by T. Lauder Brunton, M.D. "Practitioner," June 1877, vol. xviii. p. 427.

Effect of Food in causing Albumin to appear in the Urine.

Name.	Ward.	Disease.	Coagulation begins.	Observation ended.	Time.	Quantity	Sp. Gr.	Reaction.	Remarks.	Date.
Glasgow	Henry	Waxy kidney	No coagulation	..	Before dinner	...	1013	Slightly acid	Urine clear and of a normal colour	Sept. 11
			No coagulation	...	,,	...	1005	...		
			148°	152°	After dinner	...	1015	More strongly acid	Urine more turbid; the coagulation exceedingly slight	...
			No coagulation	...	,,	...	1005	...	The urine became milky	...
Mayhew	Matthew	Dysentery	No coagulation	...	Before dinner	...	1017	Acid	The urine was clear and light coloured; it was heated to 190° F.	Sept. 18
			No coagulation	...	,,	...	1005	...	No coagulation, even on boiling; urine diluted	...
			171°	174°	After dinner	...	1022	Slightly acid	Urine clear and light coloured	...
			No coagulation	...	,,	...	1005	...	No coagulation, even on boiling; urine diluted	...
			No coagulation	...	Before dinner	...	1029	Acid	Thick chocolate colour, filters clear	...
			176°	...	After dinner	...	1025	Acid		
			No coagulation	...	,,	...	1005	...	Slight turbidity at 176° F.; urine light-coloured and slightly turbid	Sept. 24

Dover's powder at night, and an occasional small dose of castor oil (2 drachms every second or third day).

At the end of about a week, he was put upon fish, and a few days later was allowed to get up in the evening.

He was discharged on October 24, having improved very much in general health, and gained a little in weight.

His bowels acted about three times a day; the motions were less scanty, always bilious, and containing mucus, but there was no longer any blood.

Effect of Food in Altering the Coagulating Point of the Urine in Cases of Albuminuria.

The following tables (pp. 298 and 299), as well as those of the urine of Gollop (p. 304) and Hill (p. 308), show that the urine passed after taking food usually has a lower coagulating point than the total urine passed during the twenty-four hours. This is not, however, invariable, the coagulating point being sometimes raised instead of lowered, as in Gollop's urine of September 19th, 21st, and 28th, and in Hill's of September 15th, when coagulation occurred in the urine of the twenty-four hours at 128°–131°, 132°–136°, and 136°–140°, and 163°–168°, and in the urine passed after dinner at 134°–136°, 136°–138°, 137°–144°, and 167°–170° respectively.

On the Presence of Peptones in the Urine.

A quantity of urine from Gollop, John Ward, whose case will afterwards be described, was acidulated with acetic acid boiled, thrown upon a filter. A very bulky precipitate of coagulated albuminous substances remained on the filter. To the filtrate alcohol was added in excess, and a copious white flocculent precipitate was thrown down. This precipitate when dried and removed from the filter formed a greyish powder, partially soluble in water, forming a somewhat milky solution. This gave the xanthoprotein reaction. With liquor potassæ and a very slight trace of copper sulphate it gave a faint rose colour. On the addition of a little more copper sulphate it became violet, and the violet deepened on heating. With more still the fluid became blue, the sulphate remaining dissolved. On boiling, a brown precipitate fell, and the fluid remained of a violet colour.

The solution gave no distinct precipitate with mercuric chloride, as peptones usually do.

These reactions appear to us to indicate either the presence of peptones or some other body closely connected with them and not yet investigated, and differing by its non-precipitation by mercuric chloride.

On Digestive Ferments in the Urine in Albuminuria.

It was shown several years ago by Brücke that pepsin was excreted in small quantities in the urine, and the same was observed by Cohnheim in regard to ptyalin. Supposing that by any cause the excretion of these ferments should become abnormally increased, it is probable that the digestive powers might become weakened, and that digestive disturbances such as are observed in albuminuria might result. Now Von Wittich [1] observed that fibrin has a very great attraction for pepsin and absorbs it most energetically. When put into water on one side of the parchment of a dialyzer, it causes pepsin to pass through more rapidly from the other side. An excess of fibrin will absorb from artificial gastric juice all the pepsin which has already digested a part of it, and when the undissolved fibrin is taken out and put into fresh acid, the pepsin it contains is generally sufficient to dissolve both it and additional fibrin added to it, while the liquid from which it has been removed has lost all peptic properties. In these experiments it is to the undissolved fibrin that the pepsin adheres, and not to the dissolved products of digestion, yet it seemed not impossible that albuminous bodies in their passage through the kidney might take along with them more of the digestive ferments than usually pass out of the body in the urine, and we therefore made the following experiments in order to ascertain the presence in the urine of pepsin and ptyalin, as described by Brücke and Cohnheim, and also to see whether other ferments, such as those of the pancreas and intestine, which are not usually present in the urine, occurred in albuminuria.

On the Presence of Pepsin.

Ten fluid ounces of urine from a case of chronic Bright's disease were mixed with twenty-eight ounces of strong methylated spirit. A copious precipitate of proteids occurred. After standing for two days the supernatant fluid was washed, decanted off, and the precipitate washed with absolute alcohol. The alcohol was then filtered off, and the precipitate immersed in glycerine, where it remained six days. It was then filtered, and the clear filtrate used for experiments.

To 10 c.c. of this clear glycerine extract 25 c.c. of a ·02 per cent. solution of hydrochloric acid was added, and a piece of boiled fibrin put into the solution. It was then kept for twenty-four hours in a water-bath at a temperature of 100° F., and the liquid tested for albuminous bodies in order to see whether any

[1] Von Wittich, Pflüger's Archiv f. Physiol., v. 435-469.

EFFECT OF FOOD IN ALTERING THE COAGULATING POINT OF ALBUMIN IN CASES OF ALBUMINURIA.

Name or No.	Ward.	Disease.	Coagulation begins.	Observation ended.	Time.	Quantity.	Sp. Gr.	Reaction.	Remarks.	Date.
No. 4	Hope	Phthisis albuminuria	169°	172°	Before dinner	...	1025	Acid	Urine clear and of moderate colour	Sept. 20
			174°	178°	,, ,,	...	1005	...		...
			167°	170°	After dinner	...	1023	Acid	Of a darker colour	...
			166°	170°	,, ,,	...	1005	...		...
No. 6	Hope	Chronic Bright	138°	142°	,, ,,	...	1019	Acid	Urine turbid; light coloured	Sept. 21
			142°	146°	,, ,,	...	1005	...		...
No. 17	Matthew	Chronic Bright	144°	148°	Before dinner	...	1014	Strongly acid	Light coloured	Sept. 19
			138°	146°	,, ,,	...	1005	...	The urine became slightly opalescent on diluting	...
			148°	150°	After dinner	...	1016	Very strongly acid	Rather darker than the preceding	...
No. 17	Luke	Chronic Bright	132°	134°	,, ,,	...	1005	...		...
			180°	184°	Before dinner	...	1015	Slightly acid	Urine light coloured; somewhat turbid; had stood for at least twenty-four hours	Sept. 13
			184°	...	,, ,,	...	1005	...		...
			164°	168°	,, ,,	...	1013	Neutral	Light coloured; not quite clear	...
			164°	170°	,, ,,	...	1005	...	Coagulation not well marked; this urine had also been kept at least twenty-four hours	...
			180°	182°	,, ,,	...	1015	Acid	The above urine was tested again after a lapse of twenty-four hours; the turbidity had increased, and it had a disagreeable smell	Sept. 14
			182°	186°	,, ,,	...	1005	...	Coagulation not well marked	...
			174°	179°	,, ,,	...	1018	...		Sept. 17
			...	...	,, ,,	...	1005	...	No perceptible coagulation occurred at 190° F.	...
			174°	176°	After dinner	2½ pts.	1012	Slightly acid	Urine clear, and of a very light colour	...
			171°	173°	,, ,,	...	1005	...	In each of the above cases the urine became cloudy at about 158° F., though the coagulation did not begin till a higher point was reached	...

No. 11	Hope	Chronic Bright	132°	138°	Before dinner	...	1005	...		Sept. 25
			132°	138°	,, ,,	...	1005	...		
			126°	128°	After dinner	...	1015	Acid	Became opalescent on diluting. *Cf.* John, No. 8	...
			136°	...	Before dinner	...	1016	Acid	The red colour disappears when the litmus paper dries. On dilating, the fluid becomes so milky that the coagulating point cannot be observed. The after-dinner urine is very thick; the thickness is not diminished by filtering	...
			120°	124°	After dinner	...	1015	Acid	... ...	Sept. 26
			110°–116°	120°	,, ,,	...	...	...	A slight coagulation occurred at 100° F., more at 131°-148°; a copious precipitate at 152°, and at a higher point the precipitate was flaky	Sept. 27
			110°	115°	Before dinner	...	1015	Acid	The urine was light coloured and turbid. At 188° F. the after-dinner urine became flaky. On diluting it was at first clear, but after standing a minute it became opalescent. On standing, the coagulated precipitates sank to the bottom, leaving a clear supernatant fluid	...
			128°	151°	After dinner	...	1016	Very acid	Excess of Na_2So_4 was added, and the mixture was boiled; the coagulated albumin was filtered off, and the filtrate was tested by the Xanthoproteic reaction for peptones; none however were found	...
			160°	166°	,, ,,	...	...	Acid		
			150°	154°	,, ,,	...	1005	...	This urine contained casts; it was very turbid and of a brown colour; after filtering it became clear, and was then seen dark coloured. The coagulation was exceedingly well marked, beginning at the surface at 160° F., and extending slowly to the bottom, which was reached at 166° F.	Oct. 3

of the fibrin had been dissolved. A similar experiment was also made using raw instead of boiled fibrin. In order to make certain that any digestion which might be observed was due to something removed by the glycerine from the urinary precipitate, and not to the glycerine alone, or to the acid, control experiments were also made with pure glycerine instead of the glycerine extract. The results of these experiments were chiefly negative, but were not sufficiently definite to prove either the absence or presence of pepsin.

On the Presence of Diastatic Ferments (Ptyalin or Pancreatic).

To 100 grains of starch mucilage 10 grains of glycerine extract of albuminous precipitate from urine were added, and the mixture was digested for ten minutes in a water-bath at 100° F. After removal the fluid was divided into two parts. To A, tincture of iodine was added; there was no change of colour. To B, freshly made Fehling's solution was added, and the mixture boiled; no change occurred immediately, but on standing a minute or two, *a slight greenish yellow colour appeared.*

In a control experiment made with glycerine only and starch paste in the same proportions, iodine *gave a slight trace of blue;* whilst after boiling with Fehling, *no* change was observed.

The above experiments were repeated after twenty minutes' digestion; *a fair amount of sugar* was shown to be present by Fehling's test, whilst in the control experiment *no trace* of sugar was found.

On the Presence of Pancreatic Ferment (Trypsin).

The following experiment seems to show the presence of this ferment in the urine, although in small quantity, so that although raw fibrin was slightly attacked, traces of albumin being apparent in the solution, boiled fibrin was not dissolved at all.

To 10 grains of the glycerine extract 100 grains of a 1 per cent. solution of sodium carbonate was added, and the whole was digested with a piece of boiled fibrin for 18 hours at 80° F.

The fluid was tested (*a*) by boiling—there was *no* precipitate; (*b*) by adding nitric acid and boiling—there was *no* precipitate; (*c*) by neutralising with acetic acid—there was *no* precipitate.

The above experiment was repeated as a control experiment with glycerine. No evidence of pancreatic digestion was obtained.

The digestion was made in presence of *raw fibrin,* and the liquid was tested (*a*) by boiling—there was *no* precipitate; (*b*) by adding HNO_3—a *turbidity was produced* which did not dis-

appear on boiling; (*c*) on neutralising with acetic acid, *a very slight turbidity was perceptible.*

The experiment was repeated with plain glycerine as a control, and the result was tested as before; *no* evidence of any digestion was obtained.

On the Presence of Intestinal Ferment Converting Grape into Cane Sugar.

The following experiment is not conclusive, but rather tends to show that such a ferment is present in the urine.

September 29.—A solution of pounded white sugar was made, of which 200 grains were added to 20 grains of extract. After 40 minutes' immersion in the water-bath at 90° F., the liquid was tested with Fehling's solution; *a slight change* of colour was observed.

To 200 grains of sugar solution 20 grains of glycerine were added, and after 40 minutes' immersion in the water-bath, was tested by boiling with Fehling's solution; a slight change of colour occurred, not so much, however, as in the preceding case.

When the glycerine only was boiled with Fehling, a slight change of colour, equal to that which occurred in the preceding experiment, took place.

On Sugar and Glycogen in the Urine.

It was noticed that this urine in Gollop's case became milky when diluted with water. At first we thought this was due to fat or oil, but on mixing it with ether, removing the ether and allowing it to evaporate, no grease spot was obtained, showing the absence of fat. The milkiness was so great that the diluted urine resembled very closely the liquid obtained by boiling a fresh liver in the preparation of glycogen. The presence of glycogen was therefore sought for, and at first we thought we had found it in quantity, but further experiment showed we were mistaken. On acidulating the urine slightly with acetic acid and boiling, a very large precipitate of coagulated albumin fell. This was separated by filtration, and alcohol in excess added to the filtrate. This threw down a copious white flocculent precipitate closely resembling glycogen. When collected on a filter and dried, and removed from the filter, it formed a grey powder. This was partially soluble in water, and formed a slightly milky solution. On testing it, however, with tincture of iodine, no red colour appeared. In a little diluted urine to which glycogen had been added, the red colour appeared, although only for a moment and then disappeared. On boiling the solution of the white

powder in water with sulphuric acid, and then neutralising and testing for sugar, no positive evidence of its presence was obtained. The same was the case when the solution of the powder was heated for some minutes at a gentle warmth with glycerine extract of pancreas. On testing the diluted urine in the same way, sugar was found; but on testing another specimen of the urine without treating it either by acids or pancreas, sugar was also found. The quantity of sugar after treatment seemed to be somewhat greater than before, but it was not sufficient so to enable us to decide positively that the sugar had been increased. From these reactions it appears that the urine in Gollop's case contained sugar as well as albumen, and that possibly, but by no means certainly, a small quantity of glycogen was also present in it.

On Albuminous Substances derived from the Blood which are present in the Urine in Albuminuria.

In the preceding part of our paper we have dealt with albuminous substances absorbed from the digestive tract and excreted by the kidneys in much the same way as urea. The occurrence of these in the urine we regard as due to imperfect performance of the functions of the digestive tract rather than of those of the kidney. But it is impossible to remove albuminuria entirely from the connection it has hitherto had with diseases of the kidney, and class it like diabetes, which formerly occupied a similar position amongst diseases of assimilation. For even if we leave out of account the changes in the structure of the organ which produce albuminuria, but which take some time to produce, and might therefore be associated with alterations in digestion, we know that ligature of the renal veins will in a few minutes produce albuminuria, although the intestine has not been interfered with, or at least to such a slight extent that it would not have produced albuminuria had the ligature been merely passed around and not tightened upon the renal vessels.

The albuminous bodies which usually pass from the blood into the urine are serum-albumin and paraglobulin. Serum-albumin, as has been already mentioned, is soluble in water; paraglobulin is not soluble in pure water, but it is soluble in water containing a small quantity of NaCl, or containing much oxygen. It is precipitated from dilute solutions by carbonic acid, and is redissolved if the water be shaken up long enough to allow it to take up oxygen and let the carbonic acid escape. Both serum-albumin and paraglobulin have been found in the urine by Edlefson and Senator. In one case of chronic Bright's

disease, that of Gollop, we have already stated that the urine in this case became so milky on dilution that we at first thought the milkiness due to glycogen. But as we have shown already, the presence of glycogen was doubtful, and if present, its quantity was too small to account for the milkiness, for which we were therefore obliged to seek another cause.

Several peculiarities in regard to this milkiness were also noted. It appeared when the urine was diluted with distilled water or with warm water, but not when water fresh from the tap was used. This at once seemed to point to paraglobulin, which is soluble in water containing oxygen. Thus the water fresh from the tap might dissolve it, while water from which the air had been expelled by boiling would not.

The following reactions tended to confirm the conclusion that paraglobulin was the cause of the milkiness. Acetic acid caused the milkiness to disappear. Acetic acid with ferrocyanide of potassium caused a precipitate. These reactions show that the turbidity was not due to mucin. The milkiness also disappeared on the addition of a 2 per cent. solution of NaCl, of dilute hydrochloric acid, of ammonia, or of caustic potash.

On passing a stream of CO_2 through the diluted urine, the milkiness became much greater, and a white flocculent precipitate appeared in considerable quantity, separating very slowly from the liquid. On filtration a nearly clear liquid was obtained. This began to get turbid when heated to 140° F., but between 160° and 165° the turbidity rapidly increased, and a flocculent precipitate (paraglobulin) separated. On filtering this, and adding its own bulk of absolute alcohol to the filtrate, a slight but distinct milkiness appeared and gradually increased. This is probably due to the presence of peptones.

On the Coagulating Point of the Urine in Different Diseases.

A comparison of the tables given already under the head of the effect of food on the coagulating point, as well as of the two following ones in the cases of Gollop and Hill, will show that the coagulating point will not infallibly indicate the nature of the disease; yet the great differences between the urine of the two following cases, the one of waxy kidney, in which the coagulating point was about 162° F., and the other of chronic Bright's disease, in which the coagulating point was about 132° F., cannot be regarded as unimportant.

TABLE SHOWING THE CONDITION OF THE URINE IN THE CASE NARRATED ON PAGE 306.

Name or No.	Ward.	Disease.	Coagulation begins.	Observation ended.	Time.	Quantity.	Sp. Gr.	Reaction.	REMARKS.	Date.
Gollop No. 8	John	Chronic Bright's disease.	131°	140°	24 hours' urine	1¾ pts.	1019	Strongly acid	The urine was of a pale straw colour, and was turbid	Sept. 5
			132°	137°	24 hours	...	1005	...	The urine was diluted with distilled water to 1005	...
			124°	126°	24 hours	Nearly 2 pts.	1020	Strongly acid	The urine was of a pale straw colour, and was turbid throughout, except when otherwise stated	Sept. 7
			130°	135°	24 hours	...	1005	...	On diluting, the urine so milky that it was difficult to observe the coagulating point	...
			128°	132°	Before dinner	...	1021	Acid	Fairly clear; thick white sediment	Sept. 8
			132°	132°	Before dinner	...	1005	...	Became opalescent on diluting (not due to fat or mucin) globulin (?)	...
			130°	134°	24 hours	2 pts.	1019	Acid		
			128°	133°	24 hours	Over 2 pts.	1021	Acid	... ...	Sept. 10
			127°	128°	After dinner	...	1024	Acid	Coagulated remarkably quickly; it had, I believe, been standing for 24 hours.	Sept. 11
			131°	134°	24 hours	2 pts.	1019	Slightly acid		
			136°	136°	24 hours	...	1005	...	At 136° it had coagulated	...
			128°	134°	24 hours	Nearly 2 pts.	1021·5	Strongly acid	So thick as to require filtering	...
			...	...	24 hours	...	1005	...	The coagulation was marked by the opalescence	Sept. 13
			...	...	After dinner	...	1025	Acid	Fairly clear	...
			135°	...	After dinner	...	1005	...	The coagulating point is approximate only	...
			131°	132°	24 hours	2 pts.	1022·5	Acid	Slightly turbid; remarkably rapid and well-defined coagulation	Sept. 14
			136°	140°	24 hours	Over 2 pts.	1022	Acid	The urine was filtered before warming	Sept. 15
			130°	132°	After dinner	...	1025	...	Prompt coagulation	...
			128°	136°	24 hours	Over 2 pts.	1024	Acid	The urine required filtering	Sept. 17
			132°	136°	After dinner	...	1024	...	On diluting, the urine as before became opalescent	Sept. 18
			134°	138°	24 hours	2·25 ts.	1022	Acid	... ...	Sept. 18
			134°	136°	After dinner	...	1013	Acid	... ...	Sept. 19
			128°	131°	24 hours	2·25 pts.	1022	Acid	Slightly turbid; becomes opalescent on diluting	

			136°	138°	After dinner	...	1023	Acid	Clear and light-coloured urine; coagulates very distinctly	Sept. 20
			138°	140°	24 hours	2¼ pts.	1021	Acid	The urine is rather turbid, and was not tested after dilution as it was opalescent	...
			136°	138°	After dinner	...	1025	Acid	Clear and light coloured; of an oily consistency	Sept. 21
			132°	136°	24 hours	2 pts.	1025	Acid	Slightly thick	...
			132°	138°	After dinner	...	1025	Slightly acid	Fairly clear	Sept. 22
			134°	138°	24 hours	1·5 pts.	1025	Slightly acid	Rather turbid; opalescence well marked on diluting	...
			136°	139°	After dinner	...	1024	Acid	... ...	Sept. 24
			136°	140°	24 hours	2 pts.	1023	Acid	Rather thick	...
			134°	136°	After dinner	...	1025	Acid	The colour was somewhat darker than usual	Sept. 25
			132°	135°	24 hours	1·75 pts.	1023	Acid	Rather thick; became as usual opalescent on dilution *Cf.* No. 11 Hope Ward	...
			144°	138°	After dinner	...	1024	Acid	The urine was quite clear, with a thick sediment	Sept. 26
			131°	138°	24 hours	2 pts.	1024	Acid	Rather thick	...
			131°	133°	24 hours	Over 2 pts.	1025	Acid	Rather darker in colour than usual; became opalescent on diluting	Sept. 27
			137°	144°	After dinner	...	1024	Acid	Urine clear	Sept. 28
			136°	140°	24 hours	1·5 pts.	1024	Acid	Slightly turbid; very opalescent on diluting	...
			132°	137°	24 hours	1½ pts.	1024	Acid	Very slightly turbid; opalescent on diluting	Sept. 29
			135°	139°	24 hours	Nearly 2 pts.	1021	Acid	Turbid with viscid masses at bottom of vessel; rather dark coloured	Oct. 1
			132°	138°	24 hours	Over 2 pts.	1022	Acid	Very thick, and of a somewhat dark colour	Oct. 2
			130°	135°	24 hours	2 pts.	1020	Acid	Turbid	Oct. 3
			131°	138°	24 hours	1 pt.	1022	Acid	Light coloured; turbid	Oct. 5
			128°	136°	24 hours	2½ pts.	1018	Acid	Light coloured; rather turbid	Oct. 6
			132°	138°	24 hours	2¼ pts.	1020	Acid	Dark coloured; turbid	Oct. 8
			136°	144°	24 hours	1½ pts.	1014	Acid	Darker coloured; turbid; the observation was made in the laboratory, with the laboratory urinometer	Oct. 9
			130°	140°	24 hours	Over 2 pts.	1018	Slightly acid	Lighter coloured; turbid; the experiment made with laboratory instruments	Oct. 10
			132°	138°	24 hours	2¼ pts.	1020	Acid	Light coloured; slightly orange tint; very turbid	Oct. 11
			132°	138°	24 hours	...	1020	Acid	Darker coloured; turbid	Oct. 12
			128°	134°	After dinner	...	1020	Acid	Rather light in colour; turbid	...

George Gollop, aged 27, chairmaker, admitted to John Ward under Dr. Church, August 30, 1877.

August 30.—Pale face, watery conjunctivæ. Œdema of lower limbs and scrotum; some ascites. Pulse, 80; rather hard. Heart sounds normal. Some impairment of resonance and feeble breathing at bases of both lungs. Urine pale; acid; white deposit, specific gravity, 1020; highly albuminous. The deposit was several times examined microscopically, and found to consist chiefly of loose renal epithelium, occasionally with casts, hyaline, granular, or epithelial.

Had scarlet-fever when quite young; smallpox (?) six years ago. Since that time has had a cough. Legs began to swell last January; abdomen about six weeks ago.

Put on D. L. and milk. Confect. jalapæ, ℥i. o. m.; Tr. ferri perchlor., m. xv.; glycerini, m. x.; aq. menth. pip., ℥i. 6tis.

September 1.—Urine has ranged from 1¾ to 2 pints. Œdema and ascites increasing. Meat makes him sick.

Put on D. L., fish, two eggs. Hst. scoparii co., ter.

September 13.—Urine has averaged 2 pints. Is no better.

Ordered Pot. acetat., gr. xx.; sp. æth. nit., ℥ss.; tr. digitalis, m. xv.; ex. hst. scoparii co., ter.

October 6.—Has complained much of thirst. Cough is getting worse.

Resinæ copaibæ, gr. x., ter.

October 11.—Urine has reached nearly 3 pints per diem, but the œdema remains.

Pot. acetat., gr. xv.; sp. juniperi, ℥i.; inf. digitalis, ℥ss.; hst. a. a. ℥. scilla ad., ℥i. 6tis. Chop or fish according to fancy.

October 24.—Urine has averaged two pints. Dyspnœa increasing.

Medicine to be discontinued, and the constant current to be applied daily, one rheophore to the lumbar region, the other to the front of the abdomen.

November 10.—Electrical treatment to be discontinued; it has produced no diuresis. The œdema, anasarca, and dyspnœa have been worse.

Lately he has had pulv. jaborandi, gr. xxv. o. n., but no effect can be certainly traced to it.

November 12.—Convulsive fit.

November 17.—Is still under treatment.

Henry Hill, aged 16, admitted to John Ward under Dr. Church, August 2, 1877.

August 2.—Small, pale, and ill nourished. Tongue clean and red. Pulse small, 100. General prominence of lower cervical, and sharp projection of lower dorsal vertebræ. A discharging sore in either groin. Abdomen very large, containing fluid. Liver enlarged to within 3 inches of umbilicus; firm, smooth, with sharp edge. Urine amber-coloured; clear; specific gravity, 1015 to 1020; moderately albuminous. Feet and legs œdematous. No loss of motor power in legs.

Has had curvature of spine six years, abscesses in groins three years, swelling of legs and abdomen two months.

Ordered D. L., milk, Oj.

August 8.—Diarrhœa, which he ascribes to the milk.

Allowed a pint of ale instead, and given bismuth. subnitr., gr. x.; pulv. tragacanth co., gr. xv.; ex. hst. cretæ aromat., 6tis.

August 10.—Diarrhœa varies.

Diet altered to beef-tea, arrowroot, pudding, egg i., soda-water, ij., brandy, ℥ij.; given pulv. kino co., gr. x.; o. n.

August 16.—Diarrhœa better.

Chop and an extra ounce of brandy added to diet. To stop the kino powder, and to take acid. sulph. aromat., m. xx.; tr. opii, m. iv.; sp. chloroform, m. x.; decoct. hæmatoxyl., ℥i. ter.

August 20.—Very fanciful about his food. Put on extras.

September 3.—Dyspnœa, and pain over liver, with increase of ascites, have been gradually coming on, and have reduced him to an extremely feeble state. Paracentesis performed; 16 pints or more of clear ascitic fluid withdrawn, to his great relief.

September 6.—Abdominal pain.] Cannot sleep without tr. opii, m. x.; o. n.

Medicine of August 8 repeated.

September 17.—Paracentesis had to be performed again.

September 22.—Died.

At the post-mortem, the liver, kidneys, and spleen were enlarged and amyloid; the intestines and stomach also stained with iodine; there was a psoas abscess on either side; but no active disease of the vertebræ.

TABLE SHOWING THE CONDITION OF THE URINE IN THE CASE OF HILL, PAGE 307.

Name or No.	Ward.	Disease.	Coagulation begins.	Observation ended.	Time.	Quantity.	Sp. Gr.	Reaction.	Remarks.	Date.
Henry Hill No. 3	John	Waxy kidney	164°	170°	24 hours	½ pt.	1016	Neutral or slightly acid	The urine was strong-smelling, dark-coloured, thick, and ropy	Sept. 5
			164°	170°	...	...	1005	...	The urine was diluted with distilled water	
			158°	164°	24 hours	...	1014	Acid	The urine continues to be dark coloured, but is less viscid, and is pretty clear	Sept. 7
			160°	160°	...	...	1005	...		
			160°	166°	Before dinner	...	1015	Acid	Dark coloured, containing blood clots; thick, and slightly viscid	Sept. 8
			166°	172°–174°	...	...	1005	...		
			164°	...	24 hours	Over ½ pt.	1014	Acid	Still viscid	
			166°	170°	...	...	1005	...	Coagulation not particularly well marked	
			162°	168°	24 hours	¾ pt.	1014·5	Acid	Less ropy	Sept. 10
			152°	166°	...	...	1005	...		
			168°	170°	10 hrs. after food	...	1019·5	Acid	The next passed after dinner; dark coloured; not ropy; coagulation distinct; had stood 16 hours	Sept. 11
			166°	172°	10 hrs. after food	...	1005	...		
			162°	168°	24 hours	½ pt.	1017·5	Acid		
			161°	165°	24 hours	...	1005	...		
			168°	171°	24 hours	1¼ pt.	1014	Acid	Slightly turbid; coagulation apparently began at 162°, but was not decided	Sept. 13
			164°–169°	174°	24 hours	...	1005	...		
			162°	168°	After dinner	...	1015	Acid		
			164°	...	After dinner	...	1005	...		
			163°	168°	24 hours	Over 1 pt.	1012	Acid	... ...	Sept. 14
			162°	164°	24 hours	...	1005	...		
			163°	168°	24 hours	...	1015	Acid	Turbid, with visible particles suspended in the fluid	Sept. 15
			162°	166°	24 hours	...	1005	...		
			167°	170°	After dinner	...	1014	Acid		
			171°	173°	After dinner	...	1005	...		

			158°–166°	170°	24 hours	Over 1 pt.	1015	Acid	Tolerably clear; the main coagulation occurred at 166°–170°	Sept. 17
			164°–166°	171°	24 hours	...	1005	...		
			161°	171°	...	...	...	Alkaline	Fluid obtained by tapping; was of a light yellow colour, and of a thick consistency	Sept. 18
			169°	172°	...	...	...	...	The fluid was diluted with its own bulk of distilled water	
			156°	160°	After dinner	...	1017	Acid	The boy was tapped at 4·30 P.M. on the 17th; examination of fluid was made at 2·30 P.M.	
			158°	162°	After dinner	...	1005	...	The urine was thick and very dark coloured; it was filtered	
			152°–158°	162°	24 hours	Under ½ pt.	1016	Acid	The urine on diluting became slightly opalescent, as in No. 8 case	
			156°	161°	24 hours	...	1005	...	Urine dark coloured	
			158°	164°	After dinner	...	...	Acid	Not sufficient to take sp. gr.; very thick, but cleared on warming; filtered	Sept. 19
			153°	156°	After dinner	...	1005	...		
			154°	160°	24 hours	¼ pt.	1017	Acid	Urine clear, and not so dark coloured as usual	
			159°	164°	24 hours	...	1005	...		
			160°	164°	After dinner	...	1014·5	Acid	Urine clear and dark coloured	Sept. 20
			162°	168°	After dinner	...	1005	...		
			162°	166°	24 hours	⅛ pt.	1015	Acid		
			154°	...	24 hours	...	1005	...	Coagulation was very indistinct, but was probably about 154° F.	
			162°	168°	After dinner	...	1013	Acid	Much lighter coloured, and nearly clear	Sept. 21
			164°	...	After dinner	...	1005	...	Coagulation not well marked, but continues onwards from 164° F. As the boy died, the urine for 24 hours was not obtained. No decided coagulation even at 182° F.; urine dark coloured; coagulation not perceptible at 182° F.	Sept. 22

In these tables "24 hours" means the collected urine passed during twenty-four hours.

General Results.

The general results at which we have arrived from the observations detailed in this paper are :—That there are various albuminous bodies which appear in the urine. Some of these are derived from the digestive canal, and others from the blood, of which they form ordinary constituents. Those derived from the digestive canal may be either albuminous substances absorbed without undergoing digestion, as, for example, the white of raw eggs, soluble albuminous substances produced from coagulated albuminous bodies by incomplete digestion, or peptones. Those albuminous bodies which form constituents of the blood, and which we have noticed in the urine, are paraglobulin and serum-albumin. The former is in comparatively small quantity, the latter forming the great bulk of the albumin in ordinary albuminuria. Glycogen was only doubtfully present. The coexistence of sugar and albumin, which we noticed in one case,[1] is interesting, as Bernard found that while puncture of one point in the fourth ventricle caused sugar to appear in the urine, puncture of an adjoining point caused albumin to appear; and Pavy succeeded by one puncture in causing them both to appear together. The effect of food is to increase the quantity of albumin in the urine, or even make it appear when it is absent during fasting. Its effect on the coagulating point is not constant, although it generally lowers it. This may be due to the fact, which we have observed, that while the earlier products of the pancreative digestion of fibrin have a lower coagulating point, the later products have one which is higher than that of serum-albumin. In connection with this point it is interesting to notice that while pepsin occurs normally in the urine, we failed to find it, possibly because we operated on too small a quantity, and nevertheless we obtained evidence of the presence of pancreatic ferment (trypsin). This is the first case we know in which this ferment has been found in the urine, and its loss in unusual quantity may possibly prove injurious to digestion, and be one cause why the albuminous products of imperfect digestion appear in the urine. Further observations are wanted, but this would seem to point to the existence of a vicious circle in this, as in other diseases, the albuminuria leading to the excretion of pancreatic ferment, and the loss of pancreatic ferment leading to imperfect digestion with excretion and loss of its soluble albuminous products. In a case recorded by one of us elsewhere, pancreatine was found to lessen the albumin, or

[1] That of Gollop.

even remove it entirely from the urine.[1] We also found a diastatic ferment which might either be ptyalin, already found by Cohnheim in normal urine, or the diastatic ferment of the pancreas. Intestinal ferment converting cane into grape sugar was doubtfully present in the urine.

It is certain that albuminuria cannot be removed, like diabetes, from its place amongst diseases of the kidney to be classed entirely with diseases of assimilation, but probably some cases are almost or entirely due to disordered assimilation, and also in those cases where the kidney is undoubtedly diseased, the loss of albumin is increased by disordered assimilation. Attention to this point in the pathology of the disease will of course have an important bearing on treatment, and while the effects of remedies as tried in a case of confirmed Bright's disease, such as Gollop's, have been hitherto unsatisfactory, it may yet be possible, under the guidance of a more correct pathology, to attain to more efficient measures of cure. The coagulating point of the albumin in urine may be readily ascertained without trouble at the bedside. The indications which it gives are not decisive as to the nature of the disease, but more extended observations than we have been able to make may, and probably will, yield much information useful both in prognosis and treatment.

[1] Arsenic in Albuminuria, by T. Lauder Brunton. "Practitioner," June 1877.

PROCEEDINGS

OF

THE ABERNETHIAN SOCIETY

FOR WINTER SESSION 1876–77.

October 12, 1876.

Dr. Gee delivered the introductory address, which is here given *in extenso*.

This evening we celebrate the opening of the forty-fourth session of the Abernethian Society by the usual introductory address. On referring to Dr. Coomb's historical sketch (Hos. Reps. vol. iv.) of our Society, I find that the first introductory address was given by Sir James Paget in 1844. But no doubt many of you know that, although this Society has borne Abernethy's name since 1832, it was founded long before, by Abernethy, in 1795, under the name of the Medical and Philosophical Society of St. Bartholomew's. So that ours is one of the oldest scientific societies in London.

The most obvious purpose of a scientific society, I suppose, is the getting of truth: ours is a medical society, and our object is medical truth. Now it seems to me that medical societies may be of two kinds. First, there may be societies of men who are no longer students in the common sense of the word—men who know what is already known, and whose object is the winning of new truth out of the boundless world of the unknown. Next, there may be societies of men who are still students, commonly so called—men whose business is not so much to discover new truth as to make themselves master of the old.

The Abernethian Society, whatever it may formerly have been, is now of the second kind rather than the first; that is to

say, it consists in greater part of students, from whom we do not expect truths absolutely new, so much as truths which are relatively new—truths which are new to most of the hearers, or old truths arranged in a new fashion. The papers which are read here are almost always, and almost necessarily, of this kind. We are not ashamed here of old truths. We welcome the new, but, as I said before, we do not much expect them; for, in Milton's words, 'Our wings here are fledging; we may meditate a future flight, but our Pegasus soars as yet on feeble pinions.'

Our wings, our Pegasus. I have hinted, before I meant it, at another, and indeed at the chief, use of our Society. Hereafter I will speak of this topic more at large, but let me say now, that if the Abernethian Society did nothing more than scatter a few truths abroad, it would have small reason to show for its existence. But ours is a dialectic society. We discuss the papers we read; we question, doubt, deny; we look at a reputed truth, whether new or old, from all points of view; we confront it with contrary and contradictory truths; and in doing so we strengthen the very spirit of truth itself. Under this discipline our wings grow strong, and our Pegasus comes to soar on pinions which carry him up to the very sun of truth. But more of this hereafter.

Let me resume the earlier part of my discourse relating to the papers we read and the specimens we show at our meetings. The profit of the papers read here is great, both to the reader and the hearers. A good paper collects truths from all times and all countries; but this is not all; greater still is the profit and the pleasure which we find in beginning or renewing an acquaintance with the great minds who have made medicine what it is. We do not read Hippocrates only for the sake of the isolated truths we learn from him. That were scarcely worth the pains. The mark of a book, written by a man of genius such as he, is that it cherishes our own spirit of truth; we seem to breathe the very air of truth; he inspires us; and as long as we feel his influence we are raised above our ordinary selves. I appeal to those who are listening to me whether this is not so. Our own love of truth vibrates in unison with the master's tones, and this power of awakening the slumbering faculties of men is the gift of genius. He is the genius, emphatically so called, who discovers the same faculty in his fellows. For what is genius? In the words of a member of our own profession, which I have been so bold as to adapt to the present occasion—

> 'For genius, bearing out of other worlds
> New freights of thought from fresh-discovered mines,
> Is but reciprocated love of truth.
> Witness Hippocrates, our guardian angel;
> Harvey, whose thought created man anew;
> And Auenbrugger, poor neglected seer,
> Who, like a moon, attracted naturally,
> Kept circling round the central sun of truth.'

Let me then exhort you who read with the purpose of making ready a paper for this Society, above all things to choose out books of genius. You will have enough to read which (even if truth) cannot be called genius in the sense we have spoken of before. And such reading is not unprofitable in its way; but it must not shut out reading which is far more profitable. To give an instance of what I mean. It is very well to read the periodical journals of the day, and you will profit by the reading; but it would be a sad mistake to allow such reading, as it easily may, to leave us no time for looking up our Hippocrates, Sydenham, and Lænnec. It is the old strife between the letter and the spirit. A truth is good, but the truth is our chiefest good. To possess truth is good, it is much better to be possessed by truth.

He, then, who would earn the name of a man learned in his art and mystery, must first of all ponder what Hippocrates has left us. Medicine begins with him. No doubt medicine has existed as long as man, and curious antiquarians find traces of it in the oldest records, Egyptian or Indian. We may compare medicine, as Celsus does, to finding food. Truly food and physic are the most immediate necessities of man. Nay, even the gorilla practises medicine, so we are told by Livingstone; but the science begins with Hippocrates. He laid its foundations deep and wide, deep and wide enough to last for ever. Of this there is proof in the remarkable fact that many men of all ages, even down to the present day, who have most deserved the name of good physicians, have taken the greatest interest in exploring the method which Hippocrates followed. For in all human knowledge the question of method must come first. The nature and scope of our understanding, the order of its operations, and the fundamental law which regulates them, these are surely worthy subjects of thought. Now the method of Hippocrates is not hard to discover. He is an empiric in the original sense of the word. In the books of Epidemics we see the first stage of his process. He collects the histories of as many patients as possible; and we may say that these histories are models of case-taking, and well worthy the study of those whose duty is to be good clinical clerks and registrars. The simple truthfulness

of the old Greek's notes is delightful. In the next stage he proceeds by what we now call logical induction from these particulars to universals, and the greater part of the other books of Hippocrates is made up of these universal propositions. But the word 'universal' has a relative meaning, admitting of less and more. Now in making universals more and more extensive, they become at last too abstract to be useful. It is here that Hippocrates shows his judgment. He is great in what Bacon calls middle propositions. I refer to that well-known passage in the 'Advancement of Learning' where he says, 'Plato, in his "Theætetus," noteth well that particulars are infinite, and the higher generalities give no sufficient direction; and that the pith of all sciences, which maketh the artsman differ from the inexpert, is in the middle propositions, which in every particular knowledge are taken from tradition and experience.' The middle propositions which Bacon speaks of are, in other words, universal propositions which have not ceased to be useful in particular cases. And I have somewhere met with a saying of Cabanis to this effect. 'Hippocrates shows that he is on his guard both against those hasty views which generalise from insufficient data, and also against that impotence of mind which is not able to perceive the relation between things, and so drags on after particulars, without result, for ever.'

The result of this Hippocratic method is aphorisms, pithy sayings complete in themselves. The book of Prognostics and the book of Aphorisms consist entirely of such precise and independent propositions. And let me try to prove to you that the Hippocratic method is the true method in the words of Littré, the famous editor of Hippocrates, disciple of Comte and lexicographer. 'In all sciences the starting-point is the facts of experience. In some sciences the experimental basis is very small, and yet supports a superstructure of immense extent; such are the mathematics. But in proportion as we leave these sciences, which are almost pure, so increases the complication of the experimental conditions, and this complication becomes excessive in physiology, and in medicine which depends on it. It is in medicine especially that we have to guard against the drawing-on of induction and the temptation to logic. The anticipations of reasoning are here most at fault; here it is that all the artifices which the human mind has devised for passing from the known to the unknown are of least avail; and, in one word, here it is that facts have most authority, and reasoning least, inasmuch as our science consists not in the infinite development of a few fundamental axioms, but in the more or less advanced co-ordination of innumerable particulars.' Those who have

gone through the process will agree with me that the deducing of true and useful aphorisms from a multitude of particular instances is no easy task. In fact, the last stage of the Hippocratic method is always in danger of being left unfulfilled by reason of the heap of facts which a man accumulates around him; mere baggage, impedimenta, or raw material of no worth whatever, unless it have passed through the workshop of the mind. We all become overloaded with notes of cases, burdening our drawers and pigeon-holes, and which we have not time enough to smelt, as it were, into useful metal.

And this intellectual smelting is a great toil. Sydenham has told us what his treatise on Gout cost him.

'I send you a short tract upon gout and dropsy, instead of the thicker volume which, in my mind, I had determined on, namely, a history of such chronic diseases as my practice has most especially met with. By applying my mind, however, to its utmost, and bringing all my powers of thought to bear upon the subject, I incurred a fit of gout, such as I had never before suffered from; so that the fact itself warned me to lay aside, even against my own will, such lucubrations, and to take care of myself, well satisfied with having, in some measure, dealt with these two diseases. Whenever I returned to my studies the gout returned to me.'

> 'Mid the wreck of *is* and *was*,
> Things incomplete and purposes betrayed
> Make sadder transits o'er thought's optic glass
> Than noblest objects utterly decayed.'

And here Hippocrates stops short. His method having provided him with useful aphorisms, he goes no further. He may arrange his aphorisms under sundry heads, those which relate to ætiology, prognosis, or therapeutics; those which relate to diseases of the head or other parts; those which relate to this symptom or that, and so forth; but he is too wise to enter upon a third stage, and to construct a system. And what is system? It is an attempt to introduce the method which is opposed to that which has brought us so far safely; system is syllogistic and not inductive; it attempts to explain things, to show us the why and wherefore, to deduce facts from principles, particulars from universals; it tries to make knowledge look as if it approached perfection. If system delude us into the notion that it can give us aphorisms, or principles useful in practice, system is an evil which cannot be denounced too strongly.

The only use of system is to serve as an intellectual gymnastic; but even in this way, applied to so practical a knowledge as

medicine, it may be highly dangerous, through our mistaking the phantoms of system for the truths of induction.

In sciences which are purely speculative, system may be carried to the highest degree; and a great delight it is to see how man's mind can create a speculative universe alongside of that which exists independently of us. Take Spinoza's ethics as an instance of what I mean. But between Spinoza's universe and the real world of things is a great gulf fixed, not bridged over, so far as I can see, in one single spot. And thus the danger of mistaking pure imagination for reality is less than when the propositions of syllogism and induction are mingled. And from this mingling the Hippocratic method is wholly free. It gives us inductions pure, detached, isolated—inexplicable they may be, but they are true. They relate to life and work. But system is mere intellectual sport—

> 'And tired with systems, each in its degree
> Substantial, and each crumbling in its turn,
> Let us build systems of our own, and smile
> At the fond work, demolished by a touch.'

Did time allow I should be greatly pleased to unfold this doctrine of the Hippocratic method yet more. I should like to examine the work on Ancient Medicine along with you, and to show how the polemics which Hippocrates directs against the dogmatists of his day might be revived against the dogmatists of our day, especially the chemists and physiologists. With the work on Airs, Waters, and Places for our text-book, we might discuss the topics of ætiology, hygienics, and prophylactics. The book on Prognostics and on Regimen in Acute Diseases would guide us in a search for the principles of therapeutics. These are some of the things which the father of physic will teach us, and I am glad to believe that ours is not more an Abernethian than a Hippocratic society.

Next to Hippocrates in respect to time comes Celsus. His merits are very different from those of Hippocrates. Celsus is not a genius; his work is so largely compiled from the writings of others, that some critics have thought that it is nothing but a compilation. But it is this very thing which makes Celsus' book so valuable as it is. He is supposed to have lived in the great Augustan age, four centuries at least after Hippocrates; and these were very busy centuries; in particular, the great Alexandrian school of medicine had flourished at that time. He refers to about eighty writers of medicine whose works are entirely lost; and, indeed if it were not for Celsus, we should not know how great had been the increase of medical knowledge

since the days of Hippocrates. Celsus is characterised strongly by truthfulness, clearness, and good sense.

After Celsus it might be expected that I should say something concerning Galen, who lived in the reigns of the Antonines; but I confess I could never read him. It is all very well that he should write commentaries on Hippocrates, but he is far enough from possessing the Hippocratic spirit. He does not work by the Hippocratic method; he is almost more of a metaphysician than a physician, and his prolixity is great.

Even Aretæus, much as he has been praised, and much as he deserves to be read, shows a falling away from the principles of the great master, the 'divine old man,' as Sydenham calls him. Aretæus does not give us the aphoristic truths which are the life of medicine, but he draws striking pictures of diseases somewhat idealised. He reminds us of Dante and Spenser more than of Hippocrates; however, his fragmentary book leaves our art a step farther on the road than Celsus.

Cælius Aurelianus is the only other ancient whom I can recommend as being worthy your study: if you can read him; for his book is written in a most barbarous style, and would, I think, puzzle a man who might have spent ten years in reading Cicero. And unluckily he has never been translated, as he well deserves to be. I confess I have dipped into him only here and there, but I have found good things in him.

Paulus Ægineta's is a poor performance. However, it has been translated, and so is worth keeping on one's shelf as a monument of what medicine was about the time of the overthrow of the Western Empire.

You may think that I speak of a long course of reading; but, my friends, take courage; the next ten centuries will give you a vacation of study.

> 'See Christians, Jews, one heavy sabbath keep,
> And all the Western world believe and sleep.'

But let us not be too hard even upon the dark ages. In the twelfth century a work was done which has had great results for us. The foundation of St. Bartholomew's indeed consisted at first, as you know, of two parts: one monastic, temporary, suitable perhaps to the time, but which (it was written in the book of fate) should not endure, and which died out, utterly corrupt, four centuries after the days of Rayer; the other part, medical, abiding, necessary to all ages, and which has gone from strength to strength, until it has become the great hospital and school of medicine in which we all feel the deep satisfaction which words are poor to express.

The first effect of the revival of learning in the fifteenth century seemed, so far as medicine was concerned, to be a kind of deification of the ancients. Hippocrates and Galen were supposed to be not less infallible than the Pope of Rome. A remarkable instance of the truth of what I say is found in the annals of the College of Physicians.

It stands thus in the annals :—

'December 22, 1559.—It has been notified to John Geynes, otherwise a man well behaved and not imprudent, that within one month he make known to the College in writing all those passages concerning which he was not ashamed to say in public and before the learned, and even in the presence of the whole College assembled in its usual committees, that Galen has erred.'

'1560.—Last year, in the month of December, John Geynes was commanded to bring forward all those passages concerning which he was wont publicly to say that Galen had erred, according to the testimony of that worthy man, Thomas Wendy, Physician to the Queen, and even of Geynes himself, openly saying so before the whole College. But when he had refused, the Sheriff of London, by order of the President of the College, compelled him to hold forth or go to gaol.

'When, however, Geynes refused to defend his cause, and clearly perceived that he, and not Galen, was wrong, he honourably gave himself up, and acknowledged his fault ; he lamented that he had propounded blunders, that he had not been more circumspect, that he had not pondered the passages of Galen more carefully, that he had not sought out Galen's meaning, that he had not understood Galen's opinion, that he had not quoted Galen's words faithfully, that he had not shown due respect to Galen, and had accused him falsely.

'Which he also confirmed by his subscription in the following words: "I, John Geynes, confess that Galen has not erred in those things which I alleged against him."'

This event is but a flicker in the socket of a dying taper : the days of Galen were numbered and finished. The kingdom was about to be taken from dogma and given to dialectics. Galen was soon to be weighed in the balances and found wanting.

William Harvey was born in 1578, or less than twenty years after Geynes' trial for heresy. The discovery of the circulation of the blood was a deadly wound to authority and Galen. The Hippocratic spirit soon revived.

I have looked into a few of the most famous books on physic written at the end of the sixteenth and beginning of the seventeenth centuries, before Harvey's discovery, particularly into Fernelius, Mercurialis, and Ballonius, and I found that the new epoch had

not yet begun. The full influence of Harvey and free-thinking is not felt in medicine until we come to Thomas Sydenham, who was born in 1624. But I will briefly refer to two English physicians who were somewhat older than Sydenham, and whose writings are not yet forgotten. I mean Francis Glisson and Thomas Willis. Glisson was the discoverer of rickets; Willis discovered saccharine diabetes.

And here I hope you will pardon me if I indulge in a short digression suggested by the name of Willis. I wish to say a few words upon the manner in which we should read these old books. I think you will admit that it is a sound principle of interpretation which Fichte lays down in his sixth lecture on the Doctrine of Religion: 'So to understand these writers as if they had indeed wished to say something, and, so far as their words permitted, as if they had said what was right and true,—a principle which seems to agree with justice and fairness.' Let me illustrate this principle by an example.

Willis talks much of the vital and animal spirits. I confess that at first these terms do not convey any idea. We feel disposed to pass them over with charitable pity for a man who had nothing more real to write about. Yet he would be a rash man who would dare to say that Willis and Sydenham were guilty of using words full of sound but signifying nothing. The Animal Spirits. First of the Spirits. The Latin *spiritus*, a word originally meaning breeze or breath, grew in comprehensiveness until at length spirit and energy of any kind came to be synonymous. The same is true of the corresponding Greek word *pneuma*, and also (so Spinoza tells us in the first chapter of his Theologico-Political Treatise) of the Hebrew word *ruagh*. Spirits then mean energy. And next of the qualification, Animal. In Galen's system, the animal constitution is defined to be that which is derived from the brain, through the nerves, to the organs of sense and motion. This is enough, the animal spirits signify the energies of the nervous system; and if in later times men have talked first of nervous fluid and then of nervous force, I doubt that they have not done much more than change the names of things. Again, the animal spirits are truly said to be formed from the vital spirits. By the vital constitution is meant the whole sanguifying and sanguineous system. Then the vital spirits are the energies and forces of the blood. The vital spirits became transformed into the animal spirits; or, in modern phrase, the energies of the nervous system are derived from the energies of the blood.

Now we who within the last forty years have become familiar with the speculations of Mayer, Joule, Grove, and others, respect-

ing the mutability and eternity of energy, will have no difficulty in confessing this doctrine, although most likely we may be at first astonished to find it current three centuries ago.

Let these examples suffice. When we read old authors, we must be quite sure that we understand them before we judge them. The meaning of words is continually shifting, and we must resist their tyranny.

In Sydenham, the Hippocratic spirit fairly arises above the horizon once more. The increase of medical knowledge in the last two centuries has been immense, oppressive. It sometimes provokes a feeling of the deepest sadness. Who is sufficient for these things? Our life is too short; scarcely do we begin to understand a little, scarcely do our powers of discerning and judging become ripened, than we go hence and are no more seen. 'And I gave my heart to know wisdom: but I perceived that this also is vexation of spirit. For in much wisdom is much grief; and he that increaseth knowledge, increaseth sorrow.'

Do you remember the famous story told of Antoine Arnauld, whose friend asked him whether he rested not sometimes. 'Rest!' he answered, 'have I not an eternity to rest in?' My friends, let us console ourselves with the thought that we have an eternity to work in.

The strong desire which we feel to live for ever, our longing to become more and more like the Eternal, our conviction of aptitude for eternity, belie the appearances of death. This present life is our infancy, in which we make a few first steps; feeble steps perhaps, but woe to him who neglects his calling here! Can he who is faithless in these few things expect to be made ruler over the many things which we look forward to in a life to come? What must happen to those who fling away their birthright of opportunity, we do not know.

Sydenham's writings are of unequal value. The books he published last are by far the best. I will not go into details; I will only say he marks a reformation in medicine.

After Sydenham's time we meet with no more books written in the old scholastic manner, like those of Glisson and Willis. And what we may call the Sydenhamian or revived Hippocratic school flourished down to the end of the last century.

A contemporary of Sydenham was Richard Morton, who published three books of great worth shortly before his death at the end of the seventeenth century. One book on diseases accompanied by Atrophy, the other two are on Fevers. They are quite Hippocratic.

Richard Mead, William Heberden, George Baker, John Fothergill, Robert Whytt, Francis Home, William Withering,

David Pitcairne, James Currie, Edward Jenner, these are the chief English names in medicine during the last century, and the same spirit pervades them all.

In Italy, Baglivi, Lancisi, Torti, and Borsieri; in France, Vieussens, Senac, and Bordeu, worked by the same method.

In Holland, Hermann Boerhaave appeared, a sort of modern Galen, bearing much the same relation to Sydenham that Galen did to Hippocrates, who wielded an authority during his lifetime not much less than that which so long belonged to Galen. They say that Boerhaave used to take off his hat when he spoke of Sydenham; but he was not a true disciple of Hippocrates or Sydenham. He invented a system of pathology which, like all our little systems, had its day; it had its day, and ceased to be. There was not in it life of truth enough to keep it sweet and sound, and few are those nowadays who read Boerhaave.

Boerhaave's praise is the men who were his pupils. First of all, Albert Haller. Next, Gerard van Swieten and Antony de Haen. These latter two men were persuaded by Maria Theresa to settle in Vienna, in order to revive the school of medicine there; and their scholar, Maximilian Stoll, was greater than either of his masters. And so the eighteenth century came to an end.

But I have not yet spoken of the two men of that century who had the greatest influence upon medicine; John Baptist Morgagni and Leopold Auenbrugger. Hippocrates marks an epoch; so do Harvey and Sydenham; and also Morgagni and Auenbrugger. The year 1761 is a year much to be remembered in the history of medicine. In that year Morgagni, eighty years old, published his book on the seats and causes of diseases: this was the beginning of morbid anatomy. In that year Auenbrugger published his book on percussion of the chest: this was the beginning of the art of examining the deeply-seated organs during life. The only year which can compare in fame with 1761 is 1628, the year of Harvey's book.

The men of whom I henceforth have to speak lived under these new influences. I will mention the dead only. In our own country we celebrate Matthew Baillie, Robert Willan, John Blackall, William Charles Wells, Richard Bright, Thomas Addison, Thomas Hodgkin, Robert James Graves, John Abercrombie, Peter Mere Latham, Robert Gooch, and Marshall Hall. But the French school of medicine was the most famous of the first thirty or forty years of the present century. Corvisart, Bayle, above all Laennec, Broussais, Piorry, Bretonneau, Cruveilhier, Andral, Louis, Trousseau, Rilliet, and, last of all, although by no means the man whose fame will be the least,

Duchenne. The Germans have worked well at physiology and microscopical anatomy, but I will not shrink from declaring my opinion that to medicine they have added very much less than the English or the French. The discovery of the ophthalmoscope by Helmholtz, and of the laryngoscope by Czermak, is their greatest praise. For my own part, I do not rate German medical books very highly. It may be a fault or failing of mine; but, to speak plainly, they are not to my taste. Time was when I followed the example of the many, and supposed that the secret of science was in Germany. I read many of their books with an honest intent, but I came to see that, for the most part, they were pervaded by two faults. The first fault was that syllogistic spirit of system which has led many of their best writers to think out over their desk symptoms of disease and methods of treatment, which the spirit of truth at the bed-side will not acknowledge. The other fault was the endeavour to say all that can be said, forgetful that there is a proportion between facts as between everything else, and that some facts are of great value, and others of little or none at all. This is a very old defect of the Germans. Montaigne alludes to it when he says, 'The Germans drink almost indifferently of all wines and liquors with delight; their business is to pour down and not to taste.'

I have now finished my historical sketch. I have spoken much of books, and I have no fears that any one of my hearers will cry down book-learning. Study and experience in medicine are sometimes opposed to each other, especially by men who usurp the name of practical. Experience is indeed the source of knowledge in medicine; but experience alone is of small worth; it is merely unwrought material. A man may pass through life with a very large experience, and yet with very little increase of knowledge. Experience without study profiteth nothing. You see a patient, you examine him as carefully as possible, you note down the details of his case, you ponder it, you compare it with other cases which you have recorded; this is the only kind of experience which deserves the name. And can we suppose that these methods of studious thought will not be carried out best by him who bears the best models in his heart? Is not the experience of that man likely to be the best who is fresh from the inspiration of the great prophets of medicine? Our book-learning will raise us common men above ourselves; we too shall see in part with the eyes of Hippocrates and Laennec. Let me take an instance of what I mean from another art. During the last great French-German war, Count von Moltke is said to have given it as his opinion that the

Algerian training of so many of the French generals was a bad training, because it led them to despise the rules drawn from the practice of the great masters of the art, and because it encouraged them in relying upon mere expedients of the hour. If this were the conduct of the French, they were condemned beforehand by a greater soldier than Von Moltke. Napoleon Buonaparte was talking with his generals one evening at Dahme, near Dresden, when the loss of the battle of Dennewitz by Ney caused the Emperor to speak of the difficulty of making successful war. Marshal St. Cyr declared that it was very doubtful whether the longest experience was indeed the best school for learning the art of war, and that of all the generals, French, Russian, Austrian, or Prussian, who had been at the head of the armies of Europe since the Revolution, not one, not even the Emperor himself, had gained by experience. And Buonaparte did not hesitate to confirm this remarkable opinion. He said that his first campaign in Italy was his masterpiece of war, and that he knew only one general who had profited by experience, and that was Turenne, whose great ability was the result of great study.

Let me now resume the topic at which I hinted in the beginning of my address. The Abernethian Society is a discussion society, and this is its chief use. For discussion is the great means of developing the spirit of truth which is within each one of us. Our faculty of discerning truth and falsehood underlies all logic, ratiocinative or inductive. Logic does but put in order the material which our faculty of truth is to judge. The simplest syllogism appeals to and presupposes this faculty. This is the essence of man, that which makes man man, that which distinguishes man from the beast. This is the dæmon of Socrates, the ruling part of Antoninus, and, if I may say so, it is the Word of God. *The* truth as distinguished from truths. Truth is in us all, but in us all is more or less obscured by the mists of passion. And passion is desire—desire of reputation, riches, or pleasure. Truth and passion are contradictory; they are the good and evil deity of man. We must choose between them; we cannot serve two masters; and if with us our *summum bonum* be to attain the full development of our nature, that is to say, to attain to a substantial union with truth (according to that sublime prayer of Thomas à Kempis, 'O Veritas Deus, fac me unum tecum in caritate perpetua'), if this be our aim, then we reach it only by casting out the great adversary, passion, which strives to drag us down below our nature. In a society such as this, desire of reputation is the passion most likely to be felt, and a discussion society will help us to subjugate it. How far short soever we may fall of our ideal, we know that we ought to come

to our meetings here saying in our hearts, 'My object is truth; to develop my own nature, and to help my brethren in developing theirs. The only reputation which I seek is that of being devoted to the truth. Men cannot praise the sharpness of my wits, and be it so; but in devotion to the service of my proper nature I need be second to none.'

Gentlemen, such thoughts as these open up the whole field not only of knowledge but also of morals; indeed, from our present point of view, truth and goodness blend. But I must draw to an end by reminding many of you that the Abernethian Society will probably afford you the only opportunity which you will have in your life of cultivating your powers in the way I have spoken of, namely, by discussion. I invite you all to join it; I would even urge you all to join it, because I am confident that you will find it to be for your good. Your student-days here are few; soon you will have to go out into the world to *do* the truth which here you have learned, and it behoves you to have your sinews well knit, and your weapons well proven before you engage in the great warfare against falsehood and ignorance, evil and disease. In the name of Saint Bartholomew's Hospital I tell you that it is expected that every man of ours will do his duty.

October 19.

Dr. Maberly showed an old form of pessary which had remained in the vagina for twenty-six years. The patient on applying for relief denied all knowledge of the presence of the foreign body, which was firmly impacted; removal was effected under chloroform by Dr. Godson.

The pessary was disc-shaped, and had to be split before removal.

Mr. Keetley read a paper on 'Some Points in the Treatment of Fractures.'

October 26.

Dr. Wharry showed a specimen of carcinoma of stomach.

Dr. Moore showed a specimen of gastric ulcer, with stricture of the pylorus, probably cancerous.

Dr. Maberly showed a specimen of hypertrophied clitoris, which had been removed by the écraseur.

Mr. Darbishire read a paper on 'The Treatment of Wounds.'

He pointed out the necessity of looking to the facts of the general behaviour of wounds in order to recognise a rational system of their treatment. By observation the surgeon learned that those wounds which were not exposed to the air, and from

which there was a free escape for all dead tissue, healed up most quickly and with least constitutional disturbance, therefore with less danger to the patient. He explained the constitutional disturbance by the early decomposition of the dead tissues, insisting that by the first chemical changes in the direction of decomposition, its most poisonous substances were formed. A free drainage was the most essential point in the treatment of a wound, for if that was intelligently arranged, then there was no need for antiseptics; the wound would keep healthy of itself.

As to the antiseptic method, it was of no use where a free drainage could be kept up from the wound; it might be of advantage when that was not the case, but only then. Every method or system of treatment was bad except the rational treatment which looked upon every wound as an individual to be treated (upon sound principles) according to its surroundings.

After a few words on the excellence of the practice of drainage as applied to wounds, the paper closed.

November 2.

Dr. Wharry showed a specimen of malignant disease of the kidney, with the lungs and portions of the intestine from the same patient.

The interest of this case lay in the fact that the disease had lasted fourteen years, and in the absence of symptoms pointing to active mischief in the lungs or bowel.

Mr. Pye showed two specimens of abdominal aneurysm, and read the notes of the cases.

Mr. Benton read notes of another case of abdominal aneurysm.

Dr. Wharry read a paper on 'Use of Sleep in the Treatment of Hospital Patients.' Three types of sleep were distinguished: the hyperæmic, anæmic, and toxæmic. Healthy sleep being due to slight degrees of cerebral anæmia, higher degrees produce insomnia, as in phthisis or delirium tremens. The characters and treatment of this form were pointed out. The use of stimulants on the one hand and narcotics on the other were condemned. Various means of producing sleep in patients whose insomnia is due to habit or mental excitement were discussed.

November 9.

Mr. Thomas read notes of case of fracture of the skull with hyperpyrexia, treated with temporary relief by the cold bath.

Mr. Bruce Clarke showed a dissection of a Colles' fracture.

Mr. Benton read a paper on 'Nurses and Nursing.'

November 16.

Mr. Pye showed a specimen of aneurysm of the arch of the aorta, with an aneurysmal dilatation of the arteria innominata; he also showed a specimen of internal strangulation of the small intestine, and read the notes of the case.

Mr. Griffiths read notes of a case of typhoid fever.

November 23.

Mr. Gabb read the notes of a case of œsophageal obstruction, for which gastrotomy was performed by Mr. Callender.

Dr. Wharry then read a paper on 'The Treatment of Cases in which Death appears Imminent.'

Sudden death.—It not rarely happens in cases of impending sudden death that there are a few moments in which something may be done with a hope of averting the calamity; if only one life in a thousand be thus preserved, it is sufficient reason for taking into consideration the treatment of sudden emergencies. Death seldom happens from syncope, asphyxia, or asthenia alone, and it is always necessary to form a just appreciation of the extent to which each of these factors is engaged in the fatal result.

Sudden death from aortic incompetence is by no means infrequent. When death does occur, it is almost instantaneous; the heart stops in an over-distended condition, and our only hope of averting death rests in rapid venesection. In rare instances death, depending upon asphyxia due to acute œdema of the lungs, has followed within an hour of paracentesis thoracis. Rapid withdrawal of the fluid from the pleural cavity causes an influx of blood to the pulmonary capillaries, and the blood being unable to obtain a sufficiently ready exit, acute œdema occurs, as evidenced by urgent dyspnœa, with albuminous expectoration, leading to death. Here again careful attention to the details of the operation is to be insisted upon. Evacuate the fluid slowly, always stopping temporarily when the patient coughs much. Carefully watch the pulse and administer brandy.

If death should threaten after a hemorrhage, the application of Esmarch's bandage to one or more of the limbs may possibly avert dissolution; but one must always guard against removing the bandage rapidly.

When death is threatening from coma, artificial respiration must be resorted to, and maintained with untiring perseverance so long as the heart is beating.

There are certain cases brought to the hospital by the police as drunk. They smell of spirits, and appear to be in

a condition resembling uræmic rather than alcoholic coma. This condition is due to excess of alcohol, with absence of food and exposure to cold. As death sometimes occurs in this state, the treatment must be conducted with care. Wash the stomach out, then inject some warm eggs and milk; apply a large mustard poultice to the chest and warmth to the surface generally. Avoid cold douches, the galvanic battery, and emetics.

In all cases where death is imminent, any treatment adopted should be resolutely carried out.

Mr. Bruce Clarke showed some microscopic specimens.

November 30.

Mr. Bruce Clarke read a paper on 'Some Points in the Treatment of Gonorrhœa.'

After alluding to the common character of this disease, the author explained that his remarks would be limited to such cases as had come under his notice in the Surgery, together with the treatment employed.

After briefly discussing the pathology of the disease, the indications for treatment were then laid down. Patients could be treated by diet alone, by local injection, by medicine, or by all three methods. The practical point to be solved, however, was how best to treat a fairly healthy patient, who either would not or could not lie up for a while.

The author was strongly in favour of the abortive treatment in the premonitory stage. For this abortive treatment he used an injection either of nitrate of silver, gr. v., or tannic acid, gr. iii., coupled with a few drops of the tincture of opium to the ounce of water.

During the acute and subacute stages, especially the former, there was but little to be done except to avoid doing harm by strong injections. Nothing more was to be desired than keeping open the bowels and washing away the irritating discharge.

Once the gleety stage was ushered in, active treatment might again begin. The author spoke very highly of an injection of acetate of zinc, gr. iv., tincture of catechu, m. viii., tincture of opium, m. viii. to the ounce of water.

The author stated his belief that the disease could be cut short in the premonitory stage or in the gleety; but at the acute period treatment to an active extent was only deleterious. The results of treatment in over seventy cases were laid before the Society.

After briefly alluding to the chronic character of many gleets, their relation to stricture, and the means to be taken for their

cure, the author concluded by urging the use of opium as an adjunct to every injection.

December 7.

Mr. Pye showed a specimen of gall stones, consisting of nearly pure crystalline cholesterine.

Dr. Shuter read a paper on 'Obscure Abdominal Injuries.'

He held that those cases in which death occurs shortly after blows upon the epigastrium may be explained in one of two ways. Either there is paralysis of the heart, produced from the blows being communicated through the diaphragm, or there is so much injury done to the solar plexus, that the abdominal blood vessels become so dilated as to be capable of holding nearly all the blood, and that those patients die from anæmia of vital organs. By way of illustration he cited several cases that had come under his notice at St. Bartholomew's Hospital, besides the two cases recorded by Mr. George Pollock, and the one by Sir A. Cooper, where a man died on the spot, following a blow on the epigastrium. He then read notes of Mr. Willett's fatal case of ruptured bladder, and said he felt very strongly that in a case of the kind it was imperative to see it and make a diagnosis at once, and then immediately to open the abdomen and stitch the bladder up, the only positive sign being the capability of passing a sound into the abdomen far beyond the normal limits of the bladder. If the case was either not seen or not diagnosed for about two days, it was then too late to open the abdomen, for it would only make matters worse to do so when peritonitis had set in; but that the patient should be kept entirely at rest, and have a soft catheter open at the tip passed several times a day to prevent the bladder becoming distended. Out of fifty-one cases that are on record, only three have recovered, and only one case of these three (Mr. Chaldecot's) had extravasation into the peritoneal cavity.

He then discussed ruptured diaphragm, and gave an account of a case which lived two days, and in which the stomach and transverse colon were found in the left pleural cavity.

After relating some cases of ruptured liver which had recovered, the rent being slight or subperitoneal, he recorded a case of a boy who lived four hours after being admitted to St. Bartholomew's Hospital with a ruptured stomach. The boy had the ordinary board-like hardness of the abdominal muscles, and complained of smarting in the abdomen after taking brandy.

Mr. Shuter showed how closely this case accorded with the symptoms of the one which Mr. Moore has placed on record.

The paper closed with a few suggestions in cases of ruptured intestines.

December 14.

Dr. Wharry showed a specimen of malignant disease of the bladder.

Mr. Adams read a paper on the 'Clinical Value of Alcohol.'

A brief account of the physiological action of alcohol was given, reference being made to Dr. Brunton's paper for a fuller discussion of the subject. Its use in collapse from injury, and in capillary bronchitis, and in allied diseases of children, was insisted on, while it was explained that its use in such cases was at least in part due to its preventing the necessity of the oxygenation of the blood. The use of alcohol in the pneumonia of adults, and in post-partum hemorrhage was then discussed. In pyæmia and septic diseases its use was explained from the results of physiological experiments on animals. The indications for its employment in typhoid and other continued fevers were given.

The uses of alcohol having been summed up, the subject of its abuse was entered into. To show that alcohol was not necessary for sustained exertion during health, Dr. Parkes' essay on the spirit ration in the Ashantee expedition was quoted, as also the experiences of the recent Arctic expedition, and Dr. Fothergill's writings. The loose and indiscriminate way in which alcoholic liquors are sometimes ordered by medical men was strongly condemned. Whisky was stated to be not so harmless a form of alcohol as is generally believed, on account of the frequency with which it contains fusel oil. The various merits and demerits of brandy, gin, rum, and absinthe were mentioned.

Total and sudden abstension from alcohol was held to be a perfectly safe procedure for habitual drinkers. The question of State regulation of the liquor traffic was then discussed. And in conclusion the importance of the subject, and the power for good or evil which the employment of alcohol placed in the hands of the medical profession, was urged on the notice of the Society.

January 11.

Dr. Brunton read a paper on 'Dyspepsia.'

He began by saying that the enjoyments of the incidents of life depended upon the judicious distribution of labour, rest, and relaxation; but when the relation between these becomes disturbed, life becomes filled with pain instead of pleasure. So with the stomach and its functions, if the supply of food is regular in

quantity and good in quality, and the energy of the system is not interfered with in carrying out digestion, eating becomes a pleasure and digestion is no labour, but if anything prevents the just apportionment of these matters, nausea is felt instead of hunger, and comfort after a meal gives place to pain; in other words, we have dyspepsia. He then gave a short sketch of the growth of our knowledge of digestion; how the ancients thought that the food taken into the stomach was simply dissolved by moisture and warmth; how, later on, a kind of putrefaction was supposed to take place, or that the food was ground into pieces by the movements of the muscular walls of the stomach, like in the gizzard of a bird. It was not till 1840 that the digestion of food was finally attributed to the action of pepsin and the presence of hydrochloric acid. We owe a great deal of our knowledge of digestion to the observations of Dr. Beaumont on the Canadian, St. Martin, who had a gastric fistula. After describing several of these observations, the writer discussed the causes of the feeling of hunger, which he attributed, first, to a certain condition of the capillaries and lymphatics; secondly, to a condition of the system requiring for its relief absorption of food into the blood. He then touched upon the diagnostic value of the state of the tongue; first, the tongue with a thin white fur, going with a good appetite, and, in St. Martin's case, accompanied by excoriations on the mucous membrane of the stomach; secondly, the tongue with a yellowish fur, going with loss of appetite and deep red patches in the stomach; thirdly, the coated tongue, with no appetite and pustules on the gastric walls.

The writer then considered the causes of dyspepsia, the irritations of the stomach being divided into those caused by excessive quantity and by improper quality of food. A mild attack required a limited, bland regimen only; chronic dyspepsia is generally caused by a persistence of the causes of an acute attack, the best treatment being that by bicarbonate of soda combined with a vegetable bitter; atonic dyspepsia is due to a debility of the circulation and nerve apparatus, and must thus be treated, strychnine being a most useful drug for fulfilling this indication.

January 25.

Mr. Macready showed a dissection of a fracture in the neighbourhood of the ankle-joint with dislocation.

Dr. Verco read a paper on 'Delirium.'

Introduction.—One of the reasons that leads me to bring this subject before this honourable Society is, that it is one on which I have not been able to find anything like a full article in any

of our medical text-books. True, one can cull not a little information respecting the matter in the shape of scattered fragments; but it is necessary to gather up these fragments if nothing is to be lost, and work them into a united whole. And this will not be found by any means useless for practical purposes.

It may be suggested that delirium is but a symptom, and not a disease, and that hence it was not to be expected that it should form the subject of any very elaborate treatise. But let it be remembered that what we as practitioners want, is not only to know the symptoms corresponding to a disease, but what are the diseases corresponding to a symptom. Patients always come to us complaining of a symptom, never of a disease; and, as physicians, we have from symptoms to discover their disease. To do this, one must know what are the chief diseases that can cause it, and from among these to choose out that one which is compatible with whatever other symptoms we may discover. Hence it is well to consider the symptoms of disease, each one separately, and to group around it the complaints which may be its cause.

Causes.—Delirium is but a symptom, nevertheless it has a very great many causes. There are many predisposing circumstances, which of themselves would never produce it, but yet bring about such a condition of system that delirium is more liable to occur.

And first of age: we all know how much more readily it arises in the young; how a child will be raving day and night with an attack of pneumonia that in an adult would cause no more than a little nocturnal wandering; and how at the onset of scarlet-fever the headache of the man is equivalent to smart delirium in the child. This is attributed to greater nervous impressibility, just as infantile predisposition to whooping-cough and other such affections is explained. We all recognise the greater liability, and as a consequence deem the delirium of children as not nearly so unfavourable, from the point of prognosis, as that of adults.

Sex, too, is not without influence. We expect to find the female, from her greater nervous impressibility, more prone to delirium. The hysterical form, which I think we might recognise almost as a distinct variety, occurs in women only.

Social condition, on the same ground, is alleged to act in a similar way. The higher classes are more liable to delirium than the lower. Their nervous system, being more highly developed and of more delicate organisation, has its equilibrium more easily disturbed.

Nervous tendency, too, may be enumerated, from whatever cause arising. Hence those unfortunates who have a neurotic taint hereditarily, whose ancestors were insane, epileptic, hysteric, neuralgic, &c., as well as those whose nervous systems have been damaged by cerebral injury, if they drink what to others would be a moderate draught become delirious madmen. So also pneumonia or other disease in a man whose nervous system has been undermined by alcoholic excess, is generally attended by violent delirium.

Diurnal influence.—Who has not noticed how a patient comparatively, if not completely, quiet all the day, grows restless towards evening, and all night long is incessantly delirious, yet falls into a quiet sleep as the morning dawns again? What is the cause of this? Is it connected with the removal of the curbing influence of the sights and sounds of day? Is it the nightly rise of temperature found in health and exaggerated in disease? What is the mysterious cause?

The *exciting* causes may be grouped under five heads.

1. *Anæmia.*—Thus in all the chronic wasting diseases, such as cancer of the stomach or elsewhere, we find that towards the end, as the patient's strength gradually diminishes, so he becomes subject to delirium. In those cases where the anæmia comes on very gradually, and the weakness of body proceeds *pari passu*, we usually find that the affection assumes the languid, low, muttering prostrate form.

In albuminuria, again, delirium is by no means uncommon; and when we remember the excessive pallor of the patient, and the great drain of albumen from his system, we cannot but attribute the mental derangement in great part to the anæmia. In this disease it may appear in various forms; it may gradually develop, as dimness of vision comes on, as a muttering wandering, or signs just like those of delirium tremens from alcoholism may arise; or the patient may have a series of uræmic fits; be comatose for hours, to awake a restless, speechless madman, to recover within a few days all his powers, but with the memory of those days completely blotted out.

After many acute diseases, such as pneumonia, a patient who has done well, and is seemingly out of danger, may suddenly become delirious. This is the 'delirium of collapse,' apparently due to cerebral anæmia, from palsy of the heart, and is very fatal in hemorrhages, profuse losses of blood from the lungs, or in fact from any other part. These are the best of all instances of delirium from anæmia, because no other causes co-exist. Thus I have known a man quietly asleep burst a blood vessel in his lungs, and within five minutes be a raving satyr, obliged to

be held down by two or three attendants, and, strangely enough, within half an hour he was as sensible and conscious as ever, doubtless from restoration of his cerebral circulation.

2. A second cause is *hyperæmia.* We have a pure, uncomplicated instance of this, at least we are told so, in the delirium of the Bacchanalian dancers, where the cerebral circulation is excited by excessive muscular exertion. But it can scarcely be doubted that in these instances it was exercise mingled with wine.

Still we see the effect of hyperæmia in the delirium of cerebral congestion, from whatever cause arising, most markedly in meningitis, encephalitis, and any other intra-cranial inflammations. The delirium which is so common when there is embarrassed flow out of and consequently into the right heart, leading to passive congestion of the brain, ought rather to be attributed to cerebral anæmia, inasmuch as the aerated blood enters the brain in much diminished amount, and so disturbs the function of the brain.

3. A third cause is *toxæmia*, the circulation of poisonous materials through the brain. This is doubtless in part also the explanation of its occurrence in albuminuria, when there is a collection of effete matters in the blood in the onset of the acute specific infectious fevers, in poisoning by many drugs, as belladonna, &c., in acute alcoholism, and delirium tremens.

4. A fourth cause is what might be called *pyræmia,* commonly known as hyperpyrexia, the circulation of high-temperature blood. This, however, is almost always connected with a rapid action of the heart, and hence with cerebral hyperæmia, so that few or no cases of pure pyræmia can be quoted.

5. A last cause may be given as *nervous shock.* This is an alleged cause. But though a very wide term, the only case I have seen which could bear this explanation was that of a woman, otherwise healthy, but extremely nervous or hysterical, who, on being refused permission to see her daughter, who was ill, became after a manner delirious, and was evidently quite irresponsible for her words and deeds, wrote a petition to the Home Secretary, &c. Doubtless this is the cause of many cases of insane delirium.

Varieties.—As one might expect in an affection of the mental processes, there are several varieties of delirium. It is very questionable, however, whether some of the names applied mark out distinct kinds, and not merely degrees.

1. There is the *low muttering delirium,* which describes itself, and which, occuring in fever, has had conferred on it the name of febris nervosa stupida.

It would seem generally to be found where there is great weakness and prostration, as in advanced typhoid, &c., or where

there is only a feeble excitement, or disturbance of the equilibrium of the nerve elements of the brain.

2. There is *delirium ferox*, where the patient is raving, noisy, and violent, requiring restraint. It would seem to arise in consequence of considerable nervous disturbance in a vigorous subject; but it is found even in the weak, when the disturbance is very great. When it is chiefly expressed in words, we have the *febris nervosa versatilis*, but to show how closely connected is this variety with the last, we have only to state that the muttering delirium may occur in the day, and the noisy garrulous form in the night, in the same patient.

3. There is *delirium tremens*, where the mental derangement is accompanied by trembling of the limbs. It is almost always the result of hard drinking. The delirium is generally suspicious, quiet, busy, and attended with horrible hallucinations. It must be remembered, however, that in alcoholic delirium there may be no trembling whatever, and that a trembling delirium not to be distinguished from the alcoholic may occur in other diseases. It is said to have been observed in typhus; and I have witnessed the same occurring in chronic albuminuria; the patient having been in Hospital some months, so that it could not be attributed to alcoholic excess.

4. There is *traumatic delirium*, coming on after the receipt of an injury, and manifesting itself under several forms. As usually interpreted, it seems to be nothing else than delirium occurring in and due to the surgical fever set up by the wound.

5. Then there is *delirium exaggerans*. This is a well-recognised form. In which a person has the highest notions with the smallest capabilities. He imagines himself the begetter of myriads while as impotent as a eunuch, fancies himself a very Hercules though he cannot rise from his bed, and bestows worlds upon his friends when he has not a penny to bless himself with. This curious variety is said to be found in general paralysis, in disseminated sclerosis of the brain and cord, and to be a frequent sign of commencing insanity.

Pathology.—If you ask me for a definition of delirium, I cannot give one, at least one that would include everything that is and exclude everything that is not delirium. The etymology of the word could be furnished; but that falls far short of the meaning of the thing. And if a definition is so difficult; how much more so a description of its pathology. Still we can inquire somewhat into its relations and physical concomitants.

In delirium we find several factors, each of which helps to make up the whole, but each of which alone might not by some be considered entitled to the full name 'delirium.'

First, We may observe that there is faulty perception. Thus a patient may tell you that the tinware of the ward seems to him like a shoal of fish, or he may mistake one of his nurses for his own wife, or fancy that the blood gushing from his own nostrils is pouring from the wounds of his next bedfellow. Here there is an impression on his organ of sense, but his perceptive centre is at fault. This is an *illusion.*

Again he may be in total darkness, unable to see any adjacent object, and yet from his words one learns that he sees himself surrounded by numerous persons and things, and sees all with the greatest vividness. Yet are they all unwelcome creatures of his imagination. Here there is no irritation of his perceptive centre through an organ of sense, but something is irritating his perceptive centre and producing in it the same condition that would have resulted from a sensory impression on it. There is perception without any external object to cause it, an *hallucination.*

Thirdly, You will find a patient who sees the tinware as though it were fish; he has an illusion, but yet knows that it is tinware, and can correct the erroneous perception by the other senses—by memory, or by reason. This patient has no *delusion.* If he believed firmly that he saw fish, and could not correct his illusion, we should say he had *delusion* as well.

Again, we find patients who have illusions which operate upon their minds, but we know nothing about them unless we institute special inquiries with the patient. Then we learn that there is a sort of dreamy disturbance of the thoughts only. This is the first stage of delirium, that which often occurs in commencing cerebral congestion from heart disease, in the fever of common colds, &c.; hence we inquire of the patient whether he is much troubled by dreams.

In the next stage we find the patient growing talkative, and the garrulity may vary from an occasional whisper during sleep up to uninterrupted and noisy utterances. Here a very much more profound effect is produced. And, lastly, we find the locomotory apparatus involved; from a little jactitation and restlessness, where the patient rolls in his bed, up to the condition in which he leaps out like a madman, and flies through the ward as for his life. In delirium tremens especially we find this very common. The poor wretch cannot be kept in bed. No sooner is the back of the attendant turned, not that he even waits for that, than he is up pacing his room, pulling about his bed-linen, building his bed in every corner, only to pull it down and pile it up elsewhere. I have seen it just the same after albuminuric fits. Here too we ought to place the picking of the

bed-clothes, so commonly seen, and dignified with the double name of carphology and floccitatio.

There would seem, in fact, to be a sort of delirium agens; not one that must think or that must talk, but must *do*. Thus you will find two persons, of about equal strength, one of whom gabbles incessantly, while the other cannot be restrained from being up and about, but says not one word, even though you seize him and force him down on his bed. It seems almost like a distinct variety. It may be due simply to a peculiarity in the disposition of the patient, but it does seem to exist.

Here let me suggest that dreaming is a mild form of delirium, from which it cannot otherwise be distinguished than by its mildness. Natural sleep in a perfectly healthy body is sleep undisturbed by dreams. Were our systems just as they should be, we should not dream. You will notice that exactly the same circumstances that predispose to dreams predispose to delirium. They are more common in children, in the higher classes, in the nervous.

Moreover, we all know that before the advent of the easily recognised delirium come dreams, as in heart disease.

Yet, again, they are indistinguishable. Stand by the bedside of a child, hear it talking in the night; is it dreaming, or is it delirious? Both.

Dreams, too, are most vivid, most rapid, most persistent, when we are out of health or physically disturbed. The proverbial mince-pie for supper is a fruitful source through causing gastric disturbance. The headache from any cause arising, points to the truth that all dreams are due to physical derangement, and are but a milder type of delirium.

Lastly, see how alike are the manifestations. They are the same illusions.

It would seem now as if in delirium there were two derangements:—

1. A perversion of the perceptive faculty.
2. An excitement of the reasoning faculty.

Thus, in order that an impression from one object should cause the perception of another, as in illusion, or in order that, with no impression from an object at all, there should be the perception of it, there must be a perversion of the perceptive centre, and the second must be an aggravated form of the first. Now for the proper action of the perceptive centre we find a certain state of the centre essential; if that state is altered, its action will be deranged. This normal state depends upon its nutrition, and this, of course, almost solely upon the blood. A

certain amount of blood, a certain quality of blood, a certain heat of blood.

Now it is to be noticed that this centre has a certain stability, consequently so long as the blood supply does not alter very greatly from the normal, the function of the centre is not deranged. If, however, there is a sudden variation to any great degree, then there is perversion. Thus I have already referred to the case in which a man sleeping quietly burst a blood vessel, and almost immediately began to rave in delirium; there was a sudden upset in the cerebral circulation, which the centre could not withstand. When, however, the blood current was again equalised, and this took place in about half an hour, then the stability of the centre was such, that the normal powers of perception completely returned, though the circulation still must have been somewhat deranged. This stability seems to increase with advancing years; hence we find that a very slight variation in the blood current of children is sufficient to produce derangement of their mental faculties.

It would seem, too, that these centres have a very great power of accommodating themselves to alterations in the condition of the blood, provided that this alteration comes on slowly, and so allows time for the accommodation to take place. Thus I have seen patients far more anæmic than the one above quoted without any delirium at all. It was the sudden disturbance in the nutritive supply which caused so rapid and sudden an effect. But there is a limit to this accommodation of the centre to changes in its blood supply; hence when these changes become very profound, however slowly brought about, the power of accommodation can no longer keep pace with the change, and the patient grows delirious, as in wasting diseases like cancer, &c. This perversion, we have seen, can be brought about by alteration either in the quantity of the blood (anæmia or hyperæmia), or in the quality of the blood (toxæmia), in the condition of the blood (pyræmia), and lastly by direct irritation of the centre itself, as in nervous shock.

But this perversion is not all. A man may fancy he sees one thing when he sees another, and yet not be accounted delirious. There is a second derangement, an excitement of the reasoning, thinking faculty.

Hence we find that when the patient is asleep, and when therefore all his mental faculties ought to be slumbering, he is mentally awake; he is thinking, he is living an imaginary life, and often at such a rate as would be actually an impossibility. His emotions are so strong that they burst forth in laughter or in tears, or in excited gestures. All this shows an intense irrita-

tion of his thinking centres, and as a matter of fact are accompanied by an abundant excretion of the products of their disintegration.

These two may be combined together in any proportions, though as a rule they vary equally the one with the other. Where there is much perversion of perception there is generally much excitement, though this is not always the case.

Now as to the cause of the difference between the low muttering delirium and the fierce boisterous kind. The former variety seems to be due either (*a*) to great prostration of the patient's strength, or (*b*) to but slight irritation of the centres; the excited form (*a*) to moderate irritation in a vigorous subject, or (*b*) to intense irritation of the centres. That it is rather, however, dependent upon the intensity of the irritation than upon the patient's strength, is shown by the fact that a low muttering delirium in the day will become a noisy boisterous delirium at night, whilst the patient's strength cannot have proportionately increased in the meantime.

Before concluding the pathology I would make one suggestion. A patient came in with hæmoptysis, and while he was bringing up blood in large amount, he had an epileptiform fit, and was well again in a few minutes. The next day he brought up a second large quantity, had a second fit, and recovered from it in a few minutes. He had no delirium at the time, and very little afterwards. He never had had a fit before. Might we not suppose that this fit was the equivalent of the other man's delirium; that in the one case the man's psychical centres were thrown out of their stable condition, and in the other the man's motorial centres? The case of an albuminuriac may be put by the side of this. A man had uræmic fits for twelve hours, then coma for thirty-six, then delirium, in which he did not speak a word, but was ever out of bed. If the fits were due to œdema of the motorial centres, might not the delirium subsequent to it have been due to an œdema of the psychical centres, and of that part nearest to his motorial, and hence his peripatetic proclivities, and hence might not the delirium be the neurotic equivalent of the fits?

If so, is insanity the analogue of total chorea, and monomania of hemichorea, or of chorea localised in one set of muscles.

Treatment.—In the treatment of delirium the best results will follow prescriptions according to causal indications.

Thus, if it be due to anæmia, lowering measures are inadmissible. On the contrary, one gives nutritious nitrogenous diet in the wasting diseases, either by the mouth if it can be

taken, or by the rectum if it cannot. Often, too, as in the wandering albuminuriac, a good dose of brandy, one or two ounces, given in the evening, will quiet the nervous system, and procure a night's sleep. So in the delirium of collapse, one administers carbonate of ammonia in five-grain doses, and tincture of opium in m. v. doses every four hours, the laudanum in these small quantities acting rather as a stimulant than as a sedative.

If it be hyperæmia, then the treatment must be the very opposite, with a view to diminish the amount of blood in the brain. Here one shaves the head, if the disease be inflammatory, and applies to the scalp evaporating lotions or the ice-bag. A good stiff purge, too, by causing derivative action to the intestines, and drawing off a quantity of fluid from the blood through the bowels, considerably lessens the intra-cranial pressure. This will not infrequently relieve in the same way the delirium due to embarrassed right heart, by diminishing the amount of blood to be passed round the system.

When it is toxæmic the treatment is not so satisfactory. The indication is to remove the poisonous material from the system; but this is generally almost if not quite impossible. For in chronic disease such as nephritis, or jaundice, &c., the poison has been increasing in spite of all our long-continued efforts to eliminate it. When, therefore, it has become so great in amount as to upset the mind, it is not to be supposed that by any measures, however heroic, we shall be able to get rid of it, directly the delirium points strongly to that course. We may use diuretics, diaphoretics, and purgatives, but they will be of but little avail, and are but a forlorn hope.

When, however, they are in excess in the blood because they have been taken as poisons through the stomach, or have accumulated rather from excessive formation in acute diseases, as typhus or typhoid, than from deficient excretion, then we may set the different emunctories to work with some advantage and some hope of success. To enumerate the different drugs would be tedious.

If the cause be pyræmia, we have a fairly efficient remedy in the cold bath or in cold packing. Perhaps it would be no easy matter to bathe delirious patients, nor have I had any experience in bathing them. But those who have used this remedy for high temperature simply cannot but have noticed how drowsy the patient becomes after a bath, who before was wakeful and restless. Hence one would presume that its effect would be sedative upon a delirious patient and beneficial.

Next as to indications from the symptoms manifested.

Since there is evidently considerable cerebral excitement, rational treatment would consist in the exhibition of sedatives.

Thus full doses of opium or morphia may be given. This, however, is inadmissible in many cases, as in albuminuria, whether it exist as the primary disease, or only as a complication of the causative disease, be this typhoid, erysipelas, an injury, or aught else. So also in children, one would hesitate to give controlling doses lest the effect should be rather greater than was intended. A far safer remedy is the hydrate of chloral, which can be given in twenty, or even forty grains, and repeated if required. This would be preferable in those cases where there is cerebral hyperæmia, as it is supposed to act by producing anæmia of the brain; but it will act even in anæmic delirium. Bromide of potassium is also recommended, but I have no experience of its use here. Maybe it would be efficient in the milder cases and in children. Chloroform by inhalation I have never seen administered for delirium; sometimes, it is said, the patient is worse after he recovers from its effect than he was before, and in severe disease it would be rather a dangerous remedy.

When the delirium is exceedingly violent, it is advised to give with the nervous sedative a cardiac depressant as well, such as antimony or digitalis. Moreover, the antimony is said to be preferable when there is structural disease of the kidney, as it acts rather upon the skin, and digitalis when there is no kidney mischief, as it is a powerful diuretic. And here one might mention that digitalis alone is used by some practitioners in the treatment of delirium tremens, not in the usual doses of m. xv. or m. xx., but in single exhibitions of half an ounce of the tincture, a second dose to be taken in four hours' time of half an ounce. But I have no experience at all of antimony or digitalis in delirium ferox, nor of digitalis thus boldly given.

By one or other of these remedies we may generally succeed in quieting the most boisterous patient. All that we require to know is, that with them we can quiet *any* patient if we like to push them far enough; and, finally, that it is not by any means necessary to *try* and quiet every patient's delirium.

February 1.

Mr. Darbishire showed a working model of a form of anterior leg-splint.

Dr. Hart showed two specimens; the first, a liver ruptured through external violence; the second, an aneurysm of the aorta,

in which the wall of the sac was composed largely of connective tissue, and greatly compressed the lung.

Mr. Weiss then showed a patient convalescing from an attack of traumatic tetanus.

A series of cases of tetanus occurring in the wards of the Hospital was then read by Mr. Weiss and Mr. Pye, and the latter showed some microscopic sections of spinal cord and sciatic nerve from a case of traumatic tetanus.[1]

On the motion of Dr. Wharry, the discussion was adjourned till the following meeting.

February 8.

Dr. Maberly showed a fœtus at full term, delivered after craniotomy had been performed, the mother suffering from cancer of the uterus. Turning was effected before delivery.

The adjourned discussion on tetanus was concluded.

February 18.

Mr. Pye showed a woman suffering from lupus exedens, in whom a considerable portion of the face had been destroyed.

Mr. Walsham read a paper on 'Palpation by the Rectum.' He treated the subject under five headings—

1. The size of the hand that could be safely introduced.
2. Method of introduction.
3. The distance the hand could be passed.
4. The structures that could be recognised in the healthy state of the parts.
5. Diseases in which palpation was of service.
6. Conclusions.

After briefly discussing the first four points, for fuller details of which he referred to his paper in the reports of the preceding year,[2] and to a short article which he had contributed to Mr. Holden's 'Landmarks,' Mr. Walsham passed on to consider the fifth heading, *i.e.*, diseases in which palpation by the rectum was of service. He stated that first and pre-eminently it was useful in the diagnosis of doubtful affections of the pelvic organs, of the uterus, ovaries, bladder, and ovarian ligaments. He referred particularly to its use in estimating the condition of the pedicle in ovarian tumours, and related a case in which, in the dead body, he distinctly determined the peritoneal connections of a small ovarian cyst.

[1] The specimens were from a case of traumatic tetanus under the care of Mr. Langton, and in which the sciatic nerve was stretched.

[2] *Vide* St. Bartholomew's Hospital Reports for 1876, vol. xii. p. 223.

It permitted the ready removal of foreign bodies from the rectum and the performance of operations on fistula high up; it was also useful in diagnosing strictures, especially those in the upper part of the rectum, and lower part of the sigmoid flexure.

It was of aid in the diagnosis of aneurysm of the abdominal and pelvic arteries, and in distinguishing between aneurysm of the pelvic arteries and cancer of the pelvic bones.

It was especially valuable in determining whether in gluteal aneurysm the sac encroaches upon the pelvis through the great sciatic foramen, so as to involve the pelvic portion of the vessel.

It was of great service in the diagnosis of obturator and ischiatic herniæ, as the obturator foramen and ischiatic notch could be thoroughly explored by the hand in the rectum. It was also of use in the diagnosis of abdominal tumours and stone in the kidney.

Mr. Walsham also suggested that benefits were likely to be derived from the adoption of this method of examination in obstetric practice, and finished his remarks by stating the conclusions he had drawn from his present experience.

March 1.

Mr. Macready showed a specimen of ruptured spleen in which considerable local repair had taken place,

Dr. Maberly read a paper on 'Placenta Prævia.'

He urged the importance of the proper understanding of placenta prævia and its treatment, since the disastrous consequences which frequently follow can be very considerably controlled by appropriate measures.

He contrasted the two methods of treatment now in practice, viz., that by turning and rapid emptying of the uterus, and that by separation of the placenta from the uterine wall, and dilatation of the os as recommended and practised by Dr. Barnes.

He cited two cases which had occurred recently in the out-patient practice of the Hospital in which each method had been used.

In the first case there had been considerable hemorrhage for at least six hours before his arrival, which was attributed to a fall some little time previously; but the patient was not in an exhausted condition.

The os would only admit one finger, and the placenta was felt just within and to the left side, with the head of the child beyond.

There had been no uterine contractions, and the patient was about a fortnight from her full time.

Under chloroform he separated the placenta as far as the finger would reach, performed bipolar version, and then ruptured the membranes.

The os uteri was very rigid, but by steady pressure of the fingers in the form of a cone, dilatation was effected, and delivery accomplished after some difficulty, the child being born dead.

The patient was in an alarming state of collapse, and was with great difficulty restored by stimulants and other means. She ultimately, however, made a good recovery, and was convalescent in a month.

The second case was one in which hemorrhage occurred two days after a fall, to the amount of about half a pint, in a healthy multiparous woman. He was sent for in consequence, and found hemorrhage going on, and without uterine contractions.

Os uteri was small and rigid, with the placenta attached to the left side and protruding through it; beyond was the head of the child.

He separated the placenta as far up as possible, ruptured the membranes, and gave f. ʒij. of extract of ergot, placing a binder tightly on the abdomen.

Uterine contractions were thus set up, and the os began to dilate. There was only slight hemorrhage after this, which ceased on again separating the placenta and stimulating the uterus to further contraction.

Labour went on steadily without further hemorrhage, but was retarded by the large size of the head. The child was still-born.

The placenta came away of its own accord, and the uterus contracted readily.

The author then endeavoured to show that the cessation of hemorrhage was due in all cases of placenta prævia to uterine contractions, which ought to be rapidly set up by artificial means.

This was to be done by endeavouring to dilate the os uteri, rupturing the membranes, and giving ergot.

The dilatation was accelerated by separating the placenta from the uterus with the finger, which thus accomplished in a moment what the unaided efforts of the uterus would take many hours to do. This separation must take place sufficiently far up to admit the presenting part, which, if it be the head, aids in arresting hemorrhage by acting as a firm plug through the medium of the placenta.

In contrasting the treatment and the results of these two cases the following rules were recommended, according to the

kind of case with which one had to deal, and which he divided into two classes—

I. Those in which hemorrhage was not so alarming as to call for immediate delivery.

II. Those in which rapid delivery is necessary from the severity of the hemorrhage, and from the exhaustion occasioned by it.

In the first class, endeavour to bring about uterine contractions by—

1. Separating the placenta from the uterus, and enlarging the os with the finger.

2. Rupturing the membranes.

3. Giving ergot.

4. Effecting dilatation by the fingers, or by the use of dilating-bags if necessary.

5. Applying an abdominal bandage to aid in retaining the head of the child at the os uteri.

In the second class, version and rapid delivery.

There were, however, a small number of cases in which the os is so rigid as to defy any of these means of dilatation, and here, where the hemorrhage is excessive, you must plug the vagina tightly, and endeavour to support the patient's strength, leaving the labour to take its own course.

One of the great advantages of the first method is that chloroform is not necessary, but it must be given in nearly all cases of turning, and whilst delaying treatment during its administration, produces also great depression, and from the vomiting so frequently caused prevents restoratives being retained by the stomach in case of collapse.

March 8.

Mr. Clubbe and Dr. Verco were appointed to audit the accounts of the Society.

Dr. Champneys showed a female infant suffering from ectopia vesicæ, who presented several points of interest, namely, (1) its sex; (2) the imperfect condition of the vagina; (3) the probable bicorned condition of the uterus; (4) the absence of the symphysis pubis; (5) the apparent openings of the Fallopian tubes into the bladder.

Dr. Hall read a paper on neuralgia.

Dr. Anstie's definition of neuralgia was quoted. The ætiology of the disease was the first question discussed. Out of sixty-four cases, to which reference was made, fifty-six were women and only eight men.

[1] *Vide* Article IX. p. 83 of present volume.

Prosopalgia was found to be more frequent in women and sciatica in men.

Hereditary predisposition was alluded to, and age was pointed out as being an important factor, children being remarkably free from it. It is very frequently met with at the adult period of life, especially among anæmic and chlorotic young women, but it is, as a rule, in the old or those prematurely old, that the intractable forms of neuralgia are met with. Worry and anxiety were stated to be the most marked of the predisposing causes. The necessity of paying attention to the direct exciting causes, such as caries of the teeth, &c., was insisted upon. The varieties of neuralgia may be classified according to their local distribution—

I. Superficial neuralgiæ.

II. Visceral neuralgiæ.

The superficial varieties may subdivided into—

(*a*) Neuralgia of the fifth nerve.
(*b*) Cervico-occipital neuralgia.
(*c*) Cervico-brachial neuralgia.
(*d*) Intercostal neuralgia.
(*e*) Lumbo-abdominal neuralgia
(*f*) Crural neuralgia.
(*g*) Sciatic neuralgia.

In the treatment of neuralgia the first thing to be done is to relieve the patient of his pain, be it only for a time, so as to afford an opportunity of treating the constitutional cause, if such there be, at the bottom of the disease.

He gave the particulars of a case, in which the presence of two minute portions of stumps had been overlooked, and the disease was not cured till these were removed; he deduced from this case the necessity for a most careful examination of the condition of the teeth in trifacial neuralgia.

Notes of three cases were read to prove what a speedy and potent remedy croton chloral in five-grain doses three times a day was. The tincture of gelseminum in twenty-minim doses had been found of use; but it was not so uniformly successful as the croton chloral. One of the most useful drugs to begin the treatment of a case of trifacial neuralgia is the chloride of ammonium in half-drachm doses, and it has the advantage that if six doses fail to give relief, it is useless to push the remedy further. Out of eighteen cases, ten were cured, one was unrelieved, and seven did not return a second time; and inasmuch as this remedy produces its effects very speedily, there is every probability that some of these also were cured.

Mention was made of the fact that chloride of ammonium with

a little perchloride of iron formed the basis of a patent medicine for the cure of tic-douloureux.

Among the local applications, the mixture of equal parts of camphor and chloral hydrate gave relief within five minutes in two cases. The aconite liniment was mentioned as occasionally of use.

In the treatment of sciatica it was found advisable to begin with a brisk purge, as the pain is often kept up by a loaded state of the rectum, and then the Haustus quiniæ c. potassii iodido with the application of a blister eight inches long by two broad along the course of the nerve.

Quinine in full doses was recommended in supra-orbital and frontal neuralgia, and in neuralgia generally if dependent on malarious influences or occurring periodically. Arsenic was said to be useful in occipital neuralgia. Iron, besides its action on the blood, also seems to act as a direct nervine tonic, and it is of course largely used in cases of neuralgia occurring in anæmic and chlorotic subjects. The carbonate is a very favourite preparation for this purpose.

In severe and intractable cases of neuralgia the hypodermic use of morphia is unrivalled for procuring sleep and breaking the habit of the pain, so as to give time for other treatment; the addition of a minute quantity of atropia to the morphia, say the $\frac{1}{120}$ of a grain of atropia with the $\frac{1}{6}$ of a grain of the acetate of morphia, appears to prevent the morphia acting as a diaphoretic, and consequently increases its anodyne effect.

Remedies such as the continuous current, acupuncture, and other surgical procedures, were only alluded to casually.

As regards general treatment, the patient must be got into the best possible condition of health.

With regard to diet, the patient should be ordered the most digestible articles. Pastry, sweets, uncooked fruits, &c., are to be eschewed, whereas fatty articles, butter, milk, cream, oil, and especially cod-liver oil are to be highly recommended.

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.		Junior.	
Foster Prize—	P. A. Steedman.	*Treasurer's Prize*—	C. J. Bamber.
2.	H. J. Steeles.	2.	C. B. Lockwood.
3.	R. H. A. Schofield.	3.	M. Prickett.
4.	W. J. Hames. / G. O. Mead.	4.	D. A. Coles.
6.	A. R. Anderson.	5.	C. A. D. Clarke.
7.	T. Wilmot.	6.	G. Coates.
		7.	E. C. Bousfield.
		8.	R. J. Colenso.
		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.	Junior.
Foster Prize—G. Coates.	*Treasurer's Prize*—C. C. Shepherd.
2. W. Graham.	2. A. J. Wharry.
3. C. Lockwood.	3. H. C. Nance.
4. C. J. Bamber. M. Prickett.	4. W. Outhwaite.
6. G. P. Sylvester.	5. G. H. Barling.
7. E. C. Bousfield.	6. C. F. Cuthbert.
8. Allen Dingley.	7. A. Franklin.
9. G. W. P. Dennys.	8. A. A. Bowlby. G. L. Pardington. K. Townsend.

EXAMINATIONS, 1876-77.

Lawrence Scholarship and Gold Medal—

(No Candidate).

Brackenbury Medical Scholarship—

W. L. Heath, P. A. Steedman, } Equal.

Brackenbury Surgical Scholarship—

G. O. Mead.

Senior Scholarship in Anatomy, Physiology, and Chemistry—

R. Gill.

Open Scholarship in Science—

A. M. Marshall, S. Nall, } Equal.

Preliminary Scientific Exhibition—

E. Clarke.

Jeaffreson Exhibition—

W. J. Collins.

Kirkes Gold Medal—

W. L. Heath.

Bentley Prize—

W. S. A. Griffith.

Hichens Prize—

W. Wickham.

Wix Prize—

(Not awarded).

PRACTICAL ANATOMY.

Senior.	Junior.
Foster Prize—A. A. Bowlby.	*Treasurer's Prize*—J. Barratt.
2. G. E. Fooks. C. C. Shepherd.	2. J. E. Risk. W. T. Wyatt.
4. H. C. Nance.	4. J. Harper. R. Jones. S. Westcott.
5. H. T. Preston.	7. E. Clarke.
6. A. J. Wharry.	8. C. S. Spackman.
7. M. Pearless.	
8. W. T. Freeman.	

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., LL.D., 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. Morrant Baker, Mr. Marsh.

Physician-Accoucheur—Dr. J. Matthews Duncan.

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. Bridges, Dr. Champneys.

Medical Registrar—Dr. 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. Morrant 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. Matthews Duncan.

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. Morrant 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. Keetley, Mr. Shuter.

Mechanical and Natural Philosophy—Mr. Graham.

Practical Physiology—Mr. Kidd.

Assistant Demonstrator—Mr. A. Cantin.

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 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 1876.

BY

THE MEDICAL REGISTRAR,

FRANCIS H. CHAMPNEYS, M.B. (Oxon.)—M.R.C.P.;

AND

THE SURGICAL REGISTRARS,

HENRY T. BUTLIN, F.R.C.S.,

AND

J. MACREADY, F.R.C.S.

LONDON:

HARRISON AND SONS, ST. MARTIN'S LANE,

Printers in Ordinary to Her Majesty.

1877.

PREFACE.

The following changes have been made this year in the Medical Tables :—

The arrangement of Table I has been brought into accordance with the "Nomenclature of Diseases," published in 1869 by the authority of the Royal College of Physicians.

All the figures referring to Deaths have been printed in black type.

No alteration is made in the Surgical Tables.

CONTENTS.

SURGICAL REPORT—

ST. BARTHOLOMEW'S HOSPITAL.

1876.

Number of Beds in Medical Wards (including 14 for Diseases of Women)	230
„ „ „ Surgical „ {including 7 for Diseases of Women and 26 for Ophthalmic Cases}	406
„ „ „ Unassigned	18
	654

GENERAL STATEMENT OF THE PATIENTS UNDER TREATMENT DURING THE YEAR.

Patients remaining January 1st, 1876 :			
Medical	169	 498	
Surgical	329		
Admitted during the year :			 5623
Medical	2327	 5125	
Surgical	2798		
Discharged Cured and Relieved :			
Medical	1664		
Surgical	2575		
Discharged Unrelieved :			
Medical	254	 393	
Surgical	139		
Died :			 5623
Medical	404	 563	
Surgical	159		
Remaining January 1st, 1877 :			
Medical	174	 428	
Surgical	254		

Patients brought in Dead	22
Number of Post-mortem Examinations	417 = 71 per cent.

OCCUPATIONS OF MALE PATIENTS.

Accountant	..	..	1	Chairmakers	..	..	10	Farm bailiff	..	..	1
Account-book maker	..		1	Chemists	..	..	4	Farriers	..	..	2
Agents	..	..	2	Chimney sweep	..		1	Felt maker	..	..	1
Artist's model	..	..	1	Cigar makers	..	..	6	Feather dresser	..		1
Artists	..	..	2	Clergymen	..	..	2	Figure maker	..	..	1
Athlete	..	..	1	Clerks	..	..	73	Firemen	..	..	6
Artificial limb maker	..		1	Cloth worker	..	..	1	Firewood-cutter	..		1
Asphalte worker	..		1	Clickers	..	..	4	Fish curers	..	..	3
Bakers	..	..	14	Coachmakers	..	..	3	Fishermen	..	..	3
Banner maker	..	..	1	Coachmen	..	..	17	Fishmongers	..	..	11
Bargemen	..	..	6	Coachsmith	..	..	1	Fish salesman	..	..	1
Barmen	..	..	13	Coalheaver	..	..	1	Fitters	..	..	4
Basketmakers	..	..	2	Coal porter	..	..	1	Floor-cloth painter	..		1
Bell-hanger	..	..	1	Coffee-house keeper	..		1	Flower makers	..		2
Bellows maker	..	..	1	Coffee sorter	..	..	1	Flute maker	..	..	1
Beer bottler	..	..	1	Collar makers	..	..	3	Forge maker	..	..	1
Billposters	..	..	2	Collier	..	..	..	1	Frame makers	..	..	2
Billiard makers	..		4	Colour grinder	..		1	French polishers	..		14
Billiard-table maker	..		1	Colporteur	..	..	1	Fringe maker	..	..	1
Blacksmiths	..	..	8	Combmakers	..	..	2	Furniture dealers	..		4
Bone boiler	..	..	1	Commercial travellers	.		20	Furriers	..	..	7
Bookbinders	..	..	6	Compositors	..	..	12	Gaoler	..	..	..	1
Bootmakers	..	..	13	Confectioners	..	..	3	Gardeners	..	..	29
Brace maker	..	..	1	Cooks	..	..	..	2	Gasfitters	..	..	9
Brass finishers	..	..	10	Coopers	..	..	5	Gas-stoker	..	..	1
Brassfounders	..	..	18	Coppersmiths	..	..	4	Gas Engineer	..	..	1
Brass moulder	..	..	1	Cork-cutters	..	..	6	Gas-stove maker	..		1
Brass workers	..	..	2	Corset maker	..	..	1	Gatekeepers	..	..	2
Brass polisher	..	..	1	Costermongers	..	..	13	Gem dealer	..	..	1
Brewers	..	..	12	Cowman	..	..	1	Gentlemen	..	..	3
Brewer's stableman	..		1	Crossing sweeper	..		1	General dealers	..		15
Bricklayers	..	..	49	Customhouse officers	..		3	Gilders	..	..	..	3
Brickmakers	..	..	5	Curriers	..	..	2	Gingerbeer maker	..		1
Brushmakers	..	..	2	Dairyman	..	..	1	Glassblowers	..	..	9
Builders	..	..	7	Decorators	..	..	3	Glass stainer	..	..	1
Builder's storekeeper	..		1	Dentist	..	..	1	Glaziers	..	..	3
Butchers	..	..	17	Diamond-cutters	..		2	Glasscutters	..	..	3
Bullet roller	..	..	1	Distiller	..	..	1	Goldbeater	..	..	1
Butler	..	..	1	Dock labourers	..		4	Goldworker	..	..	1
Button maker	..	..	1	Drapers	..	..	9	Goldsmith	..	..	1
Cabinetmakers	..		26	Drainman	..	..	1	Greengrocers	..	..	4
Cabmen	..	..	29	Draymen	..	..	6	Gravedigger	..	..	1
Cage maker	..	..	1	Dressing-case maker	..		1	Grocers	..	..	8
Cane maker	..	..	1	Drovers	..	..	10	Grooms	..	..	11
Cap maker	..	..	1	Dustmen	..	..	4	Grainer	..	..	1
Carmen	..	..	99	Dyers	..	..	..	3	Hairdressers	..	..	7
Carpenters	..	..	57	Electrotyper	..	..	1	Hairdyer	..	..	1
Carpet salesman	..		1	Electro-plater	..	..	1	Harnessmakers	..		4
Carriers	..	..	3	Engine drivers	..		11	Hatters	..	..	6
Carters	..	..	8	Engineers	..	..	11	Hawkers	..	..	22
Carvers	..	..	2	Engravers	..	..	6	Horsehair drawer	..		1
Case makers	..	..	7	Errand boys	..	..	18	Horsehair carder	..		1
Cellarmen	..	..	5	Excavator	..	..	1	Horse-breaker	..	..	1
Cement maker	..		1	Factory hands	..	..	2	Horse-tender	..	..	1
Chaff-cutters	..	..	2	Farmers	..	..	2	Horsedealer	..	..	1

OCCUPATIONS OF MALE PATIENTS (*continued*).

Ice maker	1	Phrenologist	1	Smiths	17
Ice vendor	1	Physician	1	Soap makers	2
Indiarubber worker	1	Pianoforte makers	7	Soldiers	6
Instrument makers	3	Piano-key maker	1	Stationers	3
Iron-foundry labourers	4	Piano tuner	1	Stevedores	11
Iron founder	1	Pilot	1	Stick makers	2
Ironmongers	2	Pipe makers	2	Stokers	12
Iron moulders	2	Plane maker	1	Stonemasons	4
Japanners	3	Plasterer	1	Storekeeper	1
Jewellers	7	Platelayers	7	Straw-hat maker	1
Jewel-case maker	1	Plumbers	3	Students of medicine	4
Jewel-hole maker	1	Policemen	13	Sugar factor	1
Joiners	7	Polisher	1	Sweeps	8
Knife-machine makers	2	Porters	101	Tailors	35
Labourers	360	Post-office sorters	4	Tarpaulin maker	1
Lamplighters	2	Postmen	4	Tea mixer	1
Last maker	1	Potmen	12	Telegraph examiner	1
Law writer	1	Poulterer	1	Telegraph porter	1
Lead caster	1	Printers	56	Ticket cutter	1
Leather dressers	4	Printers' boys	17	Ticket writers	3
Leather finishers	2	Publicans	9	Tinfoil workers	6
Leather workers	2	Rag bleacher	1	Tinman	1
Lightermen	3	Railway pointsman	1	Tobacconist	1
Lithographer	5	Relieving officer	1	Top sawyer	1
Locksmith	1	Rivetters	9	Toy makers	2
Looking-glass makers	3	Roller maker	1	Tramway conductors	4
Machinists	3	Rope maker	1	Trimming maker	1
Machine ruler	1	Rope winder	1	Tripe dresser	1
Market gardener	1	Saddle-tree rivetter	1	Turners	10
Masons	22	Salesmen	3	Typefounders	3
Match-box maker	1	Sailors	32	Umbrella makers	4
Meat salesman	1	Sawdust seller	1	Umbrella-stick maker	1
Map mounter	1	Sawyers	11	Undertakers	2
Merchant	1	Scalemaker	1	Upholsterers	4
Metal caster	1	Scavengers	2	Van boys	9
Metal mounter	1	Schoolboys	103	Vellum binder	1
Millers	7	Schoolmasters	10	Verger	1
Milkmen	6	Servants	9	Waiters	13
Millwright	1	Sexton	1	Warehousemen	23
Miners	3	Shepherd	1	Warder	1
M.R.C.S.	2	Shipsmith	1	Watch makers	11
Musicians	5	Ship stewards	2	Watchmen	7
Naval Officer	1	Ship rigger	1	Watermen	4
Naturalist	1	Shipwrights	3	Weavers	3
Newsboy	1	Shirt cutter	1	Well sinkers	2
Omnibus conductor	1	Shoeblack	1	Whalebone manufacturer	1
Organ grinder	1	Shoemakers	33	Whalebone cutter	1
Ostlers	29	Shoe rivetter	1	Wheelwrights	4
Ostrich-feather washer	1	Shopmen	18	Whitelead carrier	1
Packers	17	Signal fitters	2	Window cleaner	1
Packing-case makers	3	Sign writer	1	Wine merchant	1
Pages	3	Signal maker	1	Wine porters	2
Painters	41	Silk weavers	2	Wireworkers	2
Pantomimists	3	Silversmiths	2	Wood carvers	3
Paper colourer	1	Silver-pencil maker	1	Wood chopper	1
Paperhangers	2	Skeleton articulator	1	Wood cutters	6
Pensioners	2	Skinner	1		
Perambulator maker	1	Slaughterman	1		

OCCUPATIONS OF FEMALE PATIENTS.

Artificial-flower makers	14	Furriers	9	Paper colourer	1
Asylum nurse	1	Fur cleaner	1	Paper dealer	1
Bag makers	2	Furniture dealers	2	Paper-bag maker	1
Ballet girl	1	General dealers	5	Paper rulers	2
Barmaids	16	Goldbeater-skin maker	1	Pauper	1
Board liner	1	Gold printer	1	Printers	2
Bonnet maker	1	Governesses	5	Publicans	4
Bonnet-shape maker	1	Grocer	1	Pupil teacher	1
Bookfolders	24	Greengrocers	5	Relief stampers	2
Bookbinders	2	Gun-cap maker	1	Saddle stretcher	1
Bookkeeper	1	Harlots	62	Sack maker	1
Bootmakers	8	Hawkers	24	Sail maker	1
Boot translator	1	Horse-cloth maker	1	Sash-line maker	1
Boot cutter	1	Horse-hair carder	1	Schoolmistresses	6
Bottle labeller	1	Hop picker	1	School girls	56
Bottlewasher	1	Hospital nurses	56	Scrubbers	4
Box liner	1	Housekeepers	22	Secretary	1
Box makers	13	Housewives	721	Seed sorter	1
Boxing-glove maker	1	India-rubber worker	1	Servants	315
Brace maker	1	Ironers	7	Shirt makers	4
Brush makers	3	Jewel-case maker	1	Shoebinder	1
Brush drawer	1	Lace makers	4	Shopwomen	10
Card maker	1	Laundresses	97	Silk weavers	4
Cartridge-case maker	1	Lead worker	1	Stationers	2
Chair caner	3	Lemon-peel cutter	1	Straw-hat makers	3
Chandeller	1	Linen marker	1	Straw plaiter	1
Charwomen	65	Lodging-house keeper	1	Straw worker	1
Cloth sorter	1	Machinists	38	Staymaker	1
Clothes dealers	2	Mantle makers	3	Tailoresses	33
Cigar-box paperer	1	Manglers	3	Tie makers	7
Cooks	46	Match maker	1	Tie cutter	1
Collar makers	4	Match-box maker	1	Tinfoil maker	1
Confectioner	1	Mattrass makers	11	Trimming makers	2
Dressmakers	20	Milliners	11	Umbrella makers	3
Draper	1	Milk women	2	Upholsterers	6
Envelope folders	5	Monthly nurses	6	Waistcoat makers	2
Envelope cementer	1	Musician	1	Waiteresses	3
Errand girl	1	Music teacher	1	Weaver	1
Factory hands	2	Needlewomen	63	Wire maker	1
Fancy box makers	4	Nurses	28	Warpers	2
Feather sorters	2	Object-glass maker	1	White-lead carriers	2
Fishmongers	2	Old clothes woman	1	Whip maker	1
Fishing-net maker	1	Ostrich-feather curlers	2	Wood chopper	1
Fruit seller	1	Packers	3	Wool twister	1
French polisher	2	Paper sorter	1		

MEDICAL REPORT.

TABLE I,

Showing the Total Number of Cases of each Disease under Treatment during the Year 1876, *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 1876.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
GENERAL DISEASES, A.										
Small pox(1)	1	..	..	..	1	..	..	..	..	..
Chicken pox	..	1	..	1	..	..	..	..	..	..
Measles	1	3	1	2	..	..	..	..	..	1
Sequelæ of Measles	..	1	..	..	..	..	..	..	..	..
Scarlet Fever (2)	21	25	18	22	..	..	..	2	3	1
Sequelæ of Scarlet Fever (3) ..	18	18	10	18	..	..	5	..	3	..
Typhus	4	2	3	2	..	..	1	..	..	..
Enteric Fever (4)	39	20	28	16	..	..	8	3	3	1
Simple Continued Fever (5) ..	3	2	2	2	..	..	1	..	..	..
Febricula	10	14	10	14	..	..	..	..	..	..
Ague—										
Tertian (6)	3	1	3	1	..	..	..	..	..	..
Quartan	1	..	1	..	..	..	..	..	..	..
Irregular	3	1	3	1	..	..	..	..	..	..
Sequelæ of Ague	1	..	1	..	..	..	..	..	..	..
Diphtheria (7)	3	8	2	6	..	..	1	2	..	..
Hooping-cough (8)	2	1	..	1	..	..	2	..	..	..
Erysipelas—										
Cellulo-cutaneous (of face) ..	9	15	7	14	..	..		..	..	1
Pyæmia (9)	2	1	1	..	..	..	1	1	..	..
Sequelæ of Pyæmia	1	..	..	..	1	..	..	..	..	..
Puerperal Fever (10)	..	5	..	3	..	..	..	2	..	..
GENERAL DISEASES, B.										
Rheumatism—										
Acute (11)	86	108	80	101	..	..	2	1	4	6
Subacute (12)	7	11	7	10	..	..	..	..	..	1
Gonorrhœal (13)	5	..	4	..	1	..	..	..	..	..
Synovial (14)	3	1	3	1	..	..	..	..	..	..
Muscular—										
Lumbago	1	1	..	1	..	..	..	..	1	..
Chronic (15)	15	20	14	18	1	2	..	..	..	..
Gout—										
Chronic (16)	8		8	5	..	..	..	..	..	..
Gouty Synovitis (17)	2	..	2	..	..	..	..	..	..	..

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, B (*continued*).										
Chronic Osteo-arthritis (18)	5	3	5	3	..	..	..	..	..	..
Cancer—										
of Abdomen (19)	4	1	..	..	1	..	**3**	**1**	..	..
of Stomach (20)	5	1	..	..	1	..	**4**	**1**	..	..
of Liver (21)	2	8	..	..	..	2	**1**	**6**	1	..
of Intestines	1	3	..	..	..	2	**1**	**1**	..	..
of Rectum (22)	1	1	..	..	..	..	**1**	**1**	..	..
of Kidney (23)	3	..	..	..	..	..	**3**	..	..	..
of Bladder (24)	1	..	..	..	..	..	**1**	..	..	..
of Pelvis	..	1	..	..	..	1	..	..	..	..
of Uterus	..	10	..	..	..	8	..	..	..	2
of Ovary (25)	..	1	..	..	..	..	..	**1**	..	..
Epitheliomia—										
of Cervix Uteri	..	11	..	10	..	..	..	..	..	1
Lupus—										
Chronic	..	1	..	..	..	1	..	..	..	..
Scrofula	..	1	..	..	..	..	..	..	..	1
Local Scrofulous Affections—										
Tubercular Meningitis (26)	5	4	..	..	..	1	**5**	**3**	..	..
Phthisis Pulmonalis (27)	60	80	28	30	11	18	**18**	**29**	3	3
Acute Miliary Tuberculosis (28)	3	3	..	..	..	..	**3**	**3**	..	..
Tabes Mesenterica	2	..	1	..	1	..	..	..	..	..
Tubercular Peritonitis	3	1	..	1	2	..	**1**	..	..	..
Rickets	2	5	1	3	..	2	**1**	..	..	..
Diabetes (29)	11	5	6	1	1	..	**4**	**2**	..	2
Purpura—										
Simple	2	3	2	1	..	..	..	**1**	..	1
Hæmorrhagic (30)	3	1	1	1	..	..	**2**	..	..	..
Scorbutus	1	1	1	1	..	..	..	..	..	..
*Anæmia (31)	1	17	..	17	..	..	**1**	..	..	..
Chlorosis	..	6	..	6	..	..	..	..	..	..
*General Dropsy	1	1	1	1	..	..	..	..	..	..
*Local Anasarca	..	1	..	1	..	..	..	..	..	..
LOCAL DISEASES.										
DISEASES OF THE NERVOUS SYSTEM.										
DISEASES OF THE BRAIN AND ITS MEMBRANES.										
Encephalitis (32)	..	1	..	..	..	..	..	**1**	..	..
Meningitis (Simplex) (33)	..	3	..	1	..	1	..	**1**	..	..
Yellow Softening	2	1	..	..	..	..	**2**	**1**	..	..
Abscess of the Brain	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 NERVOUS SYSTEM (*continued*).										
Apoplexy—										
Sanguineous [34]	11	3	..	..	..	..	**11**	**3**	..	..
Chronic Hydrocephalus	..	1	..	..	..	1	..	..	..	..
Tumour [35]	2	..	..	..	..	..	**2**	..	..	..
Cerebral Affection	1	3	..	3	..	..	**1**	..	..	..
Diseases of the Spinal Cord and its Membranes.										
Inflammation—										
Myelitis	2	1	2	1	..	..	..	..	..	..
Diseases of the Nerves.										
*Paralysis—										
Hemiplegia [36]	35	14	18	6	15	5	**1**	**2**	1	1
Cross Paralysis [37]	1	..	..	..	1	..	..	..	..	..
Paraplegia [38]	7	5	3	4	2	1	**1**	..	1	..
Locomotor Ataxy	10	1	4	..	5	..	..	..	1	1
Infantile paralysis [39]	1	3	1	3	..	..	..	..	..	..
Local Paralysis—										
Facial Paralysis	1	2	1	1	..	1	..	..	..	..
Other Paralyses [40]	6	5	4	4	2	1	..	..	..	..
Functional Diseases of the Nervous System.										
Tetanus [41]	2	1	1	..	..	..	**1**	**1**	..	..
Hydrophobia [42]	1	..	..	..	..	..	**1**	..	..	..
*Infantile Convulsions	..	2	..	2	..	..	..	..	..	..
Epilepsy [43]	23	9	20	8	..	1	**2**	..	1	..
Epileptic Vertigo	6	..	4	..	2	..	..	..	..	..
Epileptic Hemiplegia	..	1	..	1	..	..	..	..	..	..
Laryngismus stridulus	1	..	1	..	..	..	..	..	..	..
Shaking Palsy	2	..	1	..	1	..	..	..	..	..
Spasm of Muscles	1	..	1	..	..	..	..	..	..	..
Chorea [44]	10	41	7	33	..	2	**1**	**1**	2	5
Hysteria [45]	1	27	1	23	..	4	..	..	..	..
Hysterical Paralysis [16]	..	5	..	3	..	2	..	..	..	..
Neuralgia—										
Diffused	..	4	..	4	..	..	..	..	..	..
Cephalalgia	4	2	4	2	..	..	..	..	..	..
Sciatica	2	2	2	2	..	..	..	..	..	..
Pleurodynia	5	4	5	4	..	..	..	..	..	..
*Hyperæsthesia	..	3	..	3	..	..	..	..	..	..
Hypochondriasis	3	..	1	..	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 NERVOUS SYSTEM (*continued*).										
Obscure Nerve disorder	3	..	1	..	1	..	..	..	1	..
DISORDERS OF THE INTELLECT.										
Mania	2	3	..	..	2	3	..	..	..	..
Melancholia	..	2	..	..	..	2	..	..	..	..
Dementia	1	2	..	..	1	2	..	..	..	..
Idiotcy	..	1	..	..	..	1	..	..	..	..
DISEASES OF THE CIRCULATORY SYSTEM.										
DISEASES OF THE HEART AND ITS MEMBRANES.										
Diseases of the Pericardium.										
Pericarditis ([47])	6	2	6	2	..	..	..	..	..	..
Diseases of the Endocardium.										
Endocarditis ([48])	1	2	..	..	..	..	**1**	**2**	..	..
Valve Disease—([49])										
1. Aortic	20	6	7	4	5	..	**4**	**1**	4	1
2. Mitral	34	52	16	25	2	4	**13**	**22**	3	1
3. Pulmonary	..	1	..	1	..	..	..	..	..	..
4. Tricuspid	..	..	..	..	..	..	..	..	..	..
5. Complicated	10	6	6	1	..	..	**4**	**5**	..	..
6. Congenital	2	..	..	..	2	..	..	..	..	..
Diseases of the Muscular Structure of the Heart.										
Dilatation	2	2	..	1	..	1	**2**	..	..	..
*Angina Pectoris	1	..	1	..	..	..	..	..	..	..
DISEASES OF THE BLOOD VESSELS.										
Diseases of the Arteries.										
Atheroma	1	..	..	..	1	..	..	..	..	..
Fibroid Changes ([50])	1	..	..	..	1	..	..	..	..	..
Aneurism—										
of Aorta ([51])	16	7	6	1	3	2	**5**	**4**	2	..
of Innominate	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.
DISEASES OF THE BLOOD VESSELS (*continued*).										
Aneurism (*continued*).										
of Subclavian	1	..	..	..	1	..	..	..	..	..
of Cœliac axis (52)	1	..	..	..	..	..	**1**	..	..	..
of Abdominal Arteries (53) ..	1	..	..	..	..	..	**1**	..	..	..
Epigastric Pulsation	1	2	1	1	..	1	..	..	..	..
Diseases of the Veins.										
Phlebitis	..	1	..	1	..	..	..	..	..	..
Phlegmasia Dolens	..	1	..	1	..	..	..	..	..	..
Obstruction (54)	1	..	1	..	..	..	..	..	..	..
DISEASES OF THE ABSORBENT SYSTEM.										
Hypertrophy of Glands—Chronic Enlargement of Glands (55)	1	7	..	1	..	6	**1**	..	..	..
DISEASES OF DUCTLESS GLANDS.										
DISEASES OF THE THYROID GLAND.										
Exophthalmic Bronchocele (56) ..	2	3	..	..	2	3	..	..	..	..
Enlargement	..	1	..	..	..	..	..	**1**	..	..
DISEASES OF THE SUPRA-RENAL CAPSULES.										
Addison's Disease (57)	1	..	..	..	..	..	**1**	..	..	..
DISEASES OF THE RESPIRATORY SYSTEM.										
DISEASES OF THE RESPIRATORY SYSTEM NOT SIMPLY LOCAL.										
Croup (58)	5	4	1	1	..	..	**4**	**3**	..	..
DISEASES OF THE LARYNX.										
Laryngeal Catarrh	2	3	2	3	..	..	..	..	..	..
Laryngitis—										
Acute	..	1	..	1	..	..	..	..	..	..
Chronic	3	1	2	1	..	..	..	..	1	..
Œdema of the Glottis	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 THE RESPIRATORY SYSTEM (*continued*).										
DISEASES OF THE TRACHEA AND BRONCHI.										
Bronchial Catarrh (59)	9	10	7	10	..	..	**1**	..	1	..
Bronchitis—										
Acute (60)	15	25	12	18	1	1	**2**	**5**	..	1
Chronic (61)	38	52	20	32	1	4	**16**	**9**	1	7
Asthma	..	1	..	1	..	..	..	..	..	..
DISEASES OF THE LUNG.										
Pneumonia (62)	55	26	40	22	2	..	**12**	**4**	1	..
Lobular (63)	4	4	1	2	..	..	**3**	**1**	..	1
Sequelæ of Pneumonia	..	1	..	1	..	..	..	..	..	..
Pleuro-Pneumonia (64)	23	17	20	15	..	1	**3**	..	..	1
Abscess (65)	1	..	1	..	..	..	..	..	..	..
Gangrene	2	2	1	1	..	..	..	**1**	1	..
Congestion	1	..	1	..	..	..	..	..	..	..
*Hæmoptysis	13	2	11	2	1	..	..	..	1	..
Cirrhosis (66)	9	1	6	1	1	..	**2**	..	..	..
Emphysema (67)	10	1	5	..	1	1	**4**	..	..	..
DISEASES OF THE PLEURA.										
Pleurisy (68)	37	24	31	23	1	..	**3**	**1**	2	..
Chronic Pleurisy (69)	2	2	2	1	..	..	..	**1**	..	..
Empyema (70)	4	1	3	..	..	..	..	**1**	1	..
Pneumothorax (71)	2	..	1	..	..	..	**1**	..	..	..
DISEASES OF THE MEDIASTINUM.										
Tumour (72)	1	5	..	..	..	2	**1**	**3**	..	..
DISEASES OF THE DIGESTIVE SYSTEM.										
DISEASES OF THE STOMACH.										
Gastritis (73)	5	1	3	1	1	..	**1**	..	..	..
Chronic Ulcer (74)	4	10	3	8	..	..	**1**	..	..	2
*Hæmatemesis	4	2	4	2	..	..	..	..	..	..
*Dilatation (75)	3	..	3	..	..	..	..	..	..	..
Dyspepsia	4	4	3	4	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 1876.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE DIGESTIVE SYSTEM (*continued*).										
Gastrodynia	2	3	2	3	..	..	..	..	..	..
Pyrosis	1	..	1	..	..	..	..	..	..	..
*Vomiting	2	6	2	5	..	1	..	..	..	..
DISEASES OF THE INTESTINES.										
Enteritis	1	2	..	1	..	..	**1**	**1**	..	..
Typhlitis	2	..	2	..	..	..	..	..	..	..
Dysentery (76)	5	1	4	1	..	..	**1**	..	..	..
Ulceration	..	1	..	..	..	..	..	..	..	1
Abscess in the Subperitoneal Tissue	1	1	1	..	..	..	..	**1**	..	..
*Tympanites	..	2	..	2	..	..	..	..	..	..
*Obstruction (77)	..	2	..	..	..	..	..	**2**	..	..
Diarrhœa	3	6	3	5	..	1	..	..	..	..
Colic (78)	3	2	3	2	..	..	..	..	..	..
Constipation	6	9	6	9	..	..	..	..	..	..
Diarrhœa and Vomiting (79)	4	2	4	2	..	..	..	..	..	..
DISEASES OF THE LIVER.										
Cirrhosis (80)	17	4	3	1	5	..	**8**	**3**	1	..
Lardaceous Liver (81)	..	1	..	..	..	..	..	**1**	..	..
Parasitic Disease—										
Echinococcus hominis	5	2	3	1	1	1	..	..	1	..
Jaundice	10	10	8	8	1	1	..	..	1	1
Enlargement (82)	6	8	4	3	2	1	..	**2**	..	2
DISEASES OF THE HEPATIC DUCTS AND GALL BLADDER.										
Gall Stones (83)	2	3	1	3	..	..	**1**	..	..	..
DISEASES OF THE SPLEEN.										
Hypertrophy	2	..	..	..	2	..	..	..	..	..
Leucocythæmia (84)	3	..	..	..	..	..	**3**	..	..	..
DISEASES OF THE PERITONEUM.										
Peritonitis (85)	3	5	3	2	..	..	..	**3**	..	..
*Ascites	2	7	1	4	..	1	**1**	**1**	..	1
Tumours	6	8	..	..	5	8	..	..	1	..
Congenital Tumour	..	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.
DISEASES OF THE URINARY SYSTEM.										
DISEASES OF THE KIDNEY.										
Bright's Disease—										
1. Acute (86)	18	13	13	11	..	..	**3**	**1**	2	1
2. Chronic (87)	42	24	15	11	1	2	**21**	**10**	5	1
Abscess	2	1	1	..	..	1	..	..	1	..
Pyelitis (88)	1	..	..	..	..	..	**1**	..	..	..
Calculus (89)	1	1	1	..	..	..	..	**1**	..	..
*Hæmaturia renalis (90)	5	3	2	3	2	..	..	..	1	..
*Suppression of Urine	1	1	1	1	..	..	..	..	..	..
*Diuresis	1	..	1	..	..	..	..	..	..	..
DISEASES OF THE BLADDER.										
Cystitis	1	..	1	..	..	..	..	..	..	..
DISEASES OF THE GENERATIVE SYSTEM.										
DISEASES OF THE FEMALE ORGANS OF GENERATION IN THE UNIMPREGNATED STATE.										
DISEASES OF THE OVARY.										
Inflammation	..	3	..	3	..	..	..	..	..	..
Complex Cystic Tumour (91) ..	..	14	..	5	..	5	..	**4**	..	..
DISEASES OF THE BROAD LIGAMENT.										
Inflammation—										
Pelvic Peritonitis..	..	..	..	..	..	..	..	..	..	..
Pelvic Cellulitis (92)	..	16	..	14	..	1	..	**1**	..	..
Abscess (93)	..	1	..	1	..	..	..	..	..	..
Periuterine or Pelvic Hæmatocele (94)	..	22	..	20	..	..	..	..	..	2
DISEASES OF THE UTERUS, INCLUDING THE CERVIX.										
Catarrh (Leucorrhœa)	..	4	..	4	..	..	..	..	..	..
Inflammation (Chronic)	..	13	..	10	..	2	..	..	..	1
Granular Inflammation	..	1	..	1	..	..	..	..	..	..
Congestion	..	2	..	2	..	..	..	..	..	..
Stricture of the Orifice	..	2	..	2	..	..	..	..	..	..
Stricture of the Canal	..	2	..	2	..	..	..	..	..	..
Hypertrophy	..	1	..	..	..	1	..	..	..	..
Elongation of the Cervix (95) ..	..	2	..	2	..	..	..	..	..	..

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.
DISEASES OF THE FEMALE ORGANS OF GENERATION, &c. (*continued*).										
Non-Malignant Tumour	..	1	..	..	..	1	..	..	..	..
A. Fibrous Tumour (96)	..	21	..	6	..	14	..	..	..	1
B. Polypus (97)	..	10	..	10	..	..	..	..	..	..
DISPLACEMENTS AND DISTORTIONS.										
A. Anteversion	..	..	..	..	..	..	..	..	..	..
B. Retroversion (98)	..	15	..	13	..	1	..	..	..	1
C. Anteflexion (99)	..	8	..	7	..	1	..	..	..	..
D. Retroflexion	..	6	..	4	..	1	..	..	..	1
E. Inversion (100)	..	2	..	1	..	..	..	..	..	1
F. Prolapsus— Procidentia	..	3	..	3	..	..	..	..	..	..
DISEASES OF THE VAGINA.										
Inflammation (101)	..	2	..	2	..	..	..	..	..	..
Abscess	..	1	..	1	..	..	..	..	..	..
Hernia— Cystocele	..	1	..	1	..	..	..	..	..	..
DISEASES OF THE VULVA.										
Hypertrophy	..	2	..	2	..	..	..	..	..	..
Vascular Tumour of the Meatus Urinarius	..	2	..	2	..	..	..	..	..	..
Dilated Urethra (102)	..	1	..	..	..	1	..	..	..	..
FUNCTIONAL DISEASES OF THE FEMALE ORGANS OF GENERATION.										
Amenorrhœa	..	1	..	..	..	1	..	..	..	..
Dysmenorrhœa	..	3	..	3	..	..	..	..	..	..
Menorrhagia (103)	..	2	..	1	..	..	..	1	..	..
Pelvic Tumours (104)	..	2	..	1	..	1	..	..	..	..
AFFECTIONS CONNECTED WITH PREGNANCY.										
DISORDERS OF THE GENERATIVE SYSTEM.										
Pregnancy	..	1	..	..	..	1	..	..	..	..
Hæmorrhage	..	2	..	2	..	..	..	..	..	..
Displacements of the Uterus— Retroversion (105)	..	4	..	4	..	..	..	..	..	..
Abortion	..	6	..	6	..	..	..	..	..	..
Extrauterine Gestation (106) ..	..	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.
AFFECTIONS CONNECTED WITH PREGNANCY (*continued*).										
AFFECTIONS CONNECTED WITH PARTURITION.										
Hæmorrhage (after Abortion) ..	..	3	..	3	..	..	..	..	..	..
Retention of Part of the Ovum ..	..	5	..	5	..	..	..	..	..	..
AFFECTIONS CONSEQUENT ON PARTURITION.										
Subinvolution ([107])	..	27	..	24	..	2	..	..	..	1
DISEASES OF THE MUSCLES.										
Progressive Muscular Atrophy ..	4	..	..	..	3	..	..	..	1	..
DISEASES OF THE CELLULAR TISSUE.										
Obesity	1	..	..	..	1	..	..	..	..	..
DISEASES OF THE CUTANEOUS SYSTEM.										
Erythema—										
E. Læve	1	2	1	2	..	..	..	..	..	..
E. Tuberculatum	..	1	..	1	..	..	..	..	..	..
E. Nodosum ([108])	..	5	..	4	..	..	..	1	..	..
Urticaria	1	2	1	2	..	..	..	..	..	..
Psoriasis	2	1	2	1	..	..	..	..	..	..
Herpes ([109])	..	1	..	1	..	..	..	..	..	..
H. Zoster	1	..	1	..	..	..	..	..	..	..
Pemphigus	1	..	1	..	..	..	..	..	..	..
Eczema—										
E. Simplex ([110])	9	4	8	4	..	..	1	..	..	..
E. Rubrum	..	1	..	1	..	..	..	..	..	..
E. Impetiginodes	..	1	..	1	..	..	..	..	..	..
E. Exfoliativum	2	..	1	..	..	..	..	..	1	..
E. Chronicum	..	1	..	1	..	..	..	..	..	..
Xeroderma	..	3	..	1	..	1	..	..	..	1
Molluscum	..	1	..	1	..	..	..	..	..	..
PARASITIC DISEASE OF THE SKIN.										
Tinea Favosa	1	..	..	..	1	..	..	..	..	..
Scabies	..	2	..	1	..	..	..	..	..	1
Irritation caused by Pediculi ..	..	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.
CONDITIONS NOT NECESSARILY ASSOCIATED WITH GENERAL OR LOCAL DISEASES.										
Premature Birth [111]	..	1	..	..	..	..	..	**1**	..	..
Debility [112]	20	53	16	52	..	..	**3**	..	1	1
Pain	..	2	..	1	..	1	..	..	..	..
Phantom Tumour	..	1	..	1	..	..	..	..	..	..
Fright	..	1	..	1	..	..	..	..	..	..
Malingering	2	..	..	..	2	..	..	..	..	..
Unknown	22	33	..	..	..	1	**1**	..	21	32
POISONS.										
METALS AND THEIR SALTS.										
Mercury—										
Corrosive Sublimate [113] ..	1	..	1	..	..	..	..	..	..	..
Lead— [114]										
Lead Colic	7	4	7	4	..	..	..	..	..	..
Lead Palsy	3	1	3	..	..	..	..	..	..	1
Lead Vertigo	1	..	1	..	..	..	..	..	..	..
CAUSTIC ALKALIES.										
Potash [115]	1	1	1	1	..	..	..	..	..	..
Ammonia [116]	4	..	4	..	..	..	..	..	..	..
ACIDS.										
Sulphuric Acid [117]	1	3	1	3	..	..	..	..	..	..
Nitric Acid [118]	..	4	..	4	..	..	..	..	..	..
Oxalic Acid	..	1	..	1	..	..	..	..	..	..
Acetic Acid [119]	..	1	..	1	..	..	..	..	..	..
Carbolic Acid [120]	1	1	..	1	..	..	..	..	1	..
VEGETABLE POISONS.										
Alcohol	5	1	5	..	..	..	..	**1**	..	..
Intoxication [121]	2	1	2	1	..	..	..	..	..	..
Delirium tremens [122]	11	1	11	1	..	..	..	..	..	..
Paraffin	..	1	..	1	..	..	..	..	..	..
Soap Liniment	..	1	..	1	..	..	..	..	..	..

ABSTRACT OF TABLE I.

DISEASES.	Total number of cases under treatment during the year.		Number of cases discharged cured and relieved.		Discharged unrelieved.		Died.		Remaining in the hospital at the end of the year 1876.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
General Diseases, A	122	118	90	104	2	..	**21**	**10**	9	4
Do. Do. B	243	316	161	209	19	37	**54**	**53**	9	17
Local Diseases—										
Diseases of the Nervous System	147	152	82	108	34	27	**24**	**10**	7	7
,, Circulatory System	100	82	44	38	16	8	**31**	**34**	9	2
,, Absorbent System	1	7	..	1	..	6	**1**	..	..	..
,, Ductless Glands	3	4	..	..	2	3	**1**	**1**	..	..
,, Respiratory System	237	183	167	135	8	9	**53**	**29**	9	10
,, Digestive System..	106	103	67	67	18	15	**17**	**14**	4	7
,, Urinary System	72	43	35	26	3	3	**25**	**12**	9	2
,, Female Organs of Generation ..	..	215	..	167	..	34	..	**6**	..	8
,, Muscular System	4	..	..	..	3	..	..	..	1	..
,, Cellular Tissue	1	..	..	..	1	..	..	..	..	..
,, Cutaneous System	18	26	15	22	1	1	**1**	**1**	1	2
Conditions not necessarily associated with General or Local Diseases	44	91	16	55	2	2	**4**	**1**	22	33
Poisons	37	21	36	19	..	..	..	**1**	1	1
	1135	1361	713	951	109	145	**232**	**172**	81	93
	2496		1664		254		**404**		174	
			1918							
			2496							

APPENDIX TO TABLE I.

NOTE.—*The references to "Reports" allude to St. Bartholomew's Hospital Reports*, 1876.

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1. *Small-pox.*—This case was removed to the Small-pox Hospital.

2. *Scarlet Fever.*—Among the complications of those who recovered were Morbus Cordis (double mitral murmur) in a boy aged 13, Pneumonia in a girl aged 5, Erysipelas in a girl aged 19, and acute inflammation of the knee joint, and cellular tissue of the neck in a girl aged 10. One fatal case was complicated by Pneumonia.

3. *Sequelæ of Scarlet Fever.*—Among the complications of those who recovered were the following:—Hooping cough in a boy aged 2; Pleurisy of right side in a boy aged 4; Otitis in a girl aged 5; Abscess in neck in a man aged 26. One fatal case was complicated with Pneumonia of the right apex, double Pleurisy, Pericarditis, Caries of the spine, and two Psoas abscesses. The patient was a boy aged 10.

4. *Enteric Fever.*—The abodes of the cases showed a certain prevalence in various localities. Six cases came from Whitecross-street, St. Luke's, or its immediate neighbourhood; three from Bartholomew Close—two from the same house; four from Hoxton—two from the same house; the rest were sporadic; one was a nurse in the Hospital, who died. One came from Christ's Hospital. Of the fatal cases, two had Nephritis, one Peritonitis, and one Laryngeal Ulceration. Of those who recovered, one had a relapse, three suffered from Pneumonia, two had Morbus Cordis (one with Rheumatic fever), one had right Pleurisy, one had Thrombosis of the left femoral vein.

5. *Simple Continued Fever.*—Under the head of "Fever" is included one fatal case in which post-mortem examination showed hœmorrhages into the small intestine; the tonsils swollen; the pharynx covered with a thin membrane easily peeled off, not extending into the larynx.

6. *Ague* (*tertian*).—One patient, who lived in Commercial-road, E., stated that his neighbours had been suffering from Ague. He also himself was in the habit of going "harvesting" into Kent, Shoeburyness, and other parts. He was admitted in May.

7. *Diphtheria.*—One patient, who recovered, suffered from paralysis of the left external rectus oculi muscle. One case was contracted in the Hospital, and recovered. In one fatal case, in a girl aged 7, on whom tracheotomy was performed, the membrane extended down the bronchi, and a large cavity of the size of a bantam's egg, close to the surface, was found in the base of the right lung. In another fatal case, a girl aged 5, tracheotomy had been performed.

8. *Hooping Cough.*—One fatal case, in a boy aged 2, was complicated with Measles.

9. *Pyæmia.*—One patient, who was discharged, suffered from obstruction of the vena cava inferior, dulness extending from the sixth right space to the iliac crest, a "caput Medusæ" was formed in the abdominal veins. In one fatal case a man, aged 20, the following were the post mortem appearances: There was an abscess on the thigh, not leading to bone, a slough the size of a florin over the right malar bone, many abscesses over the frontal bone, beneath which bone were hœmorrhages and exudation; the tonsils were ulcerating, a pulmonary infarct in the left lung, and two pyæmic abscesses in the right lung. In another fatal case, a girl aged 11, there

was an abscess over a metacarpo-phalangeal joint of the right hand and over the sterno-clavicular articulation, communicating with the joint; Pleurisy on the left side, small abscesses in both lungs; the spleen very large, soft, and adherent; a small commencing abscess was found in the ileum.

10. *Puerperal Fever.*—One patient, who suffered from septicæmia after miscarriage, had Pleuro-pneumonia on the left side, as well as Albuminuria, but recovered.

11. *Acute Rheumatism.—Discharged.*—The number of first attacks was 74, ages from 4 to 72; of these the heart was affected in 31, ages from 4 to 72; and of these, one, aged 13, had Chorea, and one Pneumonia; one of those without cardiac mischief had Albuminuria, and one Pleurisy. The number of second attacks was 48, ages varying from 12 to 55; of these the heart was affected in 19, ages varying from 12 to 55. The number of third attacks was 14, ages varying from 10 to 50; of these the heart was affected in 12, ages varying from 10 to 50. The number of fourth attacks was 4, ages from 25 to 45; in two, aged 30 and 34, the heart was affected. The number of fifth attacks was 6, ages from 15 to 44; in 5 of which the heart was affected. Three patients—one, aged 31, sixth attack; one, aged 48, seventh attack; one, aged 23, eleventh attack—escaped without cardiac mischief. One patient had Pleurisy, one double Pleuro-pneumonia, one Albuminuria.

The following case, which recovered, was treated with splints:—Female, aged 21. First attack. Heart only slight systolic blow, which was permanent, but its action feeble and irregular; great pain in joints. On eighth day, after swallowing a dose of morph. hydrochl. gr. $\frac{1}{3}$, she ceased to breathe; aroused by brandy, &c.; limbs put in splints; same day slept naturally; temperature fell from 103·2 to 101·8. Twelfth day, temperature 106·4, but eventually sank. Splints removed on twenty-first day. On twenty-sixth day, temperature and pulse normal. No heart murmur on discharge. Reports, p. 175.

Died.—All the three fatal cases were complicated with heart affections. All three were first cases. The male cases were aged 32 and 20 respectively; the female case 21. The male case, aged 32, had disease of both mitral and aortic valves, the liver and spleen were very soft; the small intestines were lined in part with diphtheritic membrane. The other male case had Pleurisy, Pneumonia, and Pericarditis. The female case had Pleurisy and Pericarditis.

12. *Subacute Rheumatism.*—In two female cases the heart was affected.

13. *Gonorrhœal Rheumatism.*—The joints affected were the following:—In one case the right knee, in another the left ankle, in another both knees, the right ankle, and the left shoulder.

14. *Synovial Rheumatism.*—One case had also Iritis, and one suffered from an attack of inter-current Typhoid.

15. *Chronic Rheumatism.*—In one case the sacro-iliac joint was affected, one had Periostitis, one a mitral murmur, and one Pneumonia on the left side.

16. *Chronic Gout.*—Two cases suffered from Sciatica, two had Albuminuria, one with a mitral murmur, and one had catarrh of the bladder.

17. *Gouty Synovitis.*—The joints affected were the right ankle and left knee.

18. *Chronic Osteo-arthritis.*—In one case the joint affected was the left hip-joint; another came on after Pleurisy, the right Lung was solid, and the Heart dislocated to the right of the sternum.

19. *Cancer of the Abdomen.*—The fatal cases were as follows:—In one the Omentum, Liver, and both ovaries were diseased; in another the disease had involved the Thorax; in another the Pancreas, Liver, Intestines, and Posterior Mediastinum were affected.

One case, in a boy aged 16, deserves somewhat longer notice. The duration of the disease from the time the tumour was noticed was about eight months;

both testicles had been retained in the Abdomen, and previously there had been left inguinal hernia. Post mortem the disease was found to spring from the left pubic bone, preventing descent of the left testicle and half filling the belly descending into the left thigh, embedding the Bladder and obstructing the Ureters. The left Ureter was double till near its lower end. There were growths in the Liver and right Lung.

20. *Cancer of Stomach.*—In one fatal case the Pancreas and Liver were also involved, in another the Peritoneum, in another the Liver, and in another there was Thrombosis of the vena cava inferior.

21. *Cancer of Liver.*—In one fatal case there was partial Paraplegia; in the others the organs besides the Liver which were affected were as follows:—The Lungs, Omentum, Intestines and Mesentery; Pancreas (2). In one of those in which the Pancreas was also affected, the Uterus contained many fibroid growths, one of them, of the size and hardness of a billiard ball, had to be cut with the saw.

22. *Cancer of Rectum and Liver.*—Of the female fatal case, the following are the notes:—Cachectic; temp. 99·4; no pain, but continual watery diarrhœa without tenesmus; inguinal glands enlarged; Jaundice slight. A finger's length from the anus, an irregular nodular painful growth, not much occluding the rectum, not implicating the uterine organs. P. M. partial solidification of both lungs with formation of cavities. In the Liver cancerous nodules were found, the right lobe filled by malignant growths, a few in the left, the hepatic ducts partly obstructed. The right lobe was somewhat jaundiced. Reports, p. 251.

23. *Cancer of Kidney.*—Of the fatal cases, in two the right kidney was affected; the subjects were boys, aged 4 and 5 respectively; in one of them there was Hydronephrosis, and the cancer involved the Peritoneum; in the other the Liver, both Lungs, and the right Pleura, were involved. The third, a man, aged 56, had also phthisis.

24. *Cancer of the Bladder.*—The fatal case was in a man, aged 69; the cancer was villous; the prostrate was enlarged, and contained calculous matter.

25. *Cancer of the Ovary.*—The fatal case was in a woman, aged 38. The left Ovary was affected. There was great Ascites, and the first diagnosis was that of subperitoneal Fibroid of the Uterus. She was tapped; 7 pints of bloody fluid being drawn off.

26. *Tubercular Meningitis.*—One fatal case followed caries of the cervical vertebræ.

27. *Phthisis Pulmonalis.*—Of the cases discharged the following are the principal facts as to the side affected: Right side, 15, two with albuminuria; left side, 37, one with albuminuria; double, 21.

Of the fatal cases, the right side was affected in 6, the left in 8, both sides in 27. There were 2 cases of Pneumothorax in the last-mentioned category; 1 of Pericarditis; 1 of left Pleurisy; 1 of Paraplegia, with enlargement of the liver.

One fatal case of Phthisis was complicated by cancer of the right kidney, as follows: The patient was a man, aged 36; the course of the phthisis from which he died presented nothing remarkable. Fourteen years ago he was struck by a sword hilt, in falling from a horse, in his right loin, and from that time had occasional attacks of hæmaturia, and noticed a tumour gradually growing. On admission, a tumour occupied the umbilical region, extending towards the right flank; it was irregularly quadrate, elastic, lobed, and in general outline resembled the Liver; it was dull on percussion. A tumour was felt in the right lumbar region; the whole tumour measured 15 in. across, and 7½ in. vertically. The lobes were elastic, but no fluctuation could be felt between the lobes; the tumour was freely movable, not adherent to abdominal walls, descended with respiration, and had no intestine in front of it; it was never painful, and was only tender in one spot. Urine neutral; sp. grav. 1024; no albumen, except when it contained blood occa-

sionally. The man died of his phthisis. The tumour was found filling the space between the Liver and Cœcum, and extended far back into the loins. The right Ureter was behind the tumour, the transverse Colon below it. In the pelvis of the kidney was a small mulberry calculus adherent. The tumour had apparently grown out of the medulla of the Kidney, and had pushed before it the upper part of the cortex, which was found below the liver. It consisted of schirrhous and encephaloid cancer, with one patch of cheesy and calcareous matter. *Lancet*, Feb. 10th, 1877, p. 194.

28. *Acute Miliary Tuberculosis.*—In the fatal cases the organs were affected as follows :—In one case both Lungs, Pericardium (with Endocarditis), Liver, Spleen, Intestines, and Kidneys ; in another, both Lungs, Kidneys, Intestines, Mesenteric Glands, with Endocarditis ; in another, both Lungs, Kidneys, Spleen, Intestines, Peritoneum, right Pleura ; in another, both Lungs, Pericardium, Liver, Kidneys, Intestines, Peritoneum, Meninges ; in another, both Lungs, Meninges, Peritoneum, Intestines, Liver, Spleen, both Kidneys ; and in the last, both Lungs, Liver, Spleen, both Kidneys, and both Pleuræ.

Both Lungs and Kidneys, in all 6 ; Intestines, in 5 ; Liver and Spleen, in 4 ; Peritoneum, in 3 ; Pericardium, Pleuræ, Meninges, and Endocarditis, in 2.

29. *Diabetes.*—Of the cases which were discharged one was relieved by milk diet, one had goître on the left side. Of the fatal cases one had double Phthisis ; one had atrophy of the brain and double Pleurisy ; one had Pericarditis, double Phthisis, tubercle in the Liver, Kidneys, Mesentery, and Intestines.

30. *Purpura Hæmorrhagica.*—One fatal case had laryngeal obstruction, for which tracheotomy was performed two days before her death ; another, a male, aged 23, had ecchymoses under both Pleuræ and Pericardium ; the Heart showed a patent Foramen ovale, Aortic incompetence with vegetations, infarcts in the Lungs, Liver, and Kidneys. Of another male case the following are the notes :—

Age 17 ; never strong ; "sick headache" last two or three weeks, with occasional shivering. On April 9th, attacked with severe pain in back ; then and ever since brought up blood ; the same day passed blood in urine ; next day and ever since passed blood by stool ; there was no epistaxis.

No family history of hæmorrhagic diathesis.

On admission, hæmorrhagic spots on face and trunk, chiefly on legs ; both conjunctivæ injected, pupils widely dilated. Black sordes on lips and teeth, and hæmorrhages into tongue and oral mucous membrane. Urine thick and claret-coloured. No ecchymoses from pressure. Temp. 99°.

Sickness and hæmorrhage from bowels and bladder began to cease on April 19th, when he began to pass large quantities of urine. On April 21st, right parotid region began to swell, and become red and tender. April 26th, slight epistaxis began ; then retention of urine ; symptoms of peritonitis ; "coffee ground" vomit. The temperature rose during the last week, and on April 29th was 106·3°, on which day he died.

P.M.—The brain quite healthy, no hæmorrhages, no excess of fluid in the ventricles. The parotid swelling, due to an abscess, extending inwards and downwards ; no diseased bone ; some cervical glands cheesy. Small ecchymoses in left Pleura and in Pericardium. The clots of blood in the heart pale. A few ecchymoses in Endocardium, valves healthy ; Aorta atheromatous. Peritoneum smeared in parts with blood, no clots. No Peritonitis. One large continuous extravasation, covering pubic surface of Recti, Bladder, Rectum, and Pelvis. Old adhesions joining Liver to Diaphragm and Stomach. No ulceration of Stomach or small Intestines. Mucous membrane of Rectum highly pigmented in patches size of sixpence, two of these patches having lost mucous membrane. Liver pale, friable, acini very distinct, central portions deeply coloured. Spleen small, natural. Right Kidney larger than normal ; surface smooth, ivory-like with stellate vessels, very pale on section ; ureter and pelvis dilated ; many ecchymoses in pelvis. Left Kidney much larger than normal, like right, besides, along lines of pyramids many small bodies, less than millet-seed, most distinct ; some moniliform, composed of lymphatic corpuscles in the connective tissue ; nowhere pus. A large ecchymosis occupying trigone. Reports, p. 258.

31. *Anæmia.*—In one case, a female aged 36, the anæmia was due to post partum hæmorrhage, and was accompanied by great anasarca, which soon yielded to good food and iron.

32. *Encephalitis.*—This case was syphilitic. A tumour was found in the posterior part of the roof of the left lateral ventricle, and a smaller one in a similar position at the right side.

33. *Meningitis.*—One case, a girl aged 17, began as urticaria, changes in apices of Lungs made Tuberculosis suspected. Temperature rose to 105.4, and was reduced by cold baths with brandy internally. The effect of Salicylic acid was marked in effecting this. The finger and knee joints became swollen and tender. The eyes showed slight optic neuritis.

The finger and knee-joints became swollen and tender, with effusion into the knee-joints, the left elbow became red and tender, and the left tibia, the whole of the spine occiput and sacrum, and the loins, became very sensitive without external sign. Reports, p. 267.

Another case was under treatment for five months, and left the Hospital unrelieved, the disease was chronic and its nature obscure; there was paralysis of the left third nerve, and of the right side of the face, and the patient, a woman aged 40, had also an ovarian tumour.

34. *Sanguineous Apoplexy.*—All the cases were fatal. One is particularly described in St. Barth. Hosp. Rep., p. 239, under the name of—

Aneurism of Ant. commun. art.—G. O., aged 41, van driver, 6 weeks ago pulled hard at a rope and felt something give way in head, falling down insensible. On the morning of June 16 seemed well, did no heavy work, but was suddenly seized with a curious feeling in his head, vomited, and fell insensible. On admission, two hours after, quite comatose, face dusky, eyelids almost closed, pupils very contracted, axes divergent, eyeballs occasionally rolling, respiration slow, deep, irregular, occasional sighing and stertor, mouth somewhat open, arms less motionless, and partly relaxed. Pulse 48, full regular arteries, not hard or tortuous. No Heart murmur. Temperature 96.8, surface cold, cutis anserina. An hour after admission limbs rigid, occasional relaxations of buccinators, general muscular twitchings, with chronic movements of lower jaw. Two hours after admission temperature 99.5, skin warm and moist; seven hours after admission bowels open, after purge and enema; still comatose, pupils much less contracted, right arm more powerless than left. Died day after admission.

P.M.—Effusion from an aneurism, size of small marble, of anterior communicating artery from its lower aspect. Blood has come through corpus callosum on right side into ventricles, and passed by velum interpositum to upper surface of cerebellum; fourth ventricle full of blood. No hæmorrhage in Pons; rest of cerebral arteries seem free from atheroma. Right Retina showed many small hæmorrhages, also hæmorrhages in sheath of optic nerve. Heart and Kidneys healthy; Aorta almost free from atheroma.

Four other cases are on record, two mentioned by Lebert, one by McDowall, one by Greenfield. Reports, p. 239.

In two cases the hæmorrhage took place into the right Hemisphere; in two into the left; in one into the Pons Varolii; in one into the Pons and Cerebellum; in one into the Pons and right Hemisphere; in eight the left ventricle was hypertrophied; in one the Heart was fatty; in eight there were granular Kidneys; in one fatty Kidneys; in four the Arteries were atheromatous. The fatty Heart, Liver, and Kidneys, were in the same subject.

In one case of Cerebral hæmorrhage on the left side, the Heart was hypertrophied, both Kidneys showed Cystic disease, there was a large Spleen, and there were abscesses in the Lungs; in another similar case there were infarcts in both Lungs.

One case, a man aged 56, who died the day of admission, the following is a short account:—He walked with some assistance into the hospital, saying he had had a fit, but was perfectly able to answer questions, and wanted to go home. Soon after, his pupils became contracted, and he died two hours after admission. His face was blue at the time of death. Post mortem it was found that blood had burst into the fourth ventricle, and filled the other ventricles.

35. *Cerebral Tumour.*—In one case, a boy aged 9, a tumour was found springing from the anterior surface of the Pons, and the right side of the Cerebellum.

36. *Cerebral Affection.*—In one fatal case, a man aged 58, the patient was apparently imbecile; he had paralysis of his Bladder, and there was an obscure history of fits of some kind. Cerebral softening was expected, but post mortem nothing was found in the brain, and the further examination of the body was not allowed. The patient was a German, a stranger in England.

36. *Hemiplegia.*—Of those discharged, the right side was affected in 22; the left side in 22. Ten cases were partial or slight, 3 were old-standing cases In 4 cases of right Hemiplegia the speech was affected; in 3 cases softening followed; 1 case was complicated with Hemianæsthesia; 1 with Paralysis agitans; in 3 there was a history of Syphilis: in 1 there was present an aortic cardiac murmur; in another case of slight right Hemiplegia of three years standing, Paraplegia supervened; another had Meningitis; another came on gradually after Vertigo; another case was complicated with granular Kidneys and hypertrophied Heart. In all the fatal cases the right was the paralysed side; in one there was atheroma of the mitral and aortic orifices, and softening of the left corpus stratum.

37. *Cross Paralysis.*—Of the right extremities and the left side of the face, headache on the right side and posteriorly. There was also atrophy of the choroids.

38. *Paraplegia.*—Two cases which were relieved were partial, one of them syphilitic; another complete case which was relieved was also syphilitic; one which was not relieved was the result of injury. One patient afforded a demonstration of the physiological effects of strychnine by drinking on her own account out of her medicine bottle. The effects were transient hæmoptysis; then twitching and pain, and opisthotonos. She was kept under chloroform for six hours. The amount of strychnine was estimated at half a grain.

In the fatal case a tumour was found springing from the laminæ of the sixth and seventh dorsal vertebræ, and pressing on the cord.

39. *Infantile Paralysis.*—One most interesting case of all but universal paralysis, following exposure to heat, was brought before the Medico-Chirurgical Society, on April 24, 1877, by Dr. Andrew and Dr. Duckworth. At the end of June, 1876, the patient, a girl aged 2½, had been exposed to great heat during railway travelling in the United States. On July 1st, after being exposed to severe sunstroke, she suddenly fell, having lost power in the legs, but did not lose consciousness. Subsequently she had occasional delirium, and was very irritable. On admission, at the end of July, there was complete loss of power in all the limbs and the Sphincters, sensation was lost in all the paralysed parts, and perhaps in all other parts as well. The muscles of the limbs were wasted, and did not respond to faradism. There were no cerebral symptoms, and the viscera of the Thorax and Abdomen were apparently normal, except a slight enlargement of the Liver, probably ricketty. The treatment consisted of good nourishment, codliver-oil and iron, followed by Belladonna in large doses, with faradism. Power began to return first in the arms, and all the symptoms improved. There was a slight and transient intercurrent attack of Broncho-Pneumonia; the child also passed a Lumbricus. In three weeks after admission the Sphincters regained power, and the child began to feed herself; faradisation also began to provoke muscular movements. Belladonna was stopped, and iron and strychnine given after two months. Albuminuria, with slight Anasarca appeared, the former lasting rather less than a month, but both completely disappearing; there was also a rash possibly due to Belladonna, but possibly also due to an intercurrent attack of Scarlatina. The temperature was high throughout, a point opposed to infantile Paralysis. The ophthalmoscope showed nothing. In three months after admission, four months from the first seizure, the child was quite well. The age, previous health, absence of cerebral symptoms, and electrical condition of the muscles, made the case resemble Infantile Paralysis, but the loss of sensation and of power in the Sphincters, and complete recovery, seemed opposed to this view. It was eventually considered to be a case of heat-stroke, in a frail and ill-nourished child.—*Lancet*, April 28, 1877, p. 611.

40. *Other Paralyses.*—One of right arm in a girl of 14, relieved; 1 of right hand in a girl of 11, relieved; 1 in a girl of 16, after smallpox, not relieved; 1 in a man of 49, a signal inspector on the Great Northern Railway, both arms and legs affected, not relieved; 1 of partial Paralysis of all the extremities in a woman of 37, relieved; another in a woman of 37, of the left leg with œdema, relieved; 1 of the right arm in a man of 38, relieved: 1 of the Serratus Magnus muscle in a shipwright aged 37, not relieved.

41. *Tetanus.*—One case in a boy, aged 9, who was relieved, seems to have followed a chill and fright from upsetting of a water-butt. Large doses of the Bromide of Potassium were used. Dr. Southey, Clinical Society, in *Lancet*, May 5th, 1877, p. 551.

In one of the fatal cases, a man, aged 22, post mortem examination showed much spinal fluid, softness of the spinal cord, but nothing else.

42. *Hydrophobia.*—One fatal case in a boy, aged 5. Post mortem appearances nil.

43. *Epilepsy.*—In two cases which were relieved there was a history of potus; in one fatal case there was a history of potus, and Pneumonia supervened; the other fatal case dated from the Clerkenwell explosion, 1865: post mortem appearances nil.

44. *Chorea.*—In one case, which was discharged, there was rheumatic fever for the third time; in another, Rheumatism, complicated with a cardiac murmur; in five cases there was a cardiac murmur; in two cases a history of fright; in two cases Pregnancy with a history of fright; in one Hemianæsthesia; and in one Nystagmus.

One of the fatal cases, a girl of 18, was complicated with Rheumatism. The post mortem appearances were as follows:—Brain natural; throughout the spinal cord the grey matter was indistinct; in the cervical portion the substance of the cord was diffluent. Old marks of Pleurisy on both sides; recent Pleurisy on left side; Pericardium adherent; mitral and aortic valves had vegetations; aorta atheromatous.

In the other, a boy of 14, the appearances were as follows:—Spinal cord soft throughout; all grey matter indistinguishable from white, except in lumbar region. Hæmorrhages on the summit of the brain, under the Meninges, of the size of half-a-crown; on the back surface of the heart a spot of the size of sixpence, surrounded by a zone of hæmorrhage looking like an embolism, wedge-shaped. The left side of the Heart studded with granulations. Infarcts in the kidneys. Brain natural.

45. *Hysteria.*—One case had Trismus; one Neuralgia of the left hip-joint; one general Hyperæsthesia.

46. *Hysterical Paralysis.*—In two cases Paraplegia; in one, left arm; in one, all the limbs paralysed.

47. *Pericarditis.*—In one case after Rheumatism; in two cases there was no history of Rheumatism at any time; one case was epileptic; one had Periostitis; one had right Pneumonia.

48. *Endocarditis.*—The three fatal cases of Endocarditis were as follows:—One had also Pericarditis; one presented the following post mortem appearances:—Hæmorrhage beneath the skin, Pericardium, Endocardium, Peritoneum, Intestines, Liver, Kidneys, Stomach, Meninges of Cerebrum and Cerebellum. The left Ventricle was hypertrophied, the aortic valves being principally attacked; the mitral less so. Infarcts in the Spleen and Kidneys. The other had Albuminuria, and the post mortem appearances were as follows:—The aortic and mitral valves attacked; Spleen large, adherent all round, containing a blood cyst with ragged walls; infarcts in other parts and in Kidneys.

49. *Valve Disease.*—Of the cases discharged the following are the facts:—

Aortic. One case complicated with Aneurism; one with Dementia.

Mitral. Two cases complicated with Pleurisy; one with Pneumonia; one came on after Chorea; in one there were symptoms of adherent Pericardium.

Complicated. Five cases of Aortic and Mitral disease. One complicated with left Pneumonia; one of Mitral and Tricuspid; one of Aortic and Pulmonary disease.

The fatal cases as follows:—

Aortic Disease—Malformed Heart.—Face, neck, and ears rather deeper in colour than usual; cardiac dulness increased; cardiac impulse irregular; indistinct systolic murmur, faint everywhere, but loudest at base, not louder to right than left of Sternum; no thrill felt; pulse regular, slightly sudden; severe pain at times at base of Heart.

P.M.—General dropsy; hardness of Liver, Spleen, and Kidneys; Heart much hypertrophied and dilated universally, Valves normal; Aorta abruptly narrowed, only just admitting tip of forefinger at point of junction of Ductus arteriosus, at which point was a slight internal ridge, beyond which the calibre was normal. Reports, p. 101.

Of one fatal case of mitral disease, in a boy, aged 8, the following is the history: Pains began one month before admission, swelling of joints, slight palpitation of Heart, frontal headache, a loud double murmur at apex of Heart. Ten days after admission his temperature reached 104, and symptoms got worse; pupils became unequal; capillaries of face, neck, and thorax distended; the discs of the eye became swollen, dim, with large and tortuous veins, but no hæmorrhages. This state continued for some weeks, symptoms getting worse at night; once or twice there was splenic tenderness with rise of temperature. A month after admission the mouth became drawn to the right, the eyes converged, and he complained of giddiness, dimness of sight, and buzzing in the ears. Later on the mouth became more drawn, but the other symptoms improved. About 10 weeks after admission the discs of the eyes were more obscured, the left surrounded by buff-coloured effusion; urine and fæces passed involuntarily, and became semi-conscious. Temperature 101–105, pulse 120–150. External squint and rigidity of the upper limbs were noticed, then complete unconsciousness, Purpura, and death. The urine normal throughout.

P.M.—Old extravasation over right hemisphere and lining right middle fossa; recent effusion over right half of cerebellum, along margin of right temporo-sphenordal lobe; small hæmorrhages over both hemispheres; an embolus in right middle cerebral artery at entrance of sylvian fissure; cerebellum softened, and disorganised; a large blood cavity in right half and partly in left, reaching surface on right. No ruptured vessel found. Heart left side hypertrophied; mitral valves covered with masses of fibrinous vegetations; infarcts in Spleen, which was adherent. Liver soft, nutmeg; infarcts in Kidneys, old and recent; Purpuric spots under Pleura, Peritoneum, Pericardium, Endocardium. *Lancet*, April 1, 1876, p. 494.

Complicated—Mitral, Tricuspid, Pulmonary, perforated Septum Ventriculorum.—Female, aged 20; short; fingers, toes and nose clubbed; short of breath from birth; complexion purplish, colour only deep after exertion or during a cold; slightly increased impulse; very loud systolic murmur, loudest midway between left base and apex, audible less loudly at angle of scapula. Præsystolic murmur occasionally heard at apex, and a thrill over cardiac area; pulse feeble and irregular. Died after a cold; dropsy preceding death.

P.M.—Heart hypertrophied; right ventricle much dilated; mitral and tricuspid fringed with growths; pulmonary orifice contracted by partial fusion of valves; Ductus arteriosus closed; Aorta and valves normal, in upper part of septum ventriculorum an opening as large as a sixpence. The præsystolic and systolic murmur were probably due to Tricuspid obstruction and regurgitation; Pulmonary obstruction, Mitral obstruction, and regurgitation; and imperfect Septum Ventriculorum. Reports, p. 102.

Complicated—Mitral and Aortic (slightly); dilated Heart, adherent Pericardium, Hydatid of left Kidney, with contraction, left Pleurisy.—A man aged 46. History

well till eight months ago, since when cough, dyspnœa, pains in back and shoulders, and palpitation. Rheumatic fever twenty-four years ago. On admission, cough with slight cyanosis during cough; sputa frothy, sometimes bloody; paroxysmal dyspnœa with orthopnœa; less œdematous for last three months; albuminuria; pulse irregular; cardiac impulse increased upwards; impulse best felt in 4th space; præsystolic thrill at apex; dulness increased downwards and outwards, not upwards; heart's action irregular; at the apex a double murmur heard, also outside nipple, and faintly behind; a double murmur at the right base, the diastolic part heard along the sternum, and best heard at the Ensiform cartilage; the systolic heard at the Ensiform cartilage. Before death, which occurred on July 27, there was thrombosis of the right femoral vein, with sloughing of the right thigh.

P.M.—Recent left Pleurisy; left lung œdematous with patches of pulmonary infarction at base; right pleura obliterated. Pericardium adherent in front of heart; Heart very large, dilated, especially the left side; mitral orifice much dilated, the valves thickened and opaque; the Aortic valves thickened and opaque; Liver nutmeg; Spleen large and tough; left Kidney much contracted, with cysts; pelvis much dilated, and containing three small stones. Above the pelvis a hydatid, the size of a walnut, not obstructing the passage. Reports, p. 255.

Of the remaining fatal Mitral cases 4 had Nephritis, 1 Peritonitis, 1 Erysipelas. Of the remaining complicated cases all had disease of Aortic and Mitral orifices, except 1 which had Tricuspid regurgitation with Mitral stenosis. One of those with Mitral and Aortic disease, a man aged 40, became maniacal before death.

50. *Chronic Fibroid changes in the Arteries.*—In this case with a history of alcoholism, there was Albuminuria. The patient was a colporteur aged 57.

51. *Aneurism of Aorta.*—The fatal cases were as follows:—

A greengrocer, aged 34. The aneurism began just beyond the left subclavian artery; the orifice was the size of a five-shilling piece; the cyst was filled with fibrin extending on both sides of the Aorta, and consisting of four smaller loculi. The skin of the back was almost broken through, the third and fourth ribs were necrosed and broken, 2 inches of each having disappeared. The arch of the fourth dorsal vertebra was wholly eroded to the spinal canal, and formed a cavity from the third to the tenth vertebra outside the Pleura.

A postman, aged 43. Aneurism of the first part of the arch, the orifice about the size of a shilling, about 1 inch above the Aortic valves, the cyst being the size of a small orange, pressing on the left Pulmonary Artery and both Venæ Cavæ.

A plumber, aged 47. Aneurism of the descending Thoracic Aorta; gangrene of the thigh before death.

A dock labourer, aged 41. The Aneurism affected the whole of the arch; the Innominate Artery slightly dilated, a pouch pressing on the Trachea. There was also a Hydatid of the Liver and Granular Kidneys; hæmorrhage took place into the left Lung.

A woman, aged 42. Aneurism of the descending part of the arch, the transverse part dilated. An opening into the Œsophagus.

A woman, aged 35. Aneurism of the arch, pressing on the left Bronchus. Tubercle in the Lungs, and several gangrenous cavities.

The following appears in the Reports:—A woman, aged 41. *Displaced Kidney;* history of Rheumatic Fever fifteen years ago, with subsequent attacks of Rheumatism, of Dropsy four or five years ago, and of cough for the last few months. On admission the face was puffy, but there was no Ascites or Anasarca. Cardiac dulness to right edge of Sternum and to left of left Nipple. At right base and down Sternum a double murmur, loudest at Ensiform cartilage and decreasing towards apex. She was discharged on Oct. 8th, the murmur being only audible in the erect position, and readmitted June 14, 1876, on which day she had been seized with sudden swelling of the face, lips, and eyelids. On admission the face, lips, eyelids, head and neck were swollen; there was no œdema of the trunk or limbs. Great dyspnœa which increased, a double Aortic murmur all over the front of the chest, impaired resonance at the left apex in front, and rather below the right apex behind. No air entered the left side of the chest. On June 15 Dysphagia, and voice became indistinct; in the evening she passed 3 ounces of albuminous urine for the first time for forty-eight hours; on June 16 she died suddenly.

P.M.—An aneurism of the Aorta just above the Aortic valves, pressing on and obstructing the Vena Cava superior and Pulmonary Artery. The Mitral and Aortic valves very atheromatous, the latter calcified and adherent; the Aorta very atheromatous. The left Kidney was displaced nearly into the Pelvis, much flattened, and about half the natural size, the Hilus turned forwards and supplied by an artery from the Aortic bifurcation. The left Ureter much dilated; a probe could not be passed from it into the bladder. Reports, p. 253.

52. *Aneurism of Cœliac axis.*—Death by rupture, the patient a man aged 32.

53. *Aneurism of Abdominal Arteries.*—Aneurism of the Abdominal Aorta just below the Diaphragm in a man aged 39. Death by rupture into the right Pleura.

The occupations of the patients who were the subjects of Aneurism, were as follows:—Discharges—*Men*: Costermonger, Coachman, Porter, Labourers (3), Engine-fitter, Rivetter, Warehouseman, Waterman. *Women*—Housewife, Dressmaker. Deaths—*Men*: Greengrocer, Postman, Plumber, Porter, Printer, Dock Labourer. *Women*—Housewife, Servant, Furniture Dealer. Little generalization is thus possible on this head.

54. *Obstruction of Veins.*—Obstruction to portal circulation due to large mesenteric glands. The patient, a boy aged three, was relieved.

55. *Chronic Enlargement of Glands.*—In the fatal case, a man aged 26, the glands of the anterior and posterior Mediastinum, the Mesentery and the pelvis were enlarged, and there were deposits in the Liver.

56. *Exophthalmic Bronchocele.*—The ages ranged from 20 to 33; in one there was Albuminuria.

57. *Addison's Disease.*—A boy aged 15. Family history, one brother had enlarged cervical glands, one aunt had Phthisis. He had had three attacks of Rheumatism in three years, the last attack, three months before admission, and the bronzing began subsequently. In September, 1875, he began to suffer from bronzing, Epistaxis, and giddiness, which have continued. On admission, he was small and thin, with dark smoky brown hair, dark brown eyes, the sclerotics pearly, the choroids showing increased pigmentation. Skin generally bronzed, especially the exposed parts, also the axillæ groins, navel, and nipples; patches and lines on the mucous surface of the lips and cheeks. A few black spots over the body and limbs. Breath unpleasant. Temp. 98.4, pulse 92, small and weak. Thorax constricted below, percussion impaired at left apex with coarse respiration and prolonged expiration, moist sounds above left scapula. Crepitation at the end of respiration at right base behind and in front. Heart's apex beat in fifth space, blowing systolic murmur at apex. Pain in left hypochondrium, no enlargement of spleen, no tumours, appetite poor, bowels relaxed, urine natural, 11 grammes of urea in 24 hours, no increase of Leucocytes in blood, and sleeps well, but occasionally wakes with headache. Chief ailments increasing weakness, loss of appetite, vomiting after breakfast and cough.

P.M. Face, chest, belly, thighs, arms, highly pigmented; small spots, almost black, over arms, size of pin's head. No pigmentation of Tongue, Pharynx, Gullet, or Windpipe. Thymus large. Left Pleura showed many Ecchymoses on both its layers, one as large as a shilling. Many old adhesions over right lower lobe, no fluid. Pericardium had old adhesions round vessels at base, and a large white spot on front of heart. Pericardial fluid, brown and turbid. Many small hœmorrhages over both layers of Pericardium, and large ones under Endocardium of left side of heart. Mitral valve thickened and opaque with many granulations. Aortic valves thick and opaque. Right Lung greatly dilated Bronchi, and round them patches of solid Lung. Liver, Stomach, and Kidneys, natural, Spleen almost pulpy. Ductless glands of intestine much enlarged, Mesenteric glands very large, rather hard. Semilunar Ganglia, and their nerves looked natural, surrounded by several large glands, some of them slightly pigmented. Both supra-renal bodies enlarged, especially right, and adherent to surrounding organs, Liver and Kidney. In the right several small abscesses filled with curdy pus, the tissue softer, more elastic, and more translucent than usual in Addisons's disease. Left contained also small abscesses, but fewer, some calcified nodules, tissue firmer and more opaque than

right. On section, broad and interlacing bands of white fibrous tissue were seen, the intervals filled with fat cells.—Reports, p. 257.

58. *Croup.*—Of the cases which recovered, one, a boy aged 4, had tracheotomy performed. Of the fatal cases, 5 had Tracheotomy performed. In one case air entered to both bases before the operation, œdema of Lungs. Towards the end of the operation, dyspnœa came on without obstruction of the tube. Post mortem, false membrane was found, forming a perfect cast of the Trachea, and extending to bronchial tubes of the third magnitude, and in patches beyond. In all the membrane easily stripped off. In one it extended to the two first divisions of the Bronchi. In another it covered the Velum Palati, Tonsils, Epiglottis, and Larynx, but no further. In another it was situated in the Larynx and Trachea, there were a few patches of Catarrhal Pneumonia. In another it extended below the Larynx, where a piece of false membrane was found lying across the Trachea. In another it was situated only in the Larynx, the Lungs were collapsed at the back.

59. *Bronchial Catarrh.*—One patient had intercurrent Pleurisy on the left side. One patient, a woman, aged 60, under the influence of drink, to which distress had driven her, attempted suicide by jumping into the Thames; she floated from the Temple to Blackfriars, where she was recovered.

60. *Acute Bronchitis.*—In 2 children there was Rickets; in 2 other patients Diarrhœa; in 1 Syphilis; in 2 Pleurisy supervened, one right, one left; in 1 Pneumonia; in 1 Urticaria; 1 came on after Scarlatina.

61. *Chronic Bronchitis.*—In 2 fatal cases the Heart was dilated; in 1 there was Albuminuria; in 1 Phthisis; in 1 Mania.

In 1 case, which was discharged, the Heart was dilated; in 1 Diarrhœa; in 1 double Phthisis; in 2 Albuminuria; in 1 Hæmatemesis; 1 had left Pleurisy; 1 suffered from Bronchiectasis as a sequel of Pleurisy.

62. *Pneumonia.*—Of the patients discharged, the right side was affected in 30; left side in 32; both sides in 2. One had intercurrent Varicella; one was complicated with Morbus Cordis; in one case of left Pneumonia of the apex there was right Herpes Zoster and strumous Ophthalmia.

Of the fatal cases, the right side was affected in 5; the left in 6; both in 5. One had Albuminuria; one Meningitis of the vault.

63. *Lobular Pneumonia.*—One case, which recovered, a boy, aged 7, had Pleurisy on same side, right; another a girl, aged 7, had Lobar Pneumonia on the left, and Lobular on the right.

64. *Pleuropneumonia.*—Discharges: Right side, 22; left side, 13; double, 1. One case complicated with mitral regurgitation. In all the fatal cases the left side was affected; one had Peritonitis and right Pleurisy.

One fatal case is thus described in the *Lancet.* Pleuropneumonia, Paracentesis, Embolism of Pulmonary Artery. Male, æt. 38. Patient had left Pleuropneumonia, relieved by Paracentesis, and was recovering when, after walking upstairs, he was suddenly seized with extreme dyspnœa, livid surface, pulse 90 and small, Respiration six or eight a minute, long and deep, not noisy, with action of ordinary and extraordinary muscles of inspiration, the jaw half set open, the left side of the chest motionless.

P.M.—All the systemic veins gorged, right side of Heart distended, left Ventricle contracted, orifice of Pulmonary Artery plugged with a clot with impressions of columnæ carneæ, or Musculi pectinati. Right Lung over-distended, extending across the middle line. There were usual signs of left Pleurisy, the Lung collapsed.

65. *Abscess of Lung.*—A Gangrenous Abscess of the right Lung in a drover aged 34. Recovery.

66. *Cirrhosis of Lung.* In those discharged: right side affected in 1 case, left side in 1 case, both sides in 4 cases. In one case there was spinal curvature, the Heart was dislocated, and there was a systolic murmur at the apex; in another there were symptoms of amyloid degeneration of the Kidneys and Liver. Of the

fatal cases, the right side was affected in one, and the left in the other; in the latter the Aorta was contracted, and there was a fatty Heart.

67. *Emphysema.*—In two cases discharged there was Phthisis, one of each Lung. Of the fatal cases, two had Morbus Cordis, one whose arteries were atheromatous had an Embolus in the Kidney, and also suffered from Vertigo.

68. *Pleurisy.*—Discharges: Right side, 20; left side, 30; double, 4. Three cases were tapped, in 3 there was Nephritis, in 1 Pericarditis, 1 came on after injury. Of the fatal cases, the right side was affected in 1, the left in 3; 1 had Phthisis.

69. *Chronic Pleurisy.*—Of those discharged, the left side was affected in 2 cases, the right in 1. The fatal case had the left side affected, the left Lung was completely retracted and the side fallen in.

70. *Empyema.*—In the patients discharged the left side was affected in 1 (in which the disease followed a stab and was localised), the right side in 2. In the fatal case, the left side was affected, the left Lung became gangrenous, and there was Pericarditis.

71. *Pneumothorax.*—In the case which recovered, the left side was affected. In the fatal case the left side was also affected; there was right Pneumonia and Phthisis.

72. *Mediastinal Tumour,* with progressive bulbar Paralysis, malignant growths in posterior Mediastinum, Liver, Mammæ, and inner plate of Calvaria.

I. Female, age 36. Confined two months before admission, three weeks before which she caught cold; three weeks after confinement she had severe flooding, and then the chest became sore, her tongue became enlarged and useless, and small lumps began to be felt in the breasts.

On admission she lay on the left side, expression helpless, upper lip continually quivering, lower lip everted, saliva flowing from mouth, tongue quite motionless, except *en masse* by raising the Hyoid bone and Larynx. The Uvula symmetrical, soft Palate, not paralysed. Inability to swallow anything except a little fluid, but part of that got down Trachea. Temperature 99·1; pulse 66, irregular; respiration 20. Thorax narrow and flattened; some hard nodules in both Mammæ, painless and freely movable. Axillary glands large. Palpation and deep inspiration painful. Vocal vibrations absent on left. Heart's apex not displaced, no murmur; percussion dull on left side, resonant on right with rhonchus and sibilus. Dulness began at left border of Sternum. Bronchial breathing over left side.

Abdomen tender, Liver reached to right Ilium, moving with inspiration, several elevations to be felt on Liver. Spleen felt below ribs, immovable. Slight œdema of legs. Bad cough, frothy brownish Sputum. Urine natural. Great pain in Thorax and Abdomen. Power of articulation lost, but power of making sounds retained. The tongue retained taste and touch.

She was fed with the stomach pump and appeared slightly better, but died rather suddenly after a few days from Asphyxia.

P.M.—The mammary tumours were Sarcomata. A large tumour occupied the posterior Mediastinum, occluding the left Bronchus, pressing on the vessels and causing congestion and consolidation of the left Lung, in which were several cavities; the Liver contained sarcomatous masses; the Spleen was enlarged and hard; the uterine mucous membrane was rough and fungoid. On the inner surface of the skull were some sarcomatous growths, but none was found in the Medulla oblongata or at the roots of the Hypoglossal nerves. — *Medical Times,* September 23rd, 1876.

II. *Lympho-sarcoma of Mediastinum.*—Male, aged 52. Coachmaker.

History.—Cough, twelve months; previously healthy. Six months before admission noticed lump above left collar-bone, followed by other lumps in neck and Axillæ. Legs swelled, and then left hand and arm Rapid loss of flesh, six months.

Vomiting of food till two months ago. Occasional dyspnœa and dysphagia.

Pale and thin, lying on back or left side. Respiration, quiet; sleep disturbed by cough; sputum bronchitic. Temp. 101·8°. Pulse 120.

Considerable enlargement on both sides of neck, especially left, due to supra-axillary, supraclavicular, and cervical glands, partly solitary, partly matted together. Elastic, hard, painless, skin non-adherent. Large lumps in left Axilla; smaller in right. No changes in eye. Blood contains an excess of white cells. Veins on left shoulder enlarged; also on lower and front part. Walls œdematous; chest barrel-shaped. Distinct epigastric pulsation. Resonance impaired down to both nipples; complete dulness down Sternum. and at posterior apices. Over whole chest, especially at right base, rough inspiration and loud rhonchus, Laryngeal stridor, between the scapulæ tracheal breathing. Heart sounds indistinct, Abdomen apparently natural. Legs and hands œdematous. No enlargement of inguinal glands.

On January 9th he died, with pain in left side increased by breathing, and shortness of breath.

P.M. Notch in Liver reached to Navel. Slight general Peritonitis. The second piece of the Sternum showed a tumour the size of a nut, communicating through the bone with a large mediastinal tumour, which was very adherent to upper part of Sternum, and extended into base of neck. Both Pleuræ full of fluid, and very rough. Apices of Lungs could not be separated from the mediastinal tumour, which grew into them. The rest of the Lungs collapsed. Enlarged lymphatic glands where Pericardium is attached to Diaphragm, tumour descending over Pericardium, and projecting into Pericardium by the side of the vessels at the base of Heart. Superior cava almost blocked by new growth. The clots in the Heart very white; Serum very pale. The left branch of the Pulmonary artery much narrowed, the Aorta much narrowed after giving off the large vessels. Both Carotids narrowed. The growth had infiltrated Bronchi, and lower end of Trachea without much narrowing. Gullet much narrowed opposite bifurcation of Trachea. Lower lobes of Thyroid infiltrated. Tumour tightly adherent to vertebræ. Tumour, hard, white and yellow, marked with white lines into polygonal figures, size of hemp-seeds, part of it in a capsule. Microscope showed small round cells embedded in highly fibrous tissue. The Liver was large, circumference of Acini pale, but no white lines as usual in Leukhæmia. Spleen natural size, very soft, pale red, no white nodules. Stomach showed three enlarged, hard, white, lymphatic glands. Abdominal glands natural. Reports, p. 245.

III. Female, aged 46. The tumour embedded nearly all the vessels, &c., in the Thorax, but obstructed none of them except Innominate veins.

IV. Female, aged 60. The tumour grew from the lamina of the second dorsal vertebra, spreading into the canal, and pressing on the cord, adherent to the second rib; the size of a small apple; extending below the right brachial plexus.

73. *Gastritis.*—The fatal case had also double Phthisis.

74. *Chronic Ulcer of Stomach.*—The fatal case, a male aged 36, ended in perforation and Peritonitis.

75. *Dilatation of Stomach.*—Several cases, under the heads "Gastritis," "Chronic Ulcer," and "Dilatation," were noticed by Dr. Andrew in a paper in the *Medical Times and Gazette* for September 2nd, 1876, p. 255, under the head of "Gastric Disorder treated by Carlsbad Salts." The case of "Chronic Gastritis" occurred in a man aged 50. Symptoms, twelve months, loss of flesh, pain two hours after food, daily vomiting, sarcinæ, flatulence, constipation. Two cases of "Gastric Ulcer;" one with dilatation. The male case, æt. 45, had suffered from loss of flesh; coffee-ground vomit in large quantities; pain two or three hours after food, relieved by vomiting; flatulence and constipation, epigastric distension and succussion sounds. The female case, aged 35; symptoms of gastric ulcer eight years; pain after food, vomiting, flatulence, hæmatemesis, constipation. Two cases of "Dilatation," both males, æt. 34 and 56. Both were of some years standing, one having a history of Dyspepsia eighteen years; both having pain after food, relieved by vomiting; one hæmatemesis.

In all these cases Professor Ziemssen's method of employing Carlsbad salts was tried, as follows: Milk diet at first, gradually increased; Carlsbad salts, 1 to 1½ drachms, dissolved in ¾ pint of warm water, taken in three instalments within an

hour's interval before breakfast, and compound rhubarb pill if required. The results were as follows: In the case of Chronic Gastritis, all the symptoms but slight flatulence disappeared, the patient gaining weight. One of the cases of Gastric Ulcer recovered rapidly, the other somewhat slowly. One of the cases of Dilatation progressed as follows: The constipation and pain disappeared in 11 days, the vomiting in 14, the succussion sounds and great dilatation in 17. The other case proved somewhat obstinate, and the stomach was washed out daily, the patient learning to do it himself, improvement, but not cure, resulting.

76. *Dysentery.*—The fatal case, in a man aged 56, was complicated with double Phthisis.

77. *Intestinal Obstruction.*—In one of the fatal cases, a woman, aged 70, the cause was internal strangulation through a hole in the Mesentery.

78. *Colic.*—A male patient, aged 31, who recovered, had Peritonitis. Cause unknown.

79. *Diarrhœa and Vomiting.*—One case with jaundice.

80. *Cirrhosis of Liver.*—In four cases there was Albuminuria, probably due to a similar state of Kidneys.

81. *Lardaceous Liver.*—The fatal case followed Syphilis; the patient had also an ovarian tumour.

82. *Hydatids of Liver.*—One case (a woman, aged 48) was admitted as an Ovarian tumour. It was, however, ascertained that she had been operated on for that disease nine years ago, with the result of the removal of a quantity of Hydatids of various sizes; after this it remained quiescent for eight years, and then grew rapidly. The woman was tapped, two distinct cysts being punctured, and clear fluid was withdrawn containing innumerable living buds of Tænia Echinococcus.

82. *Enlargement of Liver.*—One of the fatal cases, complicated with Paraplegia, died from double Phthisis.

83. *Gall Stones.*—In the fatal case there was, notwithstanding, bile in the duodenum.

84. *Leucocythæmia of Spleen and Lymphatics.*—One fatal case, in a man aged 39, is thus described in the Reports:—

On admission no marked cachexia, sight good, slight atrophy of choroid. Temperature 99·6. Pulse 100. Cervical and axillary glands both sides enlarged. Lungs natural. Systolic murmur at base of Heart with accentuated Pulmonary second sound. Spleen very large, occupying all left side of Abdomen. Liver, dulness slightly increased downwards. In general glands slightly enlarged. Left Tibia and Ankles somewhat enlarged. Blood contained excess of well-formed Leucocytes. Slight Albuminuria. Bowels generally confined. Enlargement of Abdomen noticed three months before admission, with giddiness, and on one occasion fainting. Large doses of Quinine (30 grains twice a day) eventually produced cinchonism without diminishing size of tumour, and had to be discontinued. A fortnight after admission, right Hemiplegia with alteration of cardiac murmur, inability to say anything but "No" and "Yes" irrelevantly, inability to write, confusion of intellect, exaggerated reflex sensibility, and increase of size of Spleen. Phosphorus pcrles were exhibited, and the tumour and number of Leucocytes in the blood decreased. Finally he sank rapidly and died.

Post mortem. Heart mitral valves thickened, covered with vegetations, valves partly adherent. Above tricuspid valves a small tumour the size of a pea. Liver large, pale, natural consistence. Spleen very large, adherent, capsule thickened, extending from fifth space one inch below navel. Substance thickened, several bodies apparently softened, infarcts embedded in it. Right Kidney contained a small mulberry calculus, in both Kidneys gritty matter between pyramids, and in their substance minute infarcts. Abdominal lymphatic glands much enlarged. Brain generally œdematous, all tract supplied by left middle cerebral artery softened, in parts cheesy and gritty, a firm white embolus in the artery at its origin. Cerebral

vessels contained agglomerations of Leucocytes. Microscope showed Leucocytes along course of capillaries in Kidneys and Liver. The matrix of the Spleen increased.

Patient's son, aged 2½, was admitted shortly after his father's death, with pallor, systolic murmur at base of Heart, enlargement of Spleen, ecchymoses about face, abdomen and thighs; bleeding from the nose. The Lymphatics were not enlarged, the blood showed no increase of Leucocytes. Patient was discharged improved, the Spleen still enlarged. Reports, p. 271.

In another fatal case, a man, aged 41, the only post mortem appearance were great anæmia of the Liver, Spleen, Kidneys, and Intestines, and great paleness of the blood clots.

85. *Peritonitis.*—Death, in one case of a girl, aged 19, resulted from perforation of the Vermiform appendix. Cause unknown. In another case of a chronic nature there was also Endocarditis, Mitral constriction, granular Kidneys, and signs of old Pericarditis.

Of the cases which recovered, one was due to a kick; two were circumscribed, one being caused by Scybala and changing its locality, one accompanied by right Pleurisy.

86. *Acute Bright's Disease.*—In one patient, a Hospital nurse, who recovered, the disease came on after Acute Rheumatism, and was associated with a mitral murmur. Another patient, aged 19, who recovered, had Pneumonia, and a boy aged four had Pneumonia of the right side, followed by Scarlatina, and then by Varicella. A fatal case, in a man aged 26, was complicated with Endocarditis.

87. *Chronic Bright's Disease.*—Of the cases which were discharged, one had pus in the urine, one uræmic convulsions, one transient right Hemiplegia with Aphasia, one case dated from parturition. Of the fatal cases, two were cases of Amyloid disease after Morbus Coxæ. In one of them, carbolic acid used externally produced a general symmetrical erythematous and pustular eruption. Two had fits. Two had Pleurisy.

88. *Pyelitis.*—The fatal case presented the following post mortem appearances:—The right Kidney passed across the middle line, and was connected to the remains of the left in a horse-shoe fashion; the left Ureter was pervious, there was no calculus; the left Kidney was a mere bag of pus.

89. *Renal Calculus.*—The case that recovered passed a large stone after colic; in the case which died, a stone the size of a walnut, was found in the right Kidney.

90. *Hæmaturia.*—One case had Morbus cordis, another enlargement of the Spleen, one was probably due to renal calculus, another followed a fall.

91. *Tumour of Ovary.*—One patient, single, aged 17, had a rise of temperature, followed by a discharge of fluid per rectum (the character of which was not ascertained), the tumour disappeared, and then became tympanitic, and rose probably from gas from the Intestines. She was discharged relieved of the tumour, but with commencing Phthisis of the left apex. Of the remainder who were not operated on, four were tapped, one was pregnant and was therefore left alone, the tumour was very large, but she has since had a perfectly normal labour at full term. Of the fatal cases, one died from Peritonitis after tapping, one from rupture of the cyst.

N.B. For the Ovariotamics see the Surgical Tables.

92. *Pelvic Cellulitis.*—One case followed instrumental labour, one followed amputation of the cervix uteri for Epithelioma, one resulted from the wearing a pessary several years unchanged; the woman was aged 40, and stated that she had worn it ever since she was 14. The size of the pessary, however, was quite incompatible with this statement, it was a rigid ring of wood, and had to be cut in two before removal. In one case of Pelvis Cellulitis in the first instance, the inflammation was exacerbated after the application of nitric acid to the cavity of the uterus. One case ended in an abscess which opened in the left groin, and one had also Pelvic Hæmatocele.

93. *Abscess in Pelvis.*—Opened in the Groin.

94. *Pelvic Hæmatocele.*—One case, with misplaced ovary, one anterior to the Uterus, one after miscarriage, one ending in an abscess which burst through the Abdominal walls.

95. *Elongation of the Cervix Uteri.*—In one case the cervix was amputated; in another case, with pregnancy, rest produced a diminution of the size.

96. *Fibrous Tumour of Uterus.*—Of these, seven cases were subperitoneal. In one case, a large mass was felt in the Vagina, which had much the feeling of a Polypus, or of an inverted Uterus; the Os uteri was at length felt posteriously high up, the diagnosis was confirmed after removal of the mass by the Ecraseur.

97. *Polypus of Uterus.*—One patient had double Exophthalmic goître, and left external squint. In one Septicæmia ensued after removal with the Ecraseur, in one case the tumour affected the posterior lip of the Cervix Uteri, and in another there were several fibrous Polypi.

98. *Retroversion.*—In three cases associated with prolapsed Ovary.

99. *Anteflexion.*—In one case, a girl aged 15, associated with nocturnal incontinence of urine which ceased on replacing the Uterus.

100. *Inversion.*—The patient aged 30. The Uterus has been inverted 2½ years, cause unknown. After three attempts at reduction, in one of which a rent was produced in the Vagina, the Uterus was removed. The patient recovered rapidly.

101. *Inflammation of Vagina.*—In one case idiopathic, in one case following prolonged use of a pessary.

102.—*Dilated Urethra.*—The actual cautery was applied four times, at intervals, to the whole Urethra, in the hope of producing contraction, and curing the incontinence which was present, but without success.

103. *Menorrhagia.*—The fatal case (age 35) was instructive and unusual. The Uterus was apparently natural; hæmorrhages under the skin, both Pleuræ, Pericardium, Endocardium, and on the Liver. There was slight Pleurisy on the right side, double Phthisis, fatty degeneration of the Heart; the Spleen was pulpy, no enlargement of Malpighian bodies; excess of white blood corpuscles in the blood clots; one white patch on the Liver, none in Glisson's capsule.

104. *Pelvic Tumour.*—One was a large cyst in the pouch of Douglas, which was punctured, and 32 ounces of fluid withdrawn.

105. *Retroversion of the Gravid Uterus.*—In one case abortion followed at the fourth month after a strain, four days' symptoms preceding the abortion. In one case, in addition to the usual retention of urine, there was a discharge of blood either from the Uterus or Bladder. The Uterus was replaced by two fingers in the Rectum, the symptoms ceasing. In another case reposition was effected by an air-ball.

106. *Extra-uterine Gestation.*—The diagnosis of this case was doubtful; the principal points were cessation of Menses four months, swelling of Hypogastrium towards the right side, blue and velvety Vagina, soft Cervix; but there was no morning sickness or enlargement of breasts, except that the follicles were rather large. It was possibly a Hæmatocele.

107. *Subinvolution.*—In two cases nitric acid was applied to the cavity of the Uterus with relief.

108. *Erythema nodosum.*—Death from intercurrent Typhoid.

109. *Herpes* on the left cheek, in a girl aged 15, corresponding with the distribution of the facial nerve.

110. *Eczema simplex.*—The fatal case, in a boy aged 9, presented the following post mortem appearances:—Lungs œdematous, a few tubercles in the left; Spleen and Liver large and soft; Kidneys, cloudy swelling. The signs in fact of high temperature.

111. *Premature Birth.*—A child born prematurely in the sixth month survived its birth six hours.

112. *Debility.*—Some of the causes were as follows:—Miscarriage in 2, in 1 Alcoholism; in 3, improper or insufficient food; in 4, Syphilis, one was associated with Psoriasis and Phthisis of the right Lung, one with Albuminuria; in 4, Debility was due to over suckling, in 1 to Dysentery, in 1 to Diarrhœa, in one to Fever (? Typhoid), in 1 to Chronic Pleurisy of the left side, in 1 to a chill, in 1 to Congenital Morbus Cordis, in 1 to Gout.

Poisons:

113. *Corrosive Sublimate.*—Attempted suicide.

114. *Lead.*—The occupations of those affected were as follows:—Painters, 6; Carriers of white Lead in Lead-factory, 3; Caster of Lead, 1; Worker in Lead-factory, 1; Hawker, Stevedore, Carpenter, each 1.

Colic.—One had also drop-wrist and Bronchitis; one had fits of syncope; one had Mania; one Epileptiform seizures.

Palsy.—One had Gout.

115. *Potash.*—Given by a boy in a solid form, as a sweetmeat, by way of a joke.

116. *Ammonia.*—One attempted suicide; one swallowed a Liniment by mistake.

117. *Sulphuric-acid.*—One attempted suicide; one by mistake for gin, while drunk; one took it by mistake for ginger-beer.

118. *Nitric-acid.*—Two were insane; one took it while drunk, but yet knowing what it was; one attempted suicide.

119. *Acetic-acid.*—Mistake.

120. *Carbolic-acid.*—By mistake, when drunk.

121. *Intoxication.*—One suffered from Hepatitis, one had a fit, one had Albuminuria.

122. *Delirium Tremens.*—One man, a tailor's cutter-out, aged 38, stated he had been in the habit of drinking two pints of brandy every morning, before taking any food for several months.

TABLE II,

Showing the comparative Frequency and Mortality of each Disease at different Ages.

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
GENERAL DISEASES, A.																																											
Small-pox.. ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Chicken-pox ..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Measles	1	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	..	..
Sequelæ of M. ..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Scarlet Fever ..	5	2	..	..	4	12	..	..	3	2	..	..	2	1	..	..	2	2	..	..	2	2	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	18	22	..	**2**
Sequelæ of S. F..	4	3	**2**	..	1	13	..	..	3	..	**2**	..	..	1	**1**	..	2	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10	18	**5**	..
Typhus	..	..	..	..	..	..	**1**	..	..	1	..	..	1	1	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	2	**1**	..
Enteric Fever ..	..	..	..	..	3	..	..	..	5	3	..	..	2	5	**3**	..	15	6	**2**	**2**	1	2	**2**	**1**	2	..	**1**	..	..	..	..	..	..	..	..	..	..	..	..	..	28	16	**8**	**3**
Simple Continued Fever	..	..	..	..	1	..	..	..	..	1	..	..	..	..	**1**	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	2	**1**	..
Febricula	2	1	..	..	1	1	..	..	1	2	..	..	2	2	..	..	3	2	..	..	1	4	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	10	14	..	..
Ague—																																											
Tertian	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	1	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	1	..	..
Quartan	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				75 and upwards.				Total.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
GENERAL DISEASES, A (*continued*).																																											
Ague—																																											
Irregular ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	3	1	..	..
Sequelæ of Ague	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Diphtheria ..	1	..	..	..	..	2	**2**	..	..	3	..	..	1	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	6	**1**	**2**
Hooping-cough ..	..	1	**1**	..	..	..	**1**	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	**2**	..
Erysipelas—																																											
Cellulo-cutaneous (of Face) ..	..	1	..	..	..	..	..	..	1	1	..	..	..	3	..	..	1	6	..	..	2	1	**1**	..	1	1	**1**	..	1	..	..	..	1	1	..	..	..	..	..	7	14	**2**	..
Pyæmia	..	..	..	..	..	..	..	..	..	..	**1**	..	1	..	..	..	..	..	**1**	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	**1**	**1**
Sequelæ of P. ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Puerperal Fever ..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	**1**	..	2	..	..	..	..	..	**1**	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	**2**
GENERAL DISEASES, B.																																											
Rheumatism—																																											
Acute	..	..	..	..	1	1	..	..	7	6	..	..	16	25	..	..	28	47	**1**	**1**	18	10	**1**	..	9	9	..	..	1	3	..	..	..	..	..	..	..	1	..	..	80	101	**2**	**1**
Subacute.. ..	..	..	..	..	..	..	..	..	..	3	..	..	1	..	..	..	4	3	..	..	1	1	..	..	1	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	7	10	..	..
Gonorrheal ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	..	..	..
Synovial	..	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	1	..	..
Muscular—																																											
Lumbago ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
GENERAL DISEASES, B (*continued*.)																																											
Rheumatism—																																											
Chronic	..	..	..	..	..	..	..	..	1	..	..	..	..	3	..	..	6	3	..	..	2	5	..	..	3	4	..	..	1	3	..	..	2	2	..	..	..	..	..	..	15	20	..	..
Gout—																																											
Chronic	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	2	2	..	..	4	2	..	..	2	..	..	..	..	..	..	..	8	5	..	..
Gouty Synovitis .	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	2	..	..	..
Chronic Osteo-Arthritis.. ..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	1	..	..	1	..	..	..	..	2	..	..	2	..	..	..	..	..	..	..	..	..	..	5	3	..	..
Cancer—																																											
of Abdomen ..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	1	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	..	**3**	**1**
of Stomach ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	**4**	..	..	1	..	..	**1**	..	..	..	..	..	..	..	1	..	**4**	**1**
of Liver	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	**1**	**2**	..	..	1	..	**2**	..	1	..	**1**	..	..	..	..	2	**1**	**6**
of Intestines ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	**1**	..	..	1	..	..	..	..	**1**	..	..	..	..	..	2	**1**	**1**
of Rectum ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	**1**	..	..	..	..	..	**1**	..	..	..	..	..	..	..	..	2	**1**	**1**
of Kidney	..	..	**1**	..	..	..	**1**	..	..	..	..	..	..	..	..	..	..	..	..	..	..	**1**	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	**1**	**1**
of Bladder ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	**3**	..
of Pelvis . ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	**1**	..	..	..	..	..	..	**1**	..
of Uterus ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..	1	..	..	..	2	..	..	..	2	..	..	..	..	..	..	..	1	..	..
of Ovary ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	8	..	..
Epithelioma—																																											
of Cervix Uteri..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	..	..	..	5	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
GENERAL DISEASES B (*continued*).																																													
Lupus—																																													
Chronic	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Scrofula—																																													
Local Scrofulous Affections—																																													
Tubercular Meningitis	..	..	1	..	..	1	3	2	..	..	..	1	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	5	3
Phthisis Pulmonalis	..	..	..	..	..	..	..	..	..	3	..	1	5	6	1	3	10	15	6	14	18	11	5	7	3	7	3	4	..	3	2	..	..	..	1	..	..	..	..	..	36	45	18	29
Acute Miliary Tuberculosis ..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	3	3
Tabes Mesenterica	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..
Tubercular Peritonitis	..	..	..	..	..	1	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1	1	..
Rickets	..	1	1	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	5	1	..
Diabetes	..	..	..	..	..	..	..	..	2	..	..	1	..	..	..	..	3	..	3	..	2	1	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	7	1	4	2
Purpura—																																												
Simple	1	..	..	1	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	2	1	..	1
Hæmorrhagic ..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	2	..
Scorbutus ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
GENERAL DISEASES B (*continued*).																																												
*Anæmia	..	1	..	..	..	..	1	..	..	1	..	..	..	5	..	..	..	7	..	..	..	2	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	17	1	..
Chlorosis	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	7	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	..	..
*General Dropsy ..	1	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..
*Local Anasarca ..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
LOCAL DISEASES.																																											
DISEASES OF THE NERVOUS SYSTEM.																																											
DISEASES OF THE BRAIN AND ITS MEMBRANES.																																											
Encephalitis ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1
Meningitis (Simplex)	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	1
Yellow Softening .	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1
Abscess of Brain ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..
Apoplexy— Sanguineous ..	..	..	..	.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	5	2	..	..	4	1	..	..	1	..	..	..	..	..	..	..	11	3

TABLE II (*continued*).

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				Total.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE NERVOUS SYSTEM (*contd.*)																																											
Chronic Hydrocephalus	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Tumour	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		2	..
Cerebral Affection	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	3	1	..
DISEASES OF THE SPINAL CORD AND ITS MEMBRANES.																																											
Inflammation—Myelitis	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1	..	..
DISEASES OF THE NERVES.																																											
*Paralysis—																																											
Hemiplegia	..	1	..	..	..	1	..	..	1	..	..	..	1	1	..	..	7	..	..	..	6	2	1	..	6	2	..	1	8	4	..	..	2	..	..	1	2	..	..	..	33	11	1	2
Cross Paralysis	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Paraplegia	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	2	..	..	1	..	..	..	1	1	..	..	..	2	..	..	..	..	1	..	..	..	..	..	5	5	1	..
Locomotor Ataxy	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	..	..	..	2	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	9	..	..	..
Infantile Paralysis	..	1	..	..	1	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	3	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE NERVOUS SYSTEM (*contd.*)																																											
Local Paralysis—																																											
Facial Paralysis	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	1	2	..	..
Other Paralysis	..	..	..	..	..	..	..	..	1	2	..	..	..	1	..	..	1	..	..	..	2	2	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	5	..	..
FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM.																																											
Tetanus	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	1
Hydrophobia	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..
Infantile Convulsions	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Epilepsy	..	..	..	..	1	..	..	..	4	2	..	..	1	1	..	..	5	3	1	..	1	2	1	..	5	..	..	..	2	..	..	..	1	..	..	..	1	..	..	..	..	2	..	..
Epileptic Vertigo	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..	..	..	..	..	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	20	9	2	..
Epileptic Hemiplegia	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	..	..	..
Laryngismus	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Shaking Palsy	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..
Spasm of Muscles	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..
Chorea	4	11	..	..	..	..	..	..	2	13	1	..	1	8	..	1	..	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Hysteria	..	..	..	..	..	..	..	..	..	..	..	..	1	27	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	7	35	1	1
Hysterical Paralysis	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	4	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	27	..	..
	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE NERVOUS SYSTEM (*contd.*)																																											
Neuralgia—																																											
Diffused	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	4	..	..
Cephalalgia ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	1	..	..	..	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	2	..	..
Sciatica	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	2	2	..	..
Pleurodynia ..	1	..	..	..	..	..	..	..	..	1	..	..	1	3	..	..	..	..	..	..	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	4	..	..
*Hyperæsthesia ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..
Hypochondriasis .	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..
Obscure Nerve Disorder	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..
DISORDERS OF THE INTELLECT.																																											
Mania	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	1	..	..	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	2	3	..	..
Melancholia ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	2	..	..
Dementia	..	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	..	..
Idiotey	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE CIRCULATORY SYSTEM.																																											
DISEASES OF THE HEART AND ITS MEMBRANES.																																											
Diseases of the Pericardium.																																											
Pericarditis ..	..	..	..	..	2	..	..	..	..	1	..	..	1	..	..	..	2	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	2	..	..
Diseases of the Endocardium.																																											
Endocarditis ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	2
Valve Disease—																																											
1. Aortic ..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	1	..	..	..	2	1	5	..	..	..	3	1	1	..	3	..	..	..	1	..	..	..	..	..	..	12	4	4	1
2. Mitral ..	..	..	..	..	2	1	1	..	2	5	2	1	5	4	1	4	3	7	1	6	2	5	2	4	1	4	3	3	3	2	1	3	..	1	2	..	..	..	..	1	18	29	13	22
3. Pulmonary ..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
4, Tricuspid ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
5. Complicated ..	..	..	..	..	1	..	..	..	1	..	..	..	2	1	..	1	1	..	2	1	..	..	..	1	1	..	1	1	..	..	..	1	..	..	1	..	..	..	..	..	6	1	4	5
6. Congenital ..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..

DISEASES.	Under 5.				5-10.				10-15.				15-20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE CIRCULATORY SYSTEM (*continued*).																																												
Diseases of the Muscular Structure of the Heart.																																												
Dilatation.. ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..	1	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	2	2	..
Angina Pectoris ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Syncope	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
DISEASES OF THE BLOOD VESSELS.																																												
Diseases of the Arteries.																																												
Atheroma.. ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Fibroid Changes ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Aneurism—																																												
of Aorta	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	2	..	1	1	3	2	4	3	4	..	..	..	..	..	..	..	..	..	..	..	9	3	5	4
of Innominate ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
of Subclavian ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
of Cœliac axis ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE CIRCULATORY SYSTEM (*continued*).																																												
Aneurism — of Abdominal Arteries	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..
Epigastric Pulsation	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	2	..	..
Diseases of the Veins.																																												
Phlebitis	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Phlegmasia Dolens	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Obstruction ..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
DISEASES OF THE ABSORBENT SYSTEM.																																												
Hypertrophy of Glands— Chronic Enlargement of Glands	..	..	..	..	..	..	..	..	..	1	..	..	..	2	..	..	..	2	1	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	7	1	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF DUCTLESS GLANDS.																																												
DISEASES OF THE THYROID GLAND.																																												
Exophthalmic Bronchocele	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	3	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	3	..	..
Enlargement ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1
DISEASES OF THE SUPRA-RENAL CAPSULES.																																											
Addison's Disease .	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..
DISEASES OF THE RESPIRATORY SYSTEM.																																											
DISEASES OF THE RESPIRATORY SYSTEM NOT SIMPLY LOCAL.																																											
Croup	1	1	1	2	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	1	3

DISEASES.	Under 5.				5-10.				10-15.				15-20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE RESPIRATORY SYSTEM. (*continued*).																																											
DISEASES OF THE LARYNX.																																											
Laryngeal Catarrh.	2	1	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	3	..	..
Laryngitis—																																											
Acute	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Chronic	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1	..	..
Œdema of the Glottis	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..
DISEASES OF THE TRACHEA AND BRONCHI.																																											
Bronchial Catarrh	2	1	..	..	3	2	..	..	..	..	..	..	..	..	..	..	1	1	..	..	..	2	..	..	1	..	1	..	..	2	..	..	..	2	..	..	..	..	..	..	7	10	1	..
Bronchitis—																																											
Acute	4	3	2	2	1	3	..	1	1	1	..	..	1	2	..	..	4	5	..	1	..	4	..	..	2	1	..	..	..	..	..	..	..	..	1	..	..	..	..	..	13	19	2	5
Chronic	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	2	..	1	5	3	2	8	11	4	2	7	8	5	3	5	5	1	1	..	1	1	1	21	36	16	9
Asthma	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE RESPIRATORY SYSTEM (*continued*).																																												
DISEASES OF THE LUNG.																																												
Pneumonia ..	11	7	2	1	4	2	..	..	4	6	..	..	3	3	..	1	5	3	5	1	9	..	3	..	5	..	1	..	..	1	1	1	1	..	..	..	..	..	..	..	42	22	12	4
Lobular	..	..	1	1	1	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1	2	3	1
Sequelæ of Pn. ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Pleuro-pneumonia.	..	..	1	..	2	1	..	..	1	3	..	..	3	1	..	..	6	6	1	..	8	3	1	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	20	16	3	..
Abscess	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Gangrene	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	1
Congestion ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
*Hæmoptysis ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	..	..	..	4	2	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	12	2	..	..
Cirrhosis	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	1	..	..	2	..	..	..	3	..	..	..	1	..	2	..	..	..	..	..	..	..	..	..	7	1	2	..
Emphysema ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	3	..	2	..	..	..	3	1	..	..	..	..	..	..	..	..	1	..	6	1	4	..
DISEASES OF THE PLEURA.																																											
Pleurisy	..	..	..	..	3	1	..	..	1	..	..	..	2	2	1	..	7	11	1	..	11	3	..	1	6	3	..	..	2	3	1	..	..	..	..	..	..	..	..	..	32	23	3	1
Chronic Pleurisy	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	1	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	2	1	..	1
Empyema.. ..	..	..	..	..	1	..	..	1	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	3	..	..	1
Pneumo-thorax ..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE RESPIRATORY SYSTEM (*continued*).																																											
DISEASES OF THE MEDIASTINUM.																																											
Tumour	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	1	..	..	1	1	..	..	..	..	..	..	1	..	..	..	..	..	2	1	3
DISEASES OF THE DIGESTIVE SYSTEM.																																											
DISEASES OF THE STOMACH.																																											
Gastritis	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1	..	1	..	2	..	..	..	1	..	..	..	..	..	..	..	4	1	1	..
Chronic Ulcer ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	..	..	2	2	1	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	8	1	..
*Hæmatemesis ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	1	1	..	..	2	..	..	..	..	..	..	..	..	..	..	..	4	2	..	..
*Dilatation ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..
Dyspepsia ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	2	2	..	..	2	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	4	..	..
Gastrodynia ..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	2	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	3	..	..
Pyrosis	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
*Vomiting ..	..	..	..	..	..	..	1	..	..	..	2	..	..	..	..	..	..	1	..	..	..	..	..	..	2	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	6	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE DIGESTIVE SYSTEM (*continued*).																																												
DISEASES OF THE INTESTINES.																																												
Enteritis	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	1	1
Typhlitis	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..
Dysentery	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	2	..	..	..	..	1	..	..	1	..	1	..	..	..	..	..	..	..	..	4	1	1	..
Ulceration	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Perforation	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Abscess in the Sub-peritoneal Tissue ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	1
*Tympanites	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	2	..	..
*Obstruction	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	2
Diarrhœa	1	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	1	2	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	3	6	..	..
Colic	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..	1	1	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	3	2	..	..
Constipation ..	..	..	..	..	..	..	..	..	1	..	..	..	2	1	..	..	1	4	..	..	2	1	..	..	..	1	..	..	..	2	..	..	..	..	..	..	..	..	..	..	6	9	..	..
Diarrhœa and Vomiting	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	1	..	..	1	1	..	.	..	..	..	..	..	..	..	..	..	..	..	..	4	2	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE DIGESTIVE SYSTEM (*continued*).																																												
DISEASES OF THE LIVER.																																												
Cirrhosis	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	1	**3**	..	3	..	**2**	**2**	2	..	**3**	..	..	..	**1**	..	..	..	..	..	8	1	**8**	**3**
Lardaceous Liver..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	**1**	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	**1**
Parasitic Disease–Echinococcus hominis	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	2	1	..	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	4	2	..	..
Jaundice	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	4	3	..	..	2	1	..	..	..	1	..	..	2	4	..	..	..	..	..	..	..	..	..	..	9	9	..	..
Enlargement ..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	2	4	..	**1**	1	..	..	**1**	..	..	..	..	..	..	..	6	4	..	**2**
DISEASES OF THE HEPATIC DUCT & GALL BLADDER.																																												
Gall Stones ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	..	**1**	..	1	3	**1**	..
DISEASES OF THE SPLEEN.																																												
Hypertrophy ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..
Leucocythæmia..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	**2**	..	..	..	**1**	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	**3**	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE DIGESTIVE SYSTEM (*continued*).																																												
DISEASES OF THE PERITONEUM.																																												
Peritonitis	..	..	..	..	..	..	..	..	1	..	..	..	..	..	1	..	2	..	..	..	1	..	..	..	1	2	..	..	..	..	..	..	..	..	..	..	..	..	..	3	2	..	3
*Ascites	1	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	4	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	1	5	1	1
Tumours	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	1	..	..	..	3	..	..	..	2	2	..	..	1	1	..	..	1	..	..	..	..	..	..	5	8	..	..
Congenital Tumour	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
DISEASES OF THE URINARY SYSTEM.																																											
DISEASES OF THE KIDNEY.																																											
Bright's Disease—																																											
1. Acute ..	1	..	..	..	..	..	..	..	1	..	..	..	3	4	1	..	1	4	1	1	3	3	1	..	1	..	..	..	1	..	..	..	2	..	..	..	..	..	..	13	11	3	1
2. Chronic ..	..	..	..	..	..	..	..	..	..	1	..	..	3	..	1	..	..	3	6	4	5	4	3	2	4	2	5	3	2	2	3	..	2	1	3	1	..	..	..	..	16	13	21	10

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE URINARY SYSTEM (*contd.*).																																												
Abscess	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..
Pyelitis	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..
Calculus	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..
*Hæmaturia renalis	..	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	3	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	3	..	..
*Suppression of Urine	..	..	..	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..
*Diuresis	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
DISEASES OF THE BLADDER.																																											
Cystitis	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE GENERATIVE SYSTEM.																																												
DISEASES OF THE FEMALE ORGANS OF GENERATION IN THE UNIMPREGNATED STATE.																																												
DISEASES OF THE OVARY.																																												
Inflammation ..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..
Complex Cystic Tumour	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	5	..	1	..	3	..	2	..	1	..	1	..	..	..	..	..	..	..	..	..	10	..	4
DISEASES OF THE BROAD LIGAMENT.																																												
Inflammation— Pelvic Cellulitis..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	6	..	..	..	5	..	..	..	3	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	15	..	1
Abscess	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Periuterine or Pelvic Hæmatocele	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	8	..	..	..	9	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	20	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE GENERATIVE SYSTEM (*continued*).																																											
DISEASES OF THE UTERUS, INCLUDING THE CERVIX.																																											
Catarrh (Leucorrhœa	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	..	..
Inflammation (Chronic)	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	..	..	..	4	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	12	..	..
Granular Inflammation	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Congestion	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..
Stricture of the Orifice	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..
Stricture of the Canal	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..
Hypertrophy	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Elongation of the Cervix	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..
Non-Malignant Tumour	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
A. Fibrous Tumour	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	..	..	..	6	..	..	..	7	..	..	..	3	..	..	..	..	..	..	..	..	..	..	..	20	..	..
B. Polypus	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	4	..	..	..	4	..	..	..	..	..	..	..	..	..	..	..	10	..	..

TABLE II (*continued*).

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE GENERATIVE SYSTEM (*continued*).																																											
DISPLACEMENTS AND DISTORTIONS.																																											
A. Anteversion	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
B. Retroversion	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..	8	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	14	..	..	..
C. Anteflexion	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	3	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	8	..	..	..
D. Retroflexion	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	2	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	5	..	..	..
E. Inversion	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
F. Prolapsus Procidentia	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..
DISEASES OF THE VAGINA.																																											
Inflammation	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..
Abscess	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Hernia—																																											
Cystocele	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
DISEASES OF THE VULVA.																																											
Hypertrophy	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE GENERATIVE SYSTEM (*continued*).																																												
Vascular Tumour of the Meatus Urinarius ..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	2	..	..
Dilated Urethra ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
FUNCTIONAL DISEASES OF THE FEMALE ORGANS OF GENERATION.																																											
Amenorrhœa ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Dysmenorrhœa ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..
Menorrhagia ..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1
Pelvic Tumours ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	2	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
AFFECTIONS CONNECTED WITH PREGNANCY.																																												
DISORDERS OF THE GENERATIVE SYSTEM.																																												
Pregnancy ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Hæmorrhage ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..
Displacements of the Uterus— Retroversion ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	..	..
Abortion	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	..	..
Extrauterine Gestation	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
AFFECTIONS CONNECTED WITH PARTURITION.																																												
Hæmorrhage (after Abortion) ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
AFFECTIONS CONNECTED WITH PREGNANCY (*contd.*).																																											
Retention of part of Ovum.. ..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	2	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	..	..
AFFECTIONS CONSEQUENT ON PARTURITION.																																											
Subinvolution ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	11	..	..	..	11	..	..	..	4	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	26	..	..
DISEASES OF THE MUSCLES.																																											
Progressive Muscular Atrophy..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..
DISEASES OF THE CELLULAR TISSUE.																																											
Obesity	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	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.																																											
Erythema—																																											
E. Læve.. ..	..	1	..	..	..	1	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	..	..
E. Tuberculatum	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
E. Nodosum ..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	1	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	..	1
Urticaria	..	..	..	..	..	..	..	..	1	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	..	..
Psoriasis	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1	..	..
Herpes	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
H. Zoster ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Pemphigus ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Eczema—																																											
E. Simplex ..	1	..	..	..	..	1	1	..	1	..	..	..	2	..	..	..	1	..	..	..	1	..	..	..	2	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	8	4	1	..
E. Rubrum ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
E. Impetiginodes	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
E. Exfoliativum	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
E. Chronicum ..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Xeroderma ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	2	..	..
Molluscum ..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	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 (*continued*).																																											
PARASITIC DISEASE OF THE SKIN.																																											
Tinea favosa ..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Scabies	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Irritation caused by Pediculi ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..
CONDITIONS NOT NECESSARILY ASSOCIATED WITH GENERAL OR LOCAL DISEASES.																																											
Premature Birth..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	**1**
Debility	1	2	1	..	1	2	..	..	1	4	..	..	1	5	..	..	5	18	..	..	2	8	1	..	2	6	..	..	3	3	1	..	..	4	..	..	..	..	..	..	16	52	**3**	..
Pain	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	2	..	..
Phantom Tumour	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
CONDITIONS NOT NECESSARILY ASSOCIATED, &c. (*continued*).																																												
Fright	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Malingering ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..
Unknown.. ..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..
POISONS.																																											
METALS AND THEIR SALTS.																																											
Mercury—																																											
Corrosive Sublimate	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Lead—																																											
Lead Colic ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	2	2	..	..	3	..	..	..	..	2	..	..	..	..	..	..	..	..	7	4	..	..
Lead Palsy ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	1	..	..	..	..	..	..	..	..	..	3	..	..	..
Lead Vertigo ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..

DISEASES.	Under 5.				5–10.				10–15.				15–20.				20—				30—				40—				50—				60—				70 and upwards.				TOTAL.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
POISONS (*contd.*)																																													
CAUSTIC ALKALIES.																																													
Potash	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..
Ammonia	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	4	..	..	..
ACIDS.																																												
Sulphuric Acid ..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	1	3	..	..
Nitric Acid ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	..	..
Oxalic Acid ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Acetic Acid ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Carbolic Acid ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
VEGETABLE POISONS.																																												
Alcohol	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	1	..	..	1	2	..	..	..	..	..	..	..	..	..	..	..	5	..	..	1
Intoxication ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1	..	..
Delirium tremens	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..	3	..	..	..	3	1	..	..	2	..	..	..	..	..	..	..	..	..	..	..	11	1	..	..
Paraffin	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Soap Liniment ..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..

Table showing the Average Stay of the Medical Cases in Hospital, &c.

Within—Weeks of admission.	Discharged.		Died.		Remaining in at end of 1876.	
	M.	F.	M.	F.	M.	F.
1	99	96	93	59	16	24
2	142	156	28	30	12	9
3	139	145	38	19	11	7
4	112	159	22	20	7	13
5	92	123	14	22	5	10
6	79	89	10	14	3	8
7	42	60	7	7	4	7
8	36	45	6	2	6	2
9	22	29	5	5	1	2
10	10	24	4	4	..	2
11	8	16	3	1	2	..
12	10	15	1	1	1	..
13	2	9	6	..	..	1
14	6	9	2	..	..	2
15	9	4	..	..	1	..
16	8	2	..	..	1	1
17	2	6	1	..	3	..
18	1	9	2	..	1	..
19	6	2	..	..	..	..
20	3	3	1	1	1	..
21	3	1	2	..	..	..
22	..	2	1	..	..	1
23	..	1	..	..	..	..
24	2	1	1	..	..	..
25	2	..	..	..	1	..
26	..	1	..	..	1	..
27	..	1	1	1	1	..
28	..	..	..	1	..	..
29	..	1	..	..	..	..
31	..	..	1	..	..	..
34	..	..	..	1	..	..
36	1	..	..	..	..	..
37	..	..	1	..	..	..
39	..	..	..	1	..	..
42	..	..	..	..	1	..
51	1	..	..	..	..	..
55	..	..	..	..	..	1

Deaths within 1 week of admission.			
Day.	M.	F.	Total.
1	38	18	56
2	13	12	25
3	9	4	13
4	9	10	19
5	5	5	10
6	9	5	14
7	10	5	15
	93	59	152
	152		

DEATHS.

The total number of *Deaths* during the year was 404

Of these there occurred within 24 hours of admission .. .. 56 = 14 per cent.

Within one week of admission .. 152 = 38 „

„ a fortnight „ .. 210 = 52 „

„ three weeks „ .. 267 = 67 „

„ a month „ .. 309 = 77 „

The average time of fatal cases in Hospital was in days .. .. Males, 25·5; Females, 27·3 } 26·3

DISCHARGES.

The total number of *Discharges* during the year was 1,918

Of these, the number discharged within 24 hours was .. .. } 19 = 1 per cent.

Within 1 week .. .. 195 = 10 „

„ 2 weeks .. .. 493 = 25 „

„ 3 „ .. .. 777 = 40 „

„ 4 „ .. .. 1,048 = 54 „

„ 5 „ .. .. 1,263 = 65 „

„ 6 „ .. .. 1,431 = 74 „

The average stay in Hospital of patients discharged was in days .. .. Males, 31·36; Females, 31·55 } 31·47

The average time of all the medical cases in the Hospital was in days .. .. Males, 30·81; Females, 30·49 } 30·78

PERNOCTATIONS.

There are 230 beds in the medical wards, giving the number of possible pernoctations for the year, 230 × 365 = .. 83,950

The actual number of pernoctations has been as follows:—

Of cases admitted in 1875 or 1876 and discharged in 1876	60,367	
Of cases remaining in at the end of the year 1875	5,813	
Of fatal cases	10,479	
		76,659
Giving a difference of		7,291

This gives a nightly average of about 20 empty beds, and the time between successive occupants about 70 hours on an average.

SURGICAL REPORT.

TABLE I,

Showing the total Number of Cases of each Disease under Treatment during the year 1876, *with the Results.*

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.										
Phagedæna	3	3	2	2	..	..	..	..	1	1
Erysipelas—										
a. Simple	19	13	16	12	..	..	1	..	2	1
b. Phlegmonous	29	9	24	7	..	..	4	1	1	1
c. Diffuse Inflammation ..	4	1	3	1	..	..	1	..	..	..
Pyæmia	3	3	1	1	..	..	2	2	..	..
Syphilis —										
A. Primary Syphilis—										
Hard Chancre	2	3	2	3	..	..	..	..	..	..
Soft Chancre	6	5	6	4	..	..	..	..	..	1
Phagedænic Sore	13	..	13	..	..	..	..	..	..	..
B. Hereditary Syphilis	2	2	1	1	1	..	..	..	..	1
C. Secondary Syphilis—										
Local Syphilitic Affections—										
Tongue	2	1	2	..	..	..	..	..	..	1
Palate and Pharynx ..	..	4	..	4	..	..	..	..	..	..
Larynx	2	4	1	4	..	..	1	..	..	..
Rectum	..	2	..	2	..	..	..	..	..	..
Testis	1	..	1	..	..	..	..	..	..	..
Bone	2	8	2	6	..	..	..	1	..	1
Skin	28	38	28	36	..	..	..	..	..	2
Eye	3	..	3	..	..	..	..	..	..	..
Cancer—										
A. Scirrhus—										
Rectum	3	3	..	..	1	..	2	3	..	..
Female Breast	..	20	..	14	..	3	..	..	..	3
Lymphatic Glands ..	..	2	..	1	..	1	..	..	..	..
Œsophagus	4	..	2	..	1	..	1	..	..	..
Penis	1	..	1	..	..	..	..	..	..	..
Bladder	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 1876.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
GENERAL DISEASES (*continued*).										
B. Medullary Cancer—										
Female Breast	..	2	..	2	..	..	..	..	..	..
Jaw	1	1	..	1	1	..	..	..	..	..
Glands	1	..	..	..	..	..	1	..	..	..
Skull	1	..	..	..	..	..	1	..	..	..
Nose	1	..	..	..	1	..	..	..	..	..
Bladder	4	..	..	..	3	..	1	..	..	..
Forearm	1	..	..	..	1	..	..	..	..	..
C. Epithelial Cancer—										
Lip	11	..	9	..	..	..	..	..	2	..
Tongue	12	5	6	3	5	1	1	1	..	..
Mouth	1	..	1	..	..	..	..	..	..	..
Pharynx	1	..	..	..	..	..	1	..	..	..
Face	3	..	..	..	2	..	..	..	1	..
Eyelid	3	1	2	1	1	..	..	..	..	..
Ear	1	..	1	..	..	..	..	..	..	..
Scrotum	3	..	3	..	..	..	..	..	..	..
Penis	1	..	1	..	..	..	..	..	..	..
Anus	1	..	1	..	..	..	..	..	..	..
Thigh	1	..	1	..	..	..	..	..	..	..
Leg	1	..	1	..	..	..	..	..	..	..
Glands	1	..	..	..	1	..	..	..	..	..
Lupus	1	1	1	1	..	..	..	..	..	..
Rodent Ulcer	1	1	1	..	..	..	..	..	..	1
Scrofula										
Lymphatic Glands ..	3	2	3	1	..	..	..	..	..	1
Testicle	2	..	..	..	1	..	..	..	1	..
Joints	32	25	24	15	1	1	..	2	7	7
Skin	2	1	2	..	..	..	..	1	..	..
Vaccination	1	..	..	..	..	..	1	..	..	..
Chronic Rheumatic Arthritis ..	5	3	5	3	..	..	..	..	..	..
DISEASES OF THE NERVOUS SYSTEM.										
Malingering	..	1	..	1	..	..	..	..	..	..
Neuralgia	..	5	..	5	..	..	..	..	..	..
Hysteria	..	13	..	11	..	2	..	..	..	..
Tetanus, Traumatic	2	..	..	..	..	..	1	..	1	..
Paralysis of Superior Maxillary ..	..	1	..	..	..	1	..	..	..	..
DISEASES OF THE EYE.										
A. Conjunctiva—										
Catarrhal Ophthalmia ..	5	3	5	3	..	..	..	..	..	..
Purulent „ ..	3	2	3	2	..	..	..	..	..	..
Diphtheritic „ ..	..	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.
DISEASES OF THE EYE (*continued*).										
Urticaria of Conjunctiva ..	1	..	1	..	..	..	..	..	..	..
Pterygium	1	..	1	..	..	..	..	..	..	..
B. Cornea—										
Keratitis	8	18	7	16	..	..	..	..	1	2
Do. Interstitial ..	8	2	8	2	..	..	..	..	..	..
Hypopyon	2	..	2	..	..	..	..	..	..	..
Ulcers	7	5	7	4	..	..	..	..	..	1
Opacity	7	7	5	6	1	..	..	..	1	1
Staphyloma	2	3	1	1	1	2	..	..	..	..
C. Iris—										
Iritis	5	7	5	7	..	..	..	..	..	..
Rheumatic Iritis	2	2	1	2	..	..	..	..	1	..
Irido-choroiditis	4	1	3	1	1	..	..	..	..	..
Synechia	5	7	4	7	1	..	..	..	..	..
Syphilitic Iritis	3	2	2	2	..	..	..	..	1	..
Occlusion of Pupil.. ..	3	2	1	2	2	..	..	..	..	..
D. Crystalline Lens—										
Cataract—										
Hard	17	19	14	18	2	1	..	..	1	..
Soft	13	9	11	9	..	..	..	..	2	..
Traumatic	7	6	6	6	1	..	..	..	..	..
Dislocation of Lens ..	1	1	1	1	..	..	..	..	..	..
Opacities of Capsule ..	5	3	5	3	..	..	..	..	..	..
E. Diseases of Retina and Optic Nerve—										
Detached Retina	1	1	1	..	..	1	..	..	..	..
Retinitis Albuminurica ..	..	1	..	..	..	..	..	1	..	..
Optic Neuritis	1	2	1	..	..	2	..	..	..	..
Retino-Choroiditis ..	1	2	1	1	..	1	..	..	..	..
White Atrophy of Optic Discs	4	..	4	..	..	..	..	..	..	..
F. Diseases of the Choroid—										
Atrophy	2	..	..	..	2	..	..	..	..	..
G. Hemiopia	2	..	1	..	1	..	..	..	..	..
H. Diseases of Vitreous—										
Hyalitis	1	..	1	..	..	..	..	..	..	..
Opacities	1	..	..	..	1	..	..	..	..	..
I. General Affections of the Eye—										
Glaucoma	2	14	2	13	..	1	..	..	..	..
Sympathetic Ophthalmia ..	7	2	6	2	..	..	..	..	1	..
Total Disorganization of Eye	1	3	1	3	..	..	..	..	..	..
Melanotic Sarcoma ..	..	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.
Diseases of the Eye (*continued*).										
J. Strabismus—										
Internal	9	12.	9	12	..	..	..	..	..	..
External	3	2	2	2	..	..	..	..	1	..
K. Hypermetropia and Asthenopia	2	3	1	2	1	1	..	..	..	..
Myopia	2	5	2	3	..	1	..	..	..	1
L. Diseases of the Lachrymal Apparatus—										
Lachrymal Obstruction	1	4	1	4	..	..	..	..	..	..
Abscess and Fistula	3	3	2	3	1	..	..	..	..	..
Dacryo-Cystitis	1	3	1	3	..	..	..	..	..	..
M. Diseases of the Eyelids—										
Entropion	..	3	..	3	..	..	..	..	..	..
Symblepharon	2	..	2	..	..	..	..	..	..	..
Granular Lids	..	4	..	4	..	..	..	..	..	..
N. Diseases of Orbit—										
Sarcoma	2	..	1	..	1	..	..	..	..	..
Melanotic Tumour	..	1	..	..	..	..	..	1	..	..
O. Diseases of Cranial Nerves	..	3	..	3	..	..	..	..	..	..
Diseases of the Ear.										
Otorrhœa	3	1	2	1	..	..	1	..	..	..
Polypus	1	..	1	..	..	..	..	..	..	..
Diseases of the Nose.										
Abscess of Septum	1	..	1	..	..	..	..	..	..	..
Polypus	3	2	3	2	..	..	..	..	..	..
Epistaxis	12	3	12	3	..	..	..	..	..	..
Ozœna	1	2	..	2	..	..	..	..	1	..
Malignant Polypus, Sarcoma	..	1	..	..	..	1	..	..	..	..
Diseases of the Circulatory and Absorbent Systems.										
Aneurism—										
Subclavian and Axillary Artery	3	..	..	..	1	..	2	..	..	..
Popliteal Artery	7	..	6	..	..	..	..	..	1	..
Dorsalis Pedis Artery	1	..	1	..	..	..	..	..	..	..
Aneurismal Varix	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.
Diseases of the Circulatory and Absorbent Systems (*continued*).										
Varicose Veins	3	2	3	2	..	..	..	..	..	..
Thrombosis	6	7	6	7	..	..	..	..	..	..
Secondary Hæmorrhage	..	1	..	1	..	..	..	..	..	..
Lymphatic Glands—										
Abscess	6	6	5	4	..	..	..	1	1	1
Enlarged Glands	3	2	3	..	..	2	..	..	..	..
Lymphangitis	2	1	2	1	..	..	..	..	..	..
Diseases of the Lips.										
Malformations—										
Single Harelip	6	3	5	3	..	..	..	..	1	..
Double „	1	..	1	..	..	..	..	..	..	..
Diseases of Mouth and Cheek.										
Stomatitis	3	1	3	1	..	..	..	..	..	..
Ranula	1	..	1	..	..	..	..	..	..	..
Diseases of Gums and Jaws.										
Epulis	5	6	4	6	1	..	..	..	..	..
Sarcoma	3	..	2	..	..	..	..	..	1	..
Gum boil	..	1	..	1	..	..	..	..	..	..
Hæmorrhage after extraction of a tooth	..	2	..	2		..	..	..	..	..
Diseases of Palate and Fauces.										
Enlarged Thyroid	..	1	..	..	..	..	..	1	..	..
„ Tonsils	2	4	2	4	..	..	..	..	..	..
Tonsillitis	..	22	..	22	..	..	..	..	..	..
Cleft Palate	6	12	5	10	1	2	..	..	..	..
Abscess	..	2	..	2	..	..	..	..	..	..
Perforation of Palate	2	..	2	..	..	..	..	..	..	..
Diseases of the Tongue.										
Ulcer	..	1	..	1	..	..	..	..	..	..
Papilloma	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 1876.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
Diseases of Salivary Glands.										
Parotid Glandular Tumour ..	3	..	3	..	..	..	..	..	..	..
Enchondroma	1	..	1	..	..	..	..	..	..	..
Diseases of Œsophagus and Larynx.										
Laryngitis	2	1	2	1	..	..	..	..	..	..
Diseases of the Intestines.										
Hernia—										
Umbilical	..	6	..	6	..	..	..	..	..	..
Inguinal	36	2	33	2	..	..	2	..	1	..
Femoral	10	15	6	13	..	..	3	2	1	..
Fæcal Fistula	1	..	1	..	..	..	..	..	..	..
Abscess of Abdomen	..	1	..	1	..	..	..	..	..	..
Diseases of Rectum and Anus.										
Fistula in Ano	9	12	7	9	..	..	..	..	2	3
Hæmorrhoids	9	6	8	5	..	1	..	..	1	..
Fissure of Anus	..	4	..	4	..	..	..	..	..	..
Fibrous Stricture	1	3	1	3	..	..	..	..	..	..
Prolapsus Ani	2	1	1	1	1	..	..	..	..	..
Polypus of Rectum	..	1	..	1	..	..	..	..	..	..
Ischio-Rectal Abscess	3	4	3	4	..	..	..	..	..	..
Recto-Vesical Fistula	2	..	1	..	1	..	..	..	..	..
Diseases of Urinary System.										
Malformation—										
Ectopia Vesicæ	2	..	..	..	1	..	1	..	..	..
Cystitis—										
Acute	1	..	1	..	..	..	..	..	..	..
Chronic	6	..	2	..	..	..	1	..	3	..
Renal Calculus	2	1	1	1	1	..	..	..	..	..
Calculus Vesicæ—										
a. Uric Acid	7	1	3	1	4	..	..	..	..	..
b. Oxalate of Lime	4	..	4	..	..	..	..	..	..	..
c. Phosphatic	3	..	1	..	..	..	1	..	1	..
Urethral Calculus	1	..	1	..	..	..	..	..	..	..
Irritable Bladder	2	..	2	..	..	..	..	..	..	..
Tubercular Disease of Urinary Tract	1	2	..	..	1	2	..	..	..	..
Elastic Tube in Bladder	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.
Diseases of Urinary System (*continued*).										
Diseases of Prostate Gland.										
Enlarged Prostate	13	..	8	..	2	..	2	..	1	..
Gonorrhœa and its Complications.										
Gonorrhœa	14	36	14	36	..	..	..	..	..	..
Paraphimosis	2	..	2	..	..	..	..	..	..	..
Bubo	5	3	5	3	..	..	..	..	..	..
Verrucæ	1	1	1	1	..	..	..	..	..	..
Perineal Abscess	3	..	2	..	..	..	..	..	1	..
Orchitis	2	..	2	..	..	..	..	..	..	..
Diseases of Urethra.										
Urethritis	1	..	1	..	..	..	..	..	..	..
Stricture—										
a. Organic	54	..	45	..	2	..	4	..	3	..
b. Traumatic	5	..	3	..	2	..	..	..	..	..
c. Spasmodic	1	..	1	..	..	..	..	..	..	..
Urinary Fistula	5	..	3	..	..	..	..	..	2	..
Extravasation of Urine	3	..	2	..	..	..	1	..	..	..
Incontinence of Urine	..	1	..	..	..	1	..	..	..	..
Retention of Urine	11	..	10	..	..	..	..	..	1	..
Urethral Abscess	1	..	1	..	..	..	..	..	..	..
Diseases of the Penis and Testis.										
Malformation—										
Phimosis	27	..	27	..	..	..	..	..	..	..
Hypospadias	4	..	3	..	..	..	..	..	1	..
Paraphimosis	5	..	4	..	..	..	..	..	1	..
Hæmatocele	5	..	5	..	..	..	..	..	..	..
Hæmatocele of Cord	1	..	1	..	..	..	..	..	..	..
Hydrocele	16	..	14	..	1	..	..	..	1	..
Encysted Hydrocele	1	..	1	..	..	..	..	..	..	..
,, ,, of Cord ..	4	..	4	..	..	..	..	..	..	..
Orchitis	5	..	5	..	..	..	..	..	..	..
Sarcoma Testis	1	..	..	..	1	..	..	..	..	..
Varicocele	5	..	5	..	..	..	..	..	..	..
Cyst of Cord	1	..	1	..	..	..	..	..	..	..
Inflamed Scrotum	2	..	1	..	..	..	..	..	1	..
Inflamed Prepuce	2	..	2	..	..	..	..	..	..	..

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.
DISEASES OF FEMALE ORGANS OF GENERATION.										
Diseases of the Ovary	..	10	..	5	..	2	..	3	..	..
Diseases of Round Ligament.										
Cyst in Canal of Nuck ..	..	1	..	..	..	..	..	..	..	1
Diseases of the Vagina.										
Vesico-Vaginal Fistula ..	..	4	..	3	..	1	..	..	..	..
Recto-Vaginal Fistula ..	..	1	..	..	..	..	..	..	..	1
Diseases of Vulva.										
Adhesion of Labia.. ..	..	1	..	1	..	..	..	..	..	..
Cystic Tumour of Labium .	..	1	..	1	..	..	..	..	..	..
Noma	..	4	..	2	..	1	..	1	..	..
Vascular Tumour of Urethra	..	1	..	1	..	..	..	..	..	..
Abscess of Labium ..	..	3	..	3	..	..	..	..	..	..
Affections connected with Parturition.										
Ruptured Perinæum ..	..	5	..	3	..	1	..	..	..	1
DISEASES OF THE FEMALE BREAST.										
Hypertrophy	..	1	..	..	..	1	..	..	..	..
Abscess	..	19	..	17	..	1	..	..	..	1
Indurated Breast	..	3	..	3	..	..	..	..	..	..
Non-Malignant Tumours—										
Sero-Cystic Sarcoma ..	..	5	..	4	..	..	..	..	..	1
Mammary Glandular ..	..	5	..	5	..	..	..	..	..	..
Cysts	..	1	..	1	..	..	..	..	..	..
DISEASES OF THE ORGANS OF LOCOMOTION.										
Diseases of Bones.										
Ostitis	2	1	2	1	..	..	..	..	..	..
Periostitis	13	7	12	7	..	..	..	..	1	..
Chronic Abscess	1	..	1	..	..	..	..	..	..	..
Diffuse Periostitis—										
Acute Necrosis	1	..	..	..	..	..	..	..	1	..
Caries	25	5	18	3	1	1	1	..	5	1
Necrosis	44	19	35	14	2	1	..	1	7	3

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.
Diseases of the Organs of Locomotion (*continued*).										
Diseases of Bones (continued).										
Perforating Ulcer	2	2	2	2	..	..	..	..	..	..
Tumours—										
a. Exostosis	3	3	3	1	..	2	..	..	..	..
b. Sarcoma	9	6	1	3	7	2	1	1	..	..
Old Amputations	2	..	2	..	..	..	..	..	..	..
Deformities after Fracture, &c. ..	1	1	..	1	1	..	..	..	..	..
Diseases of Joints.										
Acute Synovitis	23	8	19	6	..	..	..	..	4	2
Chronic do.	17	16	13	13	..	..	..	..	4	3
Chronic Disease	29	23	17	19	..	..	4	1	8	3
Ankylosis (Fibrous) ..	9	7	6	3	3	2	..	..	..	2
Loose Cartilage	3	..	3	..	..	..	..	..	..	..
Knock-Knee	12	..	9	..	..	..	..	..	3	..
Acute Arthritis of Infants .	1	..	1	..	..	..	..	..	..	..
Disease of the Sacro-Iliac—										
Synchondrosis ..	..	3	..	1	..	..	..	2	..	..
Diseases of the Spine.										
Caries	9	8	3	3	3	3	1	1	2	1
Psoas, Lumbar, and other Abscesses	8	8	5	5	1	1	..	..	2	2
Angular Curvature ..	16	5	6	5	4	..	1	..	5	..
Lateral Curvature.. ..	1	3	1	3	..	..	..	..	..	..
Spina Bifida	2	1	1	1	..	..	1	..	..	..
Diseases of Muscles, Tendons, &c.										
Malformation of Leg	1	..	..	..	1	..	..	..	..	..
Contraction of Tendons, Fasciæ, or Muscles	2	6	2	5	..	..	..	1	..	..
Club-Foot—										
a. Talipes Equinus	8	8	7	7	..	1	..	..	1	..
b. Do. Valgus	8	3	7	2	..	..	..	..	1	1
c. Do. Equino-varus.. ..	24	11	20	7	..	1	..	..	4	3
d. Do. Varus	3	2	2	2	..	..	..	..	1	..
Supernumerary Toe..	..	1	..	1	..	..	..	..	..	..
Distortion of Toes	2	..	1	..	..	..	..	..	1	..
Wry-neck	..	1	..	1	..	..	..	..	..	..
Enlarged Bursa Patellæ	4	4	4	3	..	1	..	..	..	..
Inflammation and Suppuration of Bursa Patellæ	8	23	7	22	..	..	..	..	1	1
Do. do. of other Bursæ ..	1	..	1	..	..	..	..	..	..	..
Diffuse Palmar Ganglion	1	1	1	1	..	..	..	..	..	..
Bursal Tumour	1	3	1	3	..	..	..	..	..	..

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.
Diseases of the Cellular Tissue.										
Abscess	84	70	77	66	..	..	2	2	5	2
Connective Tissue Tumours—										
a. Fatty	5	18	5	14	..	2	..	..	..	2
b. Fibro-cellular	1	2	..	2	..	..	1	..	..	..
c. Sarcoma	5	3	3	2	1	1	1	..	..	..
d. Fibrous	7	4	5	3	1	..	..	..	1	1
e. Painful Subcutaneous Tumours	1	3	1	3	..	..	..	..	..	..
Sebaceous Cysts	8	8	8	8	..	..	..	..	..	..
Dermoid Cysts	1	..	1	..	..	..	..	..	..	..
Elephantiasis	..	1	..	..	..	1	..	..	..	..
Hygroma	2	1	2	1	..	..	..	..	..	..
Diseases of the Cutaneous System.										
Ulcer	33	26	30	22	1	1	1	1	1	2
Carbuncle	8	2	8	2	..	..	..	..	..	..
Boil	2	1	2	1	..	..	..	..	..	..
Gangrene	4	1	3	1	..	..	..	..	1	..
Nævus	5	6	5	6	..	..	..	..	..	..
In-growing Toe-nail	6	6	6	5	..	..	..	..	..	1
Cicatrix	..	5	..	4	..	..	..	..	..	1
Papilloma	..	1	..	1	..	..	..	..	..	..
Corn	1	1	1	1	..	..	..	..	..	..
Mole	2	2	2	1	..	..	..	..	..	1
Onychia	2	2	2	2	..	..	..	..	..	..
General Injuries.										
Burns and Scalds	33	29	25	16	..	..	3	11	5	2
Contusions	18	6	16	6	..	..	1	..	1	..
Railway Shock	2	..	2	..	..	..	..	..	..	..
Punctured Wounds	1	..	1	..	..	..	..	..	..	..
Small Shot Wounds	1	..	1	..	..	..	..	..	..	..
Local Injuries.										
Injuries of the Head—										
Contusion	6	1	6	1	..	..	..	..	..	..
Scalp Wound	29	8	28	8	..	..	1	..	..	..
Concussion of Brain	38	9	35	7	..	..	2	1	1	1
Fracture of Vault of Skull—										
Simple	1	2	1	..	..	..	2	..	..	..
Compound	10	3	5	1	..	..	5	1	..	1
Fracture of Base of Skull	15	..	2	..	..	..	12	..	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.
LOCAL INJURIES (*continued*).										
Of the Ear.										
Wound	1	..	1	..	..	..	..	..	..	..
Bead in Ear	1	..	1	..	..	..	..	..	..	..
Of the Face.										
Contusion	1	2	1	2	..	..	..	..	..	..
Wound	14	3	14	3	..	..	..	..	..	..
Fracture of Facial Bones ..	1	1	1	..	..	..	..	..	..	1
Do. Lower Jaw ..	7	1	5	1	..	..	..	..	2	..
Injuries of the Eye.										
Foreign Body in Eye ..	3	..	3	..	..	..	..	..	..	..
Burns	2	1	2	1	..	..	..	..	..	..
Wound of Eye	11	3	10	3	..	..	..	..	1	..
Contusions of Globe ..	7	1	7	1	..	..	..	..	..	..
Injuries of the Neck.										
Scald of Throat	1	..	..	..	..	..	1	..	..	..
Contusion	2	..	2	..	..	..	..	..	..	..
Cut Throat	3	1	3	..	..	..	..	..	..	1
Foreign Body in Œsophagus	..	1	..	1	..	..	..	..	..	..
Do. in Bronchus..	1	..	1	..	..	..	..	..	..	..
Wound of Pharynx ..	1	..	1	..	..	..	..	..	..	..
Wounds	..	2	..	1	..	..	..	..	..	1
Injuries of the Chest.										
Contusions	10	..	8	..	..	..	2	..	..	..
Fractured Ribs and Sternum, Lung not Wounded	15	2	9	1	..	..	4	1	2	..
Do. Lung Wounded ..	4	2	1	1	..	..	3	1	..	..
Incised Wound (Stab) ..	2	..	1	..	..	..	1	..	..	..
Injuries of the Back.										
Contusion	3	..	3	..	..	..	..	..	..	..
Contusion with Hæmaturia	..	1	..	1	..	..	..	..	..	..
Fracture of the Spine ..	4	..	..	..	..	..	2	..	2	..
Injury to Spine, without Fracture	2	..	1	..	..	..	1	..	..	..
Incised Wound	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.
LOCAL INJURIES (*continued*).										
Injuries of the Abdomen.										
Contusion	5	4	5	4	..	..	..	..	..	..
Do. with Rupture of Viscera	3	..	..	..	..	..	3	..	..	..
Wound	1	1	1	1	..	..	..	..	..	..
Do. with Protrusion of Viscera	1	..	1	..	..	..	..	..	..	..
Injuries of the Pelvis.										
Contusion	3	3	3	3	..	..	..	..	..	..
Wound of Vulva	..	1	..	1	..	..	..	..	..	..
Ruptured Urethra.. ..	5	..	5	..	..	..	..	..	..	..
Fracture of Pelvis	3	..	3	..	..	..	..	..	..	..
Do. do. with Injury to Viscera	1	..	..	..	..	..	1	..	..	..
Injuries of the Upper Extremity.										
Contusion	2	1	2	1	..	..	..	..	..	..
Sprains	1	1	1	1	..	..	..	..	..	..
Wound—										
Of Arm	3	..	3	..	..	..	..	..	..	..
Of Forearm	11	3	9	3	..	..	1	..	1	..
Of Hand	22	2	15	1	..	..	2	..	5	1
Injuries of Vessels	1	1	1	1	..	..	..	..	..	..
Gunshot Wound of Forearm	1	..	1	..	..	..	..	..	..	..
Fracture of—										
Clavicle	7	4	6	4	..	..	..	..	1	..
Humerus—										
Simple	6	8	6	7	..	..	..	1	..	..
Compound	2	..	2	..	..	..	..	..	..	..
Forearm—										
Simple	3	3	2	3	..	..	1	..	..	..
Compound	2	..	2	..	..	..	..	..	..	..
Bones of Hand—										
Compound	3	..	2	..	..	..	1	..	..	..
Dislocation of—										
Clavicle	..	..	..	..	..	..	..	..	..	..
Humerus	7	1	6	1	1	..	..	..	..	..
Co. Dislocation of Humerus	1	..	1	..	..	..	..	..	..	..
Radius and Ulna	1	..	1	..	..	..	..	..	..	..
Foreign Body in Hand	..	1	..	1	..	..	..	..	..	..
Co. Dislocation of Radius and Ulna	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.
LOCAL INJURIES (*continued*).										
Injuries of Lower Extremities.										
Contusions	20	13	20	12	..	..	..	1	..	..
Sprained Ankle	6	7	6	7	..	..	..	..	..	..
Do. Knee	2	..	2	..	..	..	..	..	..	..
Do. Hip	2	..	2	..	..	..	..	..	..	..
Wounds—										
Of Thigh	7	..	6	..	..	..	1	..	..	..
Over Knee	4	4	4	3	..	..	..	..	..	1
Of Leg	12	2	9	1	..	..	1	..	2	1
Of Foot	7	..	6	..	..	..	..	..	1	..
Of Knee Joint	3	..	2	..	..	..	1	..	..	..
Of Buttock	..	1	..	1	..	..	..	..	..	..
Fracture of Femur—										
Simple	48	20	42	18	..	..	1	..	5	2
Compound	2	..	..	..	..	..	1	..	1	..
Greenstick	1	..	1	..	..	..	..	..	..	..
Fracture of Cervix Femoris—										
Intracapsular	4	6	3	5	..	..	..	1	1	..
Extracapsular	..	4	..	3	..	..	..	1	..	..
Ununited	1	..	1	..	..	..	..	..	..	..
Fracture of Patella	21	10	18	10	..	..	..	..	3	..
Fracture of both Bones of the Leg—										
Simple	85	44	79	36	..	..	..	..	6	8
Compound	13	3	11	3	..	..	1	..	1	..
Ununited	1	..	1	..	..	..	..	..	..	..
Fracture of Tibia alone—										
Simple	41	11	37	8	..	..	..	..	4	3
Compound	2	1	2	1	..	..	..	..	..	..
Greenstick	1	..	1	..	..	..	..	..	..	..
Ununited	..	1	..	1	..	..	..	..	..	..
Fracture of Fibula alone—										
Simple	20	7	15	7	..	..	..	..	5	..
Fracture of Bones of Foot—										
Simple	4	..	4	..	..	..	..	..	..	..
Compound	1	1	1	1	..	..	..	..	..	..
"Pott's Fracture" of Leg	12	..	9	..	..	..	1	..	2	..
Dislocations of—										
Hip	..	1	..	1	..	..	..	..	..	..
Leg	1	..	1	..	..	..	..	..	..	..
Foot	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.
LOCAL INJURIES (*continued*).										
Dislocations of (*continued*).										
Co. of Foot..	1	..	..	..	..	..	..	..	1	..
Semi-lunar Cartilage ..	1	..	1	..	..	..	..	..	..	..
Diseases and Injuries not classified.										
Rupture of Muscle and Tendon	1	..	1	..	..	..	..	..	..	..
Unclassified	2	1	2	1	..	..	..	..	..	..

ABSTRACT OF TABLE I,

With Average Duration of Surgical Patients in the Hospital.

Discharged Cured or Relieved	M. = 1568	
	F. = 1007	
Discharged Unrelieved	M. = 83	
	F. = 56	
Died	M. = 109	
	F. = 50	
Remaining in at the end of the year 1876	M. = 164	
	F. = 90	
Average stay in the Hospital	M. = 31·71 days.	
	F. = 24·88 „	

Average stay in the Hospital of all Surgical Patients = 29·08 days.

APPENDIX TO TABLE I.

General Diseases:

Cancer.—A man, aged 45, died of hard cancer of the bladder. The cavity of the organ was so much contracted that it could hold no more than two ounces. Its mucous membrane was sloughy, and here and there encrusted with phosphates. Its walls were much thickened and infiltrated with hard cancer, which had involved the surrounding parts, and destroyed the body of the right pubic bone. No variation in the size of the bladder was possible, on account of the hardness of the diseased wall.

Secondary deposits of scirrhus were found in the glands, the liver, and peritoneum.

For the last year of his life the patient had been passing blood with his water, and had suffered pain in the back, with occasional pain at the end of the penis. He was admitted three weeks before his death with total incontinence of 14 days' duration. His urine contained bright blood and blood clots, that were usually passed at the beginning of micturition. (This case will appear in the Transactions of the Pathological Society for next year.)

Œsophagotomy was attempted on a man aged 41, who was suffering from epithelial cancer of the pharynx, causing stricture.

After the tube was exposed, the disease was found to extend so far down as not to warrant the completion of the operation. The wound healed readily. Many weeks afterwards the man died of pyæmia.

Vaccination.—An infant, aged 4 months, was vaccinated on the 22nd February by five punctures on the right arm. Nine days afterwards the pustules had run together and formed one.

Admitted March 23, with a large sore on the right arm, and a pustular eruption over the arms, trunk, and thighs.

On the outer side of the right arm was a large granulating sore, reaching from the shoulder nearly to the elbow, and extending over a part of the back and front of the arm. The base was generally even, the margin sinuous, and formed by a pustular rim.

The rash consisted of pustules, exactly resembling those produced by vaccination, and existing at different parts in different stages of development. It had probably spread from the point of inoculation. The trunk was covered with fully formed pustules, but near the ulcer were blackened scabs and scaling red spots, from which scabs had separated, while at the limit of the eruption on the thighs, were small vesicles or pustules with imperfect areolæ.

At the beginning of April, when the rash had almost died out, numerous red cutaneous nodules, like blind boils, began to appear over the trunk. Some of them eventually softened, suppurated, and cicatrised.

The ulcer on the arm diminished in size, but retained the same character throughout.

On the 26th April he died, a large abscess having formed over the left side of the chest and lower part of the neck.

No signs of syphilis were detected in the child from whom this patient was vaccinated, nor in any member of that child's family, nor has syphilis developed in him since.

Diseases of the Circulatory System:

Aneurism.—In a case of subclavian aneurism, after several plans of treatment had been tried and had failed, amputation of the arm at the shoulder-

joint was performed. At the end of five weeks, death occurred from Pleurisy and Bronchitis. This case will be published in full in St. Bartholomew's Hospital Reports for next year.

Of six cases of popliteal aneurism, all males, five were cured by Digital Compression.

In the sixth case pressure could not be endured. The patient, aged 43, had suffered pain in the ham for two months, and had noticed a swelling for one week. An aneurism of the size of the fist occupied the right ham.

On the 11th March the femoral artery was tied with stout catgut under carbolised spray near the apex of Scarpa's triangle and antiseptic dressings were applied. Nine days afterwards pulsation recurred. On 27th March the patient left the Hospital, rejecting the advice offered him, with the aneurism as large as a hen's egg, pulsating feebly but expansively.

During the summer he walked about with a stick, wearing an elastic stocking, and pulsation was said to have ceased. In August the aneurism began to increase, and about the same time a second swelling was noticed in the groin.

On 21st October he was re-admitted with the popliteal aneurism larger than ever, and with another of the size of a walnut beneath the upper part of the scar in Scarpa's triangle.

On 31st October the femoral artery was tied between the two aneurisms, at a distance of 2 or 3 inches from the upper one. Antiseptic dressings and carbolised spray were used, but the ligature was of thick silk. On 2nd December the patient was discharged, cured of the aneurism in the ham, and with a lessened but still perceptible pulse in the traumatic aneurism.

Aneurismal Varix.—A boy, aged 14, stabbed his left thigh near the middle of the inner aspect with a penknife. The skin wound readily healed.

The symptoms of injury to the great vessels were—on palpation—a thrill felt over the front of the thigh about the course of the femoral artery, best marked at the site of the wound—on auscultation—a murmur heard from Poupart's ligament to the knee, over the front and inner side of the thigh. The murmur was made up of two parts—a continuous hum and a high-pitched systolic bruit, and was silenced by pressure on the femoral artery above the scar. No venous enlargement visible over the limb. Pain in the ham on straightening the knee.

He was kept at rest with the limb evenly bandaged, and at the end of the month he left the Hospital with free use of the leg and some modification of the physical signs. (See Hospital Reports, Vol. XII., page 157, for full account.)

Diseases of the Intestine:

In a woman with femoral hernia, on whom herniotomy was performed, two sacs were found. The incision opened a large sac filled with straw-coloured fluid, into which projected a second sac containing a knuckle of intestine.

After death the walls of both sacs were seen to be continuous with the parietal peritoneum, but the outer sac did not communicate with the cavity of the abdomen, being firmly adherent at its neck to the small hernia that it contained.

Diseases of the Urinary System:

Renal Calculus.—One of the cases was that of a woman, aged 40, admitted in March, 1875, with symptoms of stone in the kidney and an abscess pointing in the left groin. After the abscess was incised, the wound slowly healed, leaving a small fistula. In the summer of that year she left the Hospital, but returned in January, 1876, complaining that attacks of pain in the lumbar region had lately become frequent and severe. The fistula was still open.

On 29th January a stone, consisting of Uric acid, and measuring 1½ inches long and ½ inch broad, was extracted from the pelvis of the left kidney, through an oblique incision in the loin, between the 12th rib and iliac crest. She made a good recovery.

Foreign Body.—A man, aged 34, stated that he had had symptoms of stone in the bladder for four months. He denied that an instrument had ever been passed down the urethra.

As he was thought to have a soft calculus, Lithotrity was performed on 20th May, and again on several occasions. Much phosphatic debris escaped after each crushing. On 21st June he died with inflammation of the urinary tract.

Three pieces of indiarubber tubing, each 2½ inches long, of the size of a No. 10 catheter, were found in the bladder, thickly encrusted with phosphates.

Diseases of the Ovary:

Of the ten cases, one was discharged as unfit for operation. Three cases were relieved by Paracentesis. Ovariotomy was done three times with one fatal result.

An incomplete operation was performed in two cases. In one the Cyst was Parovarian, and lay behind the Peritoneum, by which it was covered only in front and at the sides. Cyst drained. In the second case the operation was abandoned on account of the dense adhesions in the Pelvis.

An exploratory incision was made in a case of Peritoneal Papillomata.

Diseases of Bone:

Caries.—A labourer, aged 21, was admitted in January with caries of the os calcis. The disease dated from February, 1875. Since May of that year there had been constant suppuration from the fistulæ leading to the disease.

On admission he was passing about three pints of urine daily of S.G. 1015, containing albumen, nearly ¼, with hyaline casts.

January 26th—Syme's amputation.

The quantity of urine began steadily to increase after the operation, and the S.G. to diminish, so that on 7th February he passed as much as 10¾ pints, S.G. 1,005, and about ⅓ albumen. The wound had been doing well.

On 12th February diarrhœa with sickness set in, and the daily amount of urine became gradually less. On 6th March he passed only ½ a pint, S.G. 1012, albumen ⅙, and after that day there was total suppression till March 11th, when he died.

Diseases of Joints:

In three cases the knee was opened and washed out.

In a girl, aged 18, with Chronic Disease of the synovial membrane. Suppuration having occurred within and around the joint, it was freely incised on each side and washed out with a strong solution of Carbolic acid. Suppuration still continued, and after several months she died of Phthisis Pulmonalis.

In a boy, aged 14, with Chronic Suppurative Disease, destroying the joint. On 3rd March the knee was incised and washed out under Carbolic spray, and antiseptic dressings applied. Suppuration in and about the knee followed. At the end of the year the fistulæ were still open, but the joint was firmly ankylosed.

In a man, aged 48, with Chronic Rheumatic Arthritis. The knee was treated as in the last case. Antiseptic dressings. Suppuration in and about the joint followed the operation. In six months' time he left the Hospital with the knee firmly ankylosed, and the openings discharging slightly.

Diseases of the Spine:

A Spina Bifida in a child, 5 weeks old, was treated by injection. The tumour was seated at the upper part of the lumbar spine, was the size of an orange, with a narrow base of attachment. No fluctuation between the fontanelle and tumour. Twice the swelling was tapped, and ʒss. of a solution of iodine, gr. x, pot. iod. gr. xxx, glycerine ʒi., injected.

The tumour became firm and very much reduced in size.

General Injuries:

Burns.—A woman, aged 23, was severely burnt about the upper part of the chest and right shoulder. At the end of nine weeks she was well enough to walk about the Ward, but soon the scar began to ulcerate, and she complained of feeling ill. Ten days afterwards she became suddenly collapsed, and died in 48 hours.

In the Duo denum were two small ulcers, each of the size of a six-penny piece, near the Pylorus. Their floors had given way, and allowed the contents of the bowel to enter the peritoneal cavity.

Injuries of the Chest:

A railway porter was struck in the chest by an engine, and knocked down. Two hours afterwards he died.

The aorta was divided between the Innominate and left Common Carotid as if by a vertical incision. Only a small part of the tube, about 2 lines wide, was left unsevered in the concavity of the arch. The blood had dissected up the fibrous tissue covering the aorta and great vessels of the neck and chest, and formed a layer over the pericardium and about the roots of the lungs.

No fracture of the bones of the chest, nor mark of injury on the surface. No disease of the heart or vessels.

Injuries of the Abdomen:

Rupture of the Stomach.—A printer, aged 15, was crushed between a horizontal press and a wall at the level of the epigastrium. He soon began to vomit. The food brought up the first time, he was sick, was faintly tinged with blood; but none was seen afterwards. He never rallied from the shock of the accident, and died in ten hours.

The stomach had been torn across, near the Pylorus, from the lower to the upper border. Blood and food were effused in the abdominal cavity.

Rupture of the Bladder.—A labourer, aged 48, was kicked in the hypogastrium whilst fighting on the morning of June 11. He had not passed water since the preceding evening.

At once he became very ill, with severe pain in the belly, and he soon made many ineffectual attempts to pass water. A catheter drew off some urine mixed with blood, and then some pure blood. In the afternoon he began to show symptoms of peritonitis, and continued suffering occasional attacks of agonising pain in the belly.

On 12th June—29 hours after the injury—the abdomen was opened, much dark, bloody fluid let out, and an oblique rent, 3½ inches long, found in the upper and back part of the bladder. The rent was closed with silk sutures, the abdomen sponged out and sewn up. The man survived the operation 22 hours. (St. Barth. Hosp. Reports, Vol. XII., page 209, for full account.)

Wound of the Abdomen.—A policeman, aged 21, whilst engaged in a struggle with some roughs, was stabbed in the belly, midway between the anterior superior iliac spine and the umbilicus.

He had suffered a penetrating wound of the abdomen an inch long, through which a portion of Omentum had escaped. It was returned by enlarging the wound in the skin and subcutaneous tissue. Superficial suppuration occurred about the wound, and tracked downwards to the groin. In six weeks he left the Hospital with a small ventral hernia at the seat of the injury, but in other respects well.

Injuries of the Upper Extremity:

Compound Dislocation of the Shoulder.—This was the effect of a machinery accident to a man aged 19. His arm, entangled in his coat, was dragged forwards and upwards into some machinery in motion.

A large lacerated wound crossed the axilla and exposed the head of the humerus, dislocated under the coracoid process. The great vessels and nerves were uninjured. A small fragment of bone was broken from the head of the humerus. The dislocation was reduced, the arm laid on a pillow a little away from the side, and light dressings applied.

He survived the shock of the accident and the profuse suppuration that followed it, and at the end of several weeks the wound had healed, leaving some sinuses discharging foul pus.

In the seventeenth week the necrosed head of the humerus, with one inch of the shaft, was removed through an incision over the front of the joint.

The sinuses at length closed, and the man recovered with 1½ inches shortening of the arm, with a stiff shoulder, and with fair use of the hand and elbow.

(St. Barth. Hosp. Report, Vol. XII., page 41, for full account.)

INJURIES OF THE LOWER EXTREMITY:

A man, aged 43, received a large punctured and contused wound about the middle of the back of the thigh by falling out of a cart on an upright axle-tree.

The wound suppurated deeply.

On the seventh day symptoms of tetanus began to appear, and were well-marked in 24 hours. The great sciatic nerve was then exposed by incision at the seat of injury, where it lay in a large abscess, swollen and bathed in pus, and was thoroughly stretched. The symptoms were but little affected by the operation, and death occurred on the morning of the tenth day.

Pott's Fracture.—The fatal case was that of a man, aged 58, who died on the fourth day of Delirium Tremens.

Dislocation of the Hip.—This was a case of recent injury to an old dislocation on the dorsum ilii in a child aged 7. Reduction was not effected.

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.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
GENERAL DISEASES.																																											
Phagedæna ..	..	..	..	..	..	..	..	..	..	..	..	..	2	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	2	..	..
Erysipelas—																																											
a. Simple ..	1	2	..	..	1	..	..	..	..	..	..	..	1	4	..	..	5	2	..	..	2	2	..	..	3	2	1	..	3	..	..	..	..	..	..	..	..	..	..	..	16	12	1	..
b. Phlegmonous ..	..	1	..	..	2	..	..	..	1	1	..	..	2	..	..	..	5	1	..	..	6	1	1	..	6	3	2	..	2	..	..	1	..	..	1	..	..	..	..	..	24	7	4	1
c. Diffuse Inflammation ..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	3	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	3	1	1	..
Pyæmia	..	..	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	..	1	1	1	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	2	2
Syphilis—																																											
A. Primary—																																											
Hard Chancre ..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	3	..	..
Soft Chancre ..	..	..	..	..	..	..	..	..	..	..	..	..	5	4	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	4	..	..
Phagedænic Sore ..	..	..	..	..	..	..	..	..	..	..	..	..	9	..	..	..	3	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	13	..	..	..
B. Hereditary ..	1	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1	..	..

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.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
GENERAL DISEASES (*continued*).																																													
Syphilis (*continued*)																																													
Local Syphilitic Affections—																																													
Tongue	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..
Palate and Pharynx ..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	4	..	..
Larynx	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	2	..	..	..	..	1	..	..	..	..	..	1	..	..	..	..	..	1	4	1	..
Rectum	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..
Testis	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Eye	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..
Bone	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	2	..	..	2	2	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	6	..	1
Skin	..	..	..	..	..	..	..	..	1	..	..	..	19	20	..	..	4	10	..	..	2	4	..	..	2	..	..	..	1	1	..	..	..	..	..	..	..	..	28	36	..	..
Cancer—																																												
A. Scirrhus—																																												
Rectum ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	1	..	2	1	..	..	..	..	..	..	1	..	2	3
Female Breast	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	6	..	..	..	3	..	..	..	4	..	..	..	2	..	..	..	..	17	..	..
Lymphatic Glands ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..
Œsophagus ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	1	..	..	..	1	..	..	..	..	..	3	..	1	..
Penis	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Bladder ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..

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.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
GENERAL DISEASES (*continued*).																																											
Cancer (*continued*)																																											
B. Medullary—																																										
Breast ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	2	..	..
Bones ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	1	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	3	1	1	..
Glands ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..
Bladder ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	2	..	..	..	..	..	1	..	..	..	..	..	3	..	1	..
C. Epithelial—																																											
Lip	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..	4	..	..	..	1	..	..	..	1	..	..	..	9	..	..	..
Tongue ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	1	..	1	..	2	..	..	5	1	1	..	2	..	..	..	..	..	..	..	11	4	1	1
Mouth ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Pharynx ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..
Face	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	2	..	..	..
Eyelid ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	3	1	..	..
Scrotum ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..
Anus ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..
Leg	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Lupus ..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..
Rodent Ulcer.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..

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.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
GENERAL DISEASES (*continued*).																																													
Serofula—																																													
Lymphatic Glands ..	..	1	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	1	..	..
Testicle ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Joints ..	3	4	..	..	10	8	..	1	7	1	..	..	4	2	..	..	1	1	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	25	16	..	2
Skin	..	..	..	1	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	1
Chronic Rheumatic Arthritis ..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	1	1	..	..	2	1	..	..	..	1	..	..	..	..	..	..	5	3	..	..
Vaccination ..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..
DISEASES OF NERVOUS SYSTEM.																																												
Malingering ..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Neuralgia	..	..	..	..	..	..	..	..	1	..	..	..	2	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	..	..
Hysteria	..	..	..	..	..	..	..	..	2	..	..	..	8	..	..	..	1	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	13	..	..
Tetanus, Traumatic	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..
Paralysis of Superior Maxillary Nerve	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..

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.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE EYE.																																													
Conjunctiva	1	3	..	..	..	1	..	..	..	..	..	..	4	1	..	..	1	..	..	..	3	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	10	6	..	..
Cornea	4	3	..	..	5	5	..	..	2	5	..	..	13	10	..	..	1	4	..	..	4	..	..	..	2	4	..	..	..	..	..	..	1	..	..	..	..	..	..	..	32	31	..	..
Iris	..	1	..	..	..	..	..	..	1	..	..	..	6	2	..	..	5	10	..	..	2	2	..	..	4	3	..	..	2	3	..	..	..	..	..	..	..	..	..	..	20	21	..	..
Crystalline Lens ..	..	1	..	..	7	2	..	..	5	3	..	..	7	1	..	..	..	3	..	..	..	1	..	..	7	4	..	..	11	16	..	..	3	6	..	..	..	1	..	..	40	38	..	..
General Affections of the Eye ..	..	..	..	..	2	1	..	..	2	..	..	..	2	..	..	..	..	3	..	..	..	2	..	..	1	5	..	..	1	4	..	..	1	5	..	..	..	..	..	..	9	20	..	..
Strabismus ..	..	2	..	..	5	5	..	..	3	2	..	..	2	5	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	11	14	..	..
Lachrymal Apparatus	1	..	..	..	..	1	..	..	..	2	..	..	..	2	..	..	2	3	..	..	2	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	10	..	..
Eyelids	..	..	..	..	..	2	..	..	..	..	..	..	2	1	..	..	..	..	..	..	..	1	..	..	..	2	..	..	..	1	..	..	..	..	..	..	..	..	..	..	2	7	..	..
Cranial Nerves ..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..
Retina and Optic Nerve	..	..	..	..	..	1	..	..	3	..	..	..	2	2	..	..	..	1	..	..	1	1	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	7	5	..	1
Choroid	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..
Diseases of Orbit. .	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	1
Vitreous	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..
DISEASES OF THE EAR.																																												
Otorrhœa	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1	1	..
Polypus	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..

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.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE NOSE.																																											
Epistaxis	..	..	..	..	..	..	..	..	..	..	..	..	1	2	..	..	3	1	..	..	3	..	..	..	3	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	12	3	..	..
Polypus	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	2	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	2	..	..
Malignant Polypus	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Ozœna	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..
DISEASES OF THE CIRCULATORY SYSTEM.																																											
Subclavian and Axillary Artery	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	2	..
Popliteal	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	..	..	..
Aneurismal Varix	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Thrombosis ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	4	..	..	2	1	..	..	3	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	6	7	..	..
Secondary Hæmorrhage ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Lymphatic Glands	..	..	..	1	1	..	..	..	..	1	..	..	2	1	..	..	5	3	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	8	6	..	1
Varicose Veins ..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	..	..	..	..	2	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	2	..	..
Lymphangitis ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	2	1	..	..
DISEASES OF THE LIPS.																																											
Harelip	6	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	3	..	..

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.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF MOUTH AND CHEEK.																																											
Stomatitis	..	..	..	..	1	..	..	..	..	1	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	1	..	..
Ranula	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
DISEASES OF GUMS AND JAWS.																																										
Epulis	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	..	..	2	2	..	..	1	..	..	..	1	1	..	..	..	..	1	..	..	..	5	6	..	..
Tumours	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..
Diseases of Tongue	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	..	..
DISEASES OF PALATE AND FAUCES.																																										
Enlarged Thyroid ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1
Enlarged Tonsils ..	..	..	..	..	1	..	..	..	..	1	..	..	1	2	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	4	..	..
Tonsillitis ..	..	1	..	..	..	2	..	..	..	..	..	..	..	12	..	..	..	6	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	22	..	..
Cleft Palate ..	4	2	..	..	..	3	..	..	1	2	..	..	1	5	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	12	..	..

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.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF SALIVARY GLANDS.																																												
Parotid Glandular Tumour ..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..
Enchondroma ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
DISEASES OF ŒSOPHAGUS AND LARYNX.																																											
Laryngitis ..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1	..	..
DISEASES OF THE INTESTINES.																																											
Hernia—																																											
Umbilical ..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	2	..	..	..	..	..	..	..	6	..	..
Inguinal ..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	1	..	7	..	..	..	2	..	..	..	7	2	..	..	7	..	..	..	6	..	..	..	1	..	1	..	33	2	2	..
Femoral ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	2	1	..	1	5	..	..	4	1	1	1	1	3	1	1	..	..	..	..	..	..	..	..	6	13	3	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.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE INTESTINES (*continued*).																																												
Hernia—																																											
Fæcal Fistula and Abscess	..	1	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..
DISEASES OF RECTUM AND ANUS.																																											
Fistula in Ano	..	..	..	..	..	..	..	..	..	..	..	..	5	..	..	..	3	2	..	..	2	1	..	..	1	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	7	9	..	..
Hæmorrhoids	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	2	..	..	3	1	..	..	4	2	..	..	..	..	..	..	1	..	..	..	..	..	..	..	8	6	..	..
Fissure of Anus	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	..	..
Fibrous Stricture	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	3	..	..
Prolapsus Ani	2	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1	..	..
Ischio-Rectal Abscess	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	3	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	4	..	..
Recto-Vesical Fistula	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..
Polypus	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..

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.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF URINARY SYSTEM.																																												
Malformation—																																											
Ectopia Vesicæ	..	..	1	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..
Renal Calculus and Abscess	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	1	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	2	1	..	..
Cystitis—																																											
Acute	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Chronic	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	2	..	1	..
Calculus Vesicæ—																																											
a. Uric Acid	2	..	..	..	1	1	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	7	1	..	..
b. Oxalate of Lime	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	4	..	..	..
c. Phosphatic	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	1	..	..	..	1	..	1	..
Urethral Calculus	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Irritable Bladder	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	2	..	..	..
Tubercular Disease	..	..	..	..	1	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	..	..
Foreign Body	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..
Diseases of Prostate Gland.																																											
Enlarged Prostate	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	..	..	..	4	..	2	..	1	..	..	..	10	..	2	..

TABLE II (*continued*).

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.		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		M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF URINARY SYSTEM (*continued*).																																												
Gonorrhœa and its Complications.																																												
Gonorrhœa ..	..	..	..	..	..	..	..	..	..	1	..	..	11	35	..	..	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	14	36	..	..
Paraphimosis ..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..
Bubo	..	..	..	..	..	..	..	..	..	..	..	..	1	3	..	..	4	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	3	..	..
Verrucæ	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..
Perineal Abscess ..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..
Orchitis	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..
Diseases of Urethra.																																												
Urethritis.. ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Stricture—																																												
a. Organic ..	..	..	..	..	..	..	..	..	1	..	..	..	4	..	..	..	10	..	2	..	15	..	..	..	17	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	47	..	4	..
b. Traumatic ..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	2	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	..	..	..
c. Spasmodic ..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Urinary Fistula ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..

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.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF URINARY SYSTEM (*continued*).																																												
Diseases of Urethra (continued).																																												
Extravasation of Urine	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	1	..
Incontinence of Urine	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Retention of Urine ..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	2	..	..	..	4	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	1	..	..	..	10	..	..	..
Abscess	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Diseases of Penis and Testis.																																												
Malformations—																																												
Hypospadias ..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..
Phimosis ..	7	..	..	..	4	..	..	..	6	..	..	..	8	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	27	..	..	..
Paraphimosis ..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	..	..	..
Hæmatocele ..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	3	..	..	..	..	..	..	..	..	..	..	..	6	..	..	..
Hydrocele ..	1	..	..	..	1	..	..	..	1	..	..	..	7	..	..	..	2	..	..	..	4	..	..	..	2	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	20	..	..	..
Varicocele ..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	..	..	..
Tumours ..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Inflamed Scrotum	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Orchitis ..	..	..	..	..	1	..	..	..	..	..	..	..	2	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	..	..	..

TABLE II (*continued*).

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.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF FEMALE ORGANS OF GENERATION.																																												
Diseases of the Ovary	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..	2	..	2	..	..	..	1	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	7	..	..	3
Diseases of Vagina.																																												
Vesico-Vaginal Fistula	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	..	..
Diseases of Vulva.																																											
Abscess of Labium	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..
Noma	..	2	..	1	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	1
Ruptured Perinæum	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	2	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	..	..
DISEASES OF THE FEMALE BREAST.																																											
Indurated Breast	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..
Hypertrophy ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Abscess	..	..	..	..	..	..	..	..	..	..	..	..	..	8	..	..	..	5	..	..	..	4	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	18	..	..

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.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE FEMALE BREAST (*continued*).																																											
Non-Malignant Tumours—																																											
Cysts	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Mammary Glandular	..	..	..	..	..	..	..	..	..	..	..	..	..	5	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	..	..
Sero-Cystic Sarcoma	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	4	..	..
DISEASES OF THE ORGANS OF LOCOMOTION.																																										
Periostitis ..	2	..	..	..	..	1	..	..	1	..	..	..	5	5	..	..	3	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	12	7	..	..
Chronic Abscess ..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Diffuse Periostitis—																																										
Caries ..	1	..	..	..	2	..	..	..	1	..	..	..	5	1	1	..	6	1	..	..	2	1	..	..	2	1	..	..	..	..	..	..	..	..	..	..	..	..	19	4	1	..
Necrosis ..	2	..	..	..	7	..	..	..	4	5	..	1	11	5	..	..	3	3	..	..	3	1	..	..	3	1	..	..	2	..	..	..	2	..	..	..	..	..	37	15	..	1
Perforating Ulcer .	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	2	..	..	..	..	..	..	..	..	..	..	..	..	2	2	..	..

DISEASES.	Under 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.		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	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 (*contd.*).																																												
Tumours—																																											
a. Exostosis ..	..	..	..	..	1	1	..	..	..	..	..	..	2	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	3	..	..
b. Sarcoma ..	1	1	..	..	..	..	..	..	..	..	..	..	3	2	..	..	2	1	..	..	1	1	1	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	8	5	1	1
Old Amputations, &c.	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	1	..	..
Diseases of Joints.																																											
Acute Synovitis ..	..	..	..	..	..	..	..	..	..	1	..	..	5	1	..	..	7	3	..	..	4	1	..	..	2	..	..	..	1	..	..	..	..	..	..	..	..	..	19	6	..	..
Chronic do. ..	1	..	..	..	2	..	..	..	2	1	..	..	2	5	..	..	3	4	..	..	2	1	..	..	1	2	..	..	..	..	..	..	..	..	..	..	..	..	13	13	..	..
Chronic Disease ..	..	2	..	..	1	4	..	..	2	2	1	..	6	5	1	1	2	3	..	..	3	3	..	..	3	..	1	..	..	..	1	..	..	..	..	..	..	..	17	19	4	1
Knock-Knee, &c. ..	..	..	..	..	4	..	..	..	1	..	..	..	4	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	9	..	..	..
Disease of Sacro-iliac Synchondrosis ..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	2
Loose Cartilage in Knee	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..
Diseases of the Spine.																																											
Caries	2	..	..	..	2	2	..	..	1	2	..	..	..	1	..	1	1	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	6	1	1
Psoas, Lumbar, and other Abscesses .	2	..	..	..	..	2	..	..	..	..	..	..	2	2	..	..	1	1	..	..	1	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	6	6	..	..

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.		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	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 (*contd.*).																																											
Diseases of the Spine (contd.).																																											
Angular Curvature	1	..	..	..	3	..	..	..	..	1	..	..	3	3	..	..	1	1	..	..	1	..	..	..	1	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	10	5	1	..
Lateral Curvature	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	3	..	..
Spina Bifida, &c.	1	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	1	..
Diseases of Muscles, Tendons, &c.																																											
Contraction of Tendons, Fasciæ, or Muscles ..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	..	..	..	1	..	..	..	2	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	2	5	..	1
Club-Foot—																																											
a. Talipes Equinus	..	1	..	..	4	4	..	..	1	1	..	..	1	1	..	..	1	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	7	8	..	..
b. Do. Valgus	..	..	..	..	..	..	..	..	1	..	..	..	6	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	7	2	..	..
c. Do. Equino-Varus	15	5	..	..	3	2	..	..	2	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	20	8	..	..
d. Do. Varus	1	..	..	..	..	..	..	..	1	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	2	..	..
Wry-Neck	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..

TABLE II (*continued*).

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.		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	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 (*contd.*).																																											
Diseases of Muscles, Tendons, &c. (contd.)																																											
Enlarged Bursa Patellæ										1											1	2			3					1											4	4		
Bursal Tumour																	1	2								1															1	3		
Inflammation and Suppuration of Bursa Patellæ													3	15			1	2			2	3			1	2															7	22		
Do. do. of other Bursæ																	1																								1			
Diffuse Palmar Ganglion													1													1															1	1		
DISEASES OF THE CELLULAR TISSUE.																																											
Abscess	6	6			10	5			8	4			20	25			15	14		1	9	5	1		5	4	1		1	2		1	3	1						77	66	2	2
Connective Tissue Tumours—																																											
a. Fatty						1				1				3			1	6			2	2			2	2							1							5	16		
b. Fibro-cellular																						2					1													2		1	

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.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
DISEASES OF THE CELLULAR TISSUE (*continued*).																																											
Tumours—																																											
c. Sarcoma ..	1	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	2	1	..	..	..	..	..	..	..	1	..	..	1	..	..	..	..	..	..	..	4	3	1	..
d. Fibrous ..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	2	2	..	..	2	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	3	..	..
Painful Subcutaneous Tumour	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1	3	..	..
Sebaceous Cysts ..	1	2	..	..	..	..	..	..	1	1	..	..	3	2	..	..	1	1	..	..	1	2	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	8	8	..	..
Dermoid Cysts ..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Elephantiasis ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Hygroma	2	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1	..	..
DISEASES OF THE CUTANEOUS SYSTEM.																																											
Ulcer	1	1	1	..	1	1	..	..	1	1	..	..	11	6	..	..	9	..	..	..	4	2	..	..	1	6	..	..	1	6	..	..	2	..	..	1	..	..	..	..	31	23	1	1
Carbuncle	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	1	..	..	..	3	2	..	..	1	..	..	..	1	..	..	..	..	..	..	..	8	2	..	..
Gangrene	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	1	..	..	..	..	..	..	..	3	1	..	..
Cicatrix	..	..	..	..	..	1	..	..	..	1	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	..	..
Nævus	3	3	..	..	..	1	..	..	1	1	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	6	..	..
In-growing Toe-Nail	..	..	..	..	..	..	..	..	1	..	..	..	5	3	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	5	..	..
Mole	..	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1	..	..
Papilloma ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Boil	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	2	1	..	..
Onychia	..	..	..	..	..	2	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	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.			
	Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		Discharged.		Died.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
GENERAL INJURIES.																																											
Burns and Scalds..	13	7	..	6	2	2	2	2	2	2	..	..	4	2	..	2	2	2	1	..	2	1	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	25	16	3	11
Contusions ..	..	1	1	..	1	..	..	..	3	..	..	..	5	1	..	..	3	1	..	..	3	1	..	..	1	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	16	6	1	..
LOCAL INJURIES.																																											
Injuries of the Head.																																											
Contusions ..	..	..	..	..	3	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	1	..	..
Scalp Wounds ..	..	..	..	..	2	..	..	..	3	..	..	..	1	2	..	..	2	..	..	..	6	4	1	..	11	2	..	..	2	..	..	..	1	..	..	..	..	..	..	..	28	8	1	..
Concussion of Brain	3	2	..	..	4	1	..	..	5	3	..	..	7	..	1	..	6	..	..	..	4	1	..	..	4	..	1	..	1	..	..	1	1	..	..	..	..	..	..	..	35	7	2	1
Fractures of Skull.	..	..	1	..	1	..	1	..	..	1	..	..	6	..	5	1	..	..	2	..	..	..	4	..	1	..	3	..	..	..	1	..	..	..	2	..	..	..	..	..	8	1	19	1
Injuries of the Face.																																											
Contusions ..	..	..	..	..	..	..	..	..	1	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	..	..
Wounds	1	..	..	..	1	..	..	..	4	..	..	..	1	..	..	..	2	..	..	..	3	1	..	..	1	1	..	..	..	1	..	..	1	..	..	..	..	..	..	..	14	3	..	..
Fractures, &c. ..	..	..	..	..	..	1	..	..	..	..	..	..	1	..	..	..	2	..	..	..	2	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	1	..	..
Injuries to Ear ..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..
Injuries of the Eye.	3	..	..	..	2	3	..	..	1	..	..	..	5	1	..	..	4	..	..	..	4	..	..	..	1	1	..	..	2	..	..	..	..	..	..	..	..	..	..	..	22	5	..	..
Injuries of the Neck.																																											
Cut Throat ..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	1	..	..
Other Injuries ..	2	..	1	..	2	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	1	1	..

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.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
LOCAL INJURIES (*continued*).																																													
Injuries of the Chest.																																													
Contusions ..	1	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	1	..	..	..	2	..	1	..	..	..	..	..	2	..	1	..	..	..	..	..	..	..	..	..	8	..	2	..
Wounds	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..
Fractured Ribs ..	1	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	2	..	2	..	2	..	..	..	1	1	1	1	3	..	1	..	1	1	1	1	..	..	..	..	10	2	7	2
Injuries of the Back.																																												
Contusions ..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	1	..	..
Fracture of Spine ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..
Other Injuries ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	1	..
Injuries of the Abdomen.																																												
Contusions ..	..	..	..	..	..	..	..	..	2	..	..	..	2	..	..	..	1	3	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	4	..	..
Do., with Rupture of Viscera.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..
Injuries of the Pelvis.																																												
Contusions ..	..	..	..	..	1	..	..	..	1	..	..	..	..	1	..	..	..	1	..	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	3	..	..
Wounds	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..

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.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Local Injuries (*continued*).																																												
Injuries of the Pelvis (continued).																																												
Fractures	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	1	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	1	..
Ruptured Urethra.	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	2	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	..	..	..
Do. Bladder ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..
Injuries of the Upper Extremity.																																											
Contusions and Sprains	..	..	..	..	2	..	..	..	..	..	..	..	1	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	2	..	..
Wound—																																											
Of Arm ..	..	..	..	..	1	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..
„ Forearm ..	..	..	..	..	1	1	..	..	4	..	..	..	3	..	..	..	1	..	1	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	10	3	1	..
„ Hand ..	..	..	..	..	..	..	..	..	4	1	..	..	6	..	2	..	5	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	15	1	2	..
Injuries of Vessels	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	1		
Fracture—																																											
Of Clavicle ..	..	..	..	..	..	1	..	..	..	1	..	..	1	..	..	..	2	..	..	..	..	..	..	..	1	1	..	..	2	..	..	..	..	1	..	..	..	..	..	..	6	4	..	..
„ Humerus ..	..	..	..	..	1	..	..	..	1	..	..	..	2	..	..	..	2	1	..	..	2	1	..	..	..	1	..	..	..	2	..	..	..	1	..	..	..	1	..	1	8	7	..	1
„ Forearm and Hand ..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	3	..	..	..	1	..	1	..	..	..	1	..	..	..	..	..	..	1	..	..	..	2	..	..	6	3	2	..
Dislocations, &c. ..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	2	..	..	..	3	1	..	..	2	1	..	..	1	..	..	..	1	..	..	..	..	..	..	..	10	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.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Local Injuries (*continued*).																																												
Injuries of Lower Extremities.																																												
Contusions ..	1	1	..	..	..	1	..	..	3	1	..	..	3	..	..	..	6	1	..	..	2	1	..	..	1	3	..	..	1	2	..	..	2	2	..	..	1	..	..	1	20	12	..	1
Sprained Ankle ..	..	..	..	..	1	..	..	..	..	..	..	..	1	3	..	..	2	1	..	..	1	1	..	..	..	2	..	..	1	..	..	..	..	..	..	..	..	..	..	..	6	7	..	..
,, Knee ..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..
,, Hip ..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..
Wounds—																																											
Of Thigh ..	1	..	..	..	..	..	..	..	2	..	..	..	2	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	..	1	..
Over Knee ..	..	..	..	..	..	..	..	..	..	1	..	..	2	..	..	..	2	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	4	3	..	..
Of Leg	1	..	..	..	..	..	..	..	2	..	..	..	1	1	..	..	1	..	..	..	2	..	1	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	9	1	1	..
Of Foot ..	..	..	..	..	1	..	..	..	4	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	..	..	..
Of Knee-Joint ..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	1	..
Fracture of Femur	11	6	..	..	8	3	..	..	6	..	..	..	6	..	..	..	2	1	1	..	2	2	..	..	5	1	1	..	3	2	..	..	..	3	..	..	..	..	..	..	43	18	2	..
Cervix Femoris ..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	1	..	..	1	2	..	..	..	1	..	1	..	4	..	1	3	8	..	2
Patella	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	9	1	..	..	3	7	..	..	4	1	..	..	..	1	..	..	1	..	..	..	..	..	..	..	18	10	..	..
Both Bones of the Leg—																																											
Simple	3	..	..	..	2	..	..	..	6	1	..	..	12	2	..	..	16	6	..	..	13	8	..	..	15	6	..	..	6	12	..	..	5	1	..	..	1	..	..	..	79	36	..	..
Compound ..	1	..	..	..	..	..	..	..	1	..	..	..	2	..	..	..	3	..	..	..	1	..	..	..	1	3	..	..	1	..	..	..	1	..	1	..	..	..	..	..	11	3	1	..
Fracture of Tibia—																																											
Simple	1	..	..	..	..	1	..	..	4	2	..	..	3	..	..	..	3	..	..	..	9	1	..	..	7	3	..	..	6	1	..	..	2	..	..	..	3	..	..	..	38	8	..	..
Compound ..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	2	1	..	..

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.		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	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
LOCAL INJURIES (*continued*).																																													
Injuries of Lower Extremities (contd.).																																													
Fracture of Fibula—Simple ..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	6	3	..	..	6	2	..	..	1	1	..	..	1	1	..	..	..	..	..	..	..	..	..	..	15	7	..	..
Fracture of Bones of Foot	..	..	..	..	1	..	..	..	2	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	5	1	..	..
"Pott's Fracture" of Leg	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	..	2	..	..	..	1	..	..	..	..	..	..	..	3	..	1	..	..	..	..	..	..	..	..	9	..	1	..
Dislocations ..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	1	..	..
Rupture of Muscles and Tendons	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..

SURGICAL OPERATIONS PERFORMED.

OPERATIONS.	AGE AND SEX.																									
	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
Operations on the Eye.																										
Strabismus	..	1	5	5	5	6	1	..	..	..	..	..	..	..	..	..	..	..	11	12	11	12	..	..	..	..
Iridectomy	2	2	3	2	6	1	3	2	3	4	2	2	5	6	1	6	..	..	25	25	23	25	2	..	..	..
Sclerotomy	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	2	..	2	..	..	..	..
Cataract—																										
Linear Extraction	..	..	3	1	2	1	..	..	..	..	2	1	2	6	9	11	1	3	19	23	18	23	1	..	..	..
Needle Operation	1	1	1	..	4	2	2	3	..	..	..	..	..	1	..	1	..	..	8	8	8	8	..	..	..	..
Suction „	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..
Wecker's „	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..	1	1	1	1	..	..	..	..
Abscission	..	..	1	1	2	2	2	..	..	..	..	..	..	..	..	..	..	..	5	3	5	3	..	..	..	..
Extirpation of Globe	..	..	4	1	3	..	1	1	1	2	2	1	1	4	..	..	..	1	12	10	12	10	..	..	..	..
Syndectomy	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..
Entropion	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	1	..	..	..	3	..	3	..	..	..	..
Anterior chamber tapped	..	..	..	..	1	..	..	1	1	1	1	..	..	..	..	..	..	..	3	2	3	2	..	..	..	..
Canaliculi slit up	..	..	..	1	..	3	..	1	..	2	..	1	..	..	..	..	..	..	..	8	..	8	..	..	..	..
Excision of Joints and Bones.																										
Hip	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	1	1	..	..	..	..

OPERATIONS.	AGE AND SEX.																									
	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*).																										
Knee	..	..	..	..	2	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	1	..	..	..	1	..
Elbow	..	..	..	..	..	1	1	..	1	..	1	..	..	..	..	..	..	..	3	1	2	1	..	..	1	..
Removal of Sequestra:																										
From Head and Face	1	..	..	..	..	2	..	1	1	..	..	..	..	..	..	..	..	..	2	3	2	3	..	..	..	..
„ Upper Extremity	..	..	..	..	3	1	1	2	..	..	..	..	1	..	1	..	..	..	6	3	6	3	..	..	..	..
„ Lower Extremity	..	..	7	..	6	2	1	1	2	..	..	..	..	1	..	..	..	..	16	4	16	4	..	..	..	..
Amputations.																										
Primary:																										
Shoulder joint	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..
Arm	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	2	..	2	..	..	..	..	..
Forearm	..	..	..	..	..	1	..	..	..	..	1	..	..	..	..	..	..	..	1	1	..	1	..	..	1	..
Parts of Hand	Many		..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Thigh	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..
Leg	..	..	..	..	1	..	..	..	..	..	1	..	..	..	1	..	..	..	3	..	1	..	..	..	2	..
Parts of Foot	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..
Secondary:																										
Thigh..	..	..	..	..	..	..	1	..	1	..	..	..	..	..	..	..	..	..	2	..	1	..	..	..	1	..
Parts of Hand	..	..	..	..	1	..	1	..	1	..	..	..	..	..	..	..	..	..	3	..	3	..	..	..	..	..

OPERATIONS.	AGE AND SEX.																									
	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
Amputations (*continued*).																										
For Disease:																										
Shoulder Joint	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	1	..
Arm	..	..	..	..	..	..	1	..	..	..	3	..	..	..	..	..	..	..	4	..	3	..	..	..	1	..
Forearm	..	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..	..	..	2	..	2	..	..	..	..	..
Hip Joint	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	1
Thigh	..	..	1	1	2	2	1	..	..	..	..	1	1	..	..	..	..	..	5	4	4	4	..	..	1	..
Leg	..	..	..	..	1	..	..	..	..	..	1	..	..	..	..	..	..	..	2	..	1	..	..	..	1	..
Ankle Joint	..	..	..	..	1	..	4	..	..	..	..	1	..	..	..	..	..	..	5	1	4	1	..	..	1	..
Parts of Hand	..	..	Many		..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Parts of Foot	..	..	Many		..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Removal of Tumours.																										
Cancer:																										
Jaw	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	..
Breast	..	..	..	..	..	..	..	1	..	4	..	4	..	5	..	1	..	..	..	15	..	15	..	..	..	..
Tongue	..	..	..	..	..	..	..	..	..	2	1	..	1	2	2	..	1	..	5	4	4	3	..	..	1	1
Lip	..	..	..	..	..	..	..	..	..	..	2	..	5	..	..	..	2	..	9	..	9	..	..	..	..	..
Mouth	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	1	..	..	..	..	..
Penis and Scrotum	..	..	..	..	..	..	..	..	2	..	1	..	2	..	..	..	..	..	5	..	5	..	..	..	..	..
Skin	..	..	..	..	..	..	..	..	..	1	..	..	..	..	1	..	2	..	3	1	3	1	..	..	..	..
Rodent Ulcer	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	1	..	1	..	..	..	..	..

OPERATIONS.	AGE AND SEX.																									
	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
Removal of Tumours (*continued*).																										
Adenoid Tumours:																										
Breast	..	..	..	..	..	1	..	4	..	..	..	..	..	..	..	..	..	..	..	5	..	5	..	..	..	..
Parotid Gland	..	..	..	..	..	..	2	..	1	..	..	..	..	..	..	..	..	..	3	..	3	..	..	..	..	..
Connective Tissue Tumours:																										
Fatty	..	..	..	1	..	2	..	4	1	5	2	1	1	..	..	1	..	..	4	14	4	14	..	..	..	..
Fibro-cellular and Fibrous	..	..	..	..	1	..	1	2	..	2	2	1	2	..	..	..	..	..	6	5	5	5	..	..	1	..
Sarcomata	..	..	..	1	1	..	1	..	..	1	1	3	..	..	1	1	..	..	4	6	4	6	..	..	..	..
Exostosis	..	..	1	1	1	..	1	..	..	..	..	..	..	..	..	..	..	..	3	1	3	1	..	..	..	..
Enchondroma	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..
Epulis	..	..	..	..	..	..	1	..	..	3	3	1	..	1	..	..	..	1	4	6	4	6	..	..	..	..
Bursal	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	2	..	2	..	..	..	..
Sebaceous Tumours	..	..	1	1	..	..	2	2	3	1	..	..	1	1	..	1	..	..	7	6	7	6	..	..	..	..
Papillomata	..	..	..	..	..	1	1	1	..	..	..	..	..	1	..	..	..	..	1	3	1	3	..	..	..	..
Tonsils removed	..	..	..	..	1	..	1	3	..	..	..	1	..	..	..	..	..	..	2	4	2	4	..	..	..	..
Testicle removed	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..
Lower Jaw removed	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..
Nœvus	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	3	3	3	..	..	..	..
Removal of Calculi.																										
By Lithotrity	..	..	..	..	..	..	1	..	..	..	1	..	..	..	2	..	..	..	4	..	3	..	..	..	1	..

OPERATIONS.	AGE AND SEX.																									
	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
REMOVAL OF CALCULI—*continued.*																										
By Lithotomy—Lateral	2	..	1	..	1	..	..	..	..	..	..	..	..	..	1	..	..	..	5	..	5	..	..	..	..	..
Dilatation of Urethra	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	..
From Kidney	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	1	..	..	..	..
INCISIONS.																										
Colotomy	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	1	..	..	2	1	..	..	..	..	2	1
Gastrotomy	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	1	..
Abdominal Section	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	..	..	..	1	..
Ovariotomy	..	..	..	..	..	..	..	2	..	1	..	..	..	..	..	..	..	..	..	3	..	2	..	..	..	1
Incomplete Operation	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	2	..	..	..	..	..	2
Exploratory Incision	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..
For Hernia:																										
Inguinal:																										
Herniotomy—																										
Opening of Sac	..	..	..	..	1	..	1	..	..	..	2	..	..	..	2	..	1	..	7	..	6	..	..	..	1	..
Without opening	..	..	..	..	..	..	1	..	..	..	..	..	1	..	..	..	1	..	3	..	2	..	..	..	1	..
Femoral:																										
Herniotomy—																										
Opening of Sac	..	..	..	..	..	..	..	..	1	1	..	..	1	4	2	2	..	2	4	9	1	7	..	..	3	2
Without opening	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	1	..	..	..	..

OPERATIONS.	AGE AND SEX.																									
	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
Incisions (*continued*).																										
Hæmorrhoids—																										
By Excision or Ligature	..	..	..	..	..	..	..	..	..	1	1	..	..	1	1	..	..	..	2	2	2	2	..	..	..	..
Fistula in Ano	..	..	..	..	..	1	..	3	5	2	1	..	1	..	..	..	..	..	7	6	7	6	..	..	..	..
Tenotomy	14	3	6	1	4	1	1	..	..	..	..	..	..	..	..	..	..	..	25	5	25	5	..	..	..	..
Diffuse Palmar Ganglion	..	..	..	..	..	..	1	..	..	..	..	..	..	1	..	..	..	..	1	1	1	1	..	..	..	..
Phimosis	7	..	4	..	11	..	2	..	1	..	1	..	..	..	..	..	..	..	26	..	26	..	..	..	..	..
Tracheotomy	2	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	3	..	2	..	..	..	1	..
Perinæal Section	..	..	..	..	..	..	1	..	2	..	..	..	1	..	..	..	..	..	4	..	4	..	..	..	..	..
Reparative Operations.																										
Harelip	7	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	7	2	7	2	..	..	..	..
Cleft Palate	1	1	..	3	1	5	2	..	..	..	..	..	..	..	..	..	..	..	4	9	4	9	..	..	..	..
Deformity from Cicatrices	..	..	..	1	..	2	1	1	..	..	..	..	..	..	..	..	..	..	1	4	1	4	..	..	..	..
Ectopia Vesicæ	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	1	..	1	..
Recto-vesical Fistula	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..
Recto-vaginal Fistula	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	..
Vesico-vaginal Fistula	..	..	..	..	..	..	..	1	..	..	..	3	..	..	..	..	..	..	..	4	..	3	..	1	..	..
Ruptured Perinæum	..	..	..	..	..	..	..	1	..	2	..	1	..	..	..	..	..	..	..	4	..	3	..	1	..	..
Ligature of Vessels.																										
Subclavian	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	1	..	..	..	..	..	1	..

OPERATIONS.	AGE AND SEX.																									
	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
Ligature of Vessels (*continued*).																										
Popliteal	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	2	..	2	..	..	..	..	..
Dorsalis Pedis	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..
Miscellaneous Operations.																										
Loose Cartilage in Knee	..	..	..	..	..	..	1	..	1	..	..	..	..	..	..	..	..	..	2	..	2	..	..	..	..	..
Ingrowing Toe-nail	..	..	..	..	..	..	4	1	2	2	..	..	..	2	..	..	..	..	6	5	6	5	..	..	..	..
Nerve Stretching	..	..	..	..	..	..	..	..	..	1	..	..	1	..	..	..	..	..	1	1	..	1	..	..	1	..
Trephining	..	..	..	..	..	..	1	..	1	..	1	..	1	..	..	..	..	..	4	..	1	..	..	..	3	..
Hæmatocele	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	1	..	..	2	..	2	..	..	..	..	..
Radical Cure of Hydrocele	..	..	1	..	1	..	2	..	4	..	1	.	3	..	1	..	..	..	..	..	13	..	13	..	..	..	..
„ „ Varicocele	..	..	..	..	..	..	1	..	1	..	..	..	..	..	..	..	..	..	..	..	2	..	2	..	..	..	..
„ „ Varicose Veins	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	..
„ „ Spina Bifida	..	..	2	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1	1	1	..	..	1	..
Paracentesis Vesicæ (Supra-pubic)	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	1	..	3	..	3	..	..	..	..	..

During the year 1876 Anæsthetics were administered 1726 times.

Of these Chloroform was administered.. ..	670	times.
Nitrous Oxide Gas alone	24	,,
Ether alone	28	,,
Ether, preceded by Nitrous Oxide Gas	1004	,,
	1726	

Youngest Patient	14 days.	
Oldest Patient	86 years.	No Death.

APPENDIX

TO

TABLE OF OPERATIONS.

EXCISION OF JOINTS AND BONES:

Knee.—Amputation of the thigh was performed after resection of the knee in a boy, aged 14, who died many weeks afterwards of chronic pyæmia.

Elbow.—In two cases the arm was amputated after excision had failed. In a man, aged 47, signs of phthisis existed at the apex of the left lung before the first operation. The disease became active after amputation. Old caseous masses were found at the apex of the lung, and general miliary tuberculosis of both lungs.

Removal of Sequestra.—Amputation of the leg was done on a boy, aged 13, from whose Tibia a sequestrum had been previously removed.

AMPUTATIONS:

Primary—

Forearm.—The patient had sustained a compound fracture of the skull in addition to the injury to the hand, and died in a few hours.

Leg.—On a man, age 40, who died of delirium tremens, and on one aged 69, who died of exhaustion on the 12th day. The third patient recovered.

Secondary—

Thigh.—In both cases the operation followed punctured wound of the knee-joint. In one case death occurred after profuse suppuration of the stump.

For Disease—

Shoulder.—For subclavian aneurism. (Case related in Appendix I.)

Arm.—For chronic disease of the elbow in one case, and after resection in two.

In the fourth case—of a man, aged 44—phlegmonous inflammation had destroyed the deep soft parts of the forearm, and caused ulceration of the anterior and posterior interosseous arteries. From the openings in these vessels hæmorrhage occurred into the forearm, and blood escaped several times in large quantities from an incision that had been made in the skin near the wrist. Amputation was performed in consequence of the hæmorrhage, and the man recovered.

Forearm.—For disease of the carpus.

Hip.—For Sarcoma of the Femur in a woman aged 38. She suffered from Diarrhœa after the operation, and from exhausting suppuration, of which she died. A cauliflower growth about the neck of the Uterus was found post-mortem, and the Pelvic and Lumbar glands were much enlarged by secondary deposits of cancer.

Thigh.—In four men and three women for disease of the knee.

In a girl, aged 17, for sarcoma of the femur.

In a boy, aged 16, for caries of the tibia.

Leg.—For Sarcoma of the Calf in a man aged 44, who died on the seventh day of Pleurisy. Numerous secondary deposits were found in the Lungs and Pleuræ.

After Sequestrotomy, in a boy aged 13.

Foot.—In two cases Syme's amputation was performed for caries of the os calcis.

The first was that of a man, aged 27, who suffered with disease of the os calcis in a shortened and withered limb. Over the base of his sacrum was a large prominent cicatrix, which marked the site of a Spina Bifida that had been cured by operation in infancy.

The second case is related in Appendix I.

The same amputation was performed twice for disease of the ankle, and once for perforating ulcer of the foot.

Roux operation was done in one case of diseased ankle.

Removal of Tumours:

A large fibro-cellular tumour, bigger than a man's head, was removed from the abdominal wall of a patient, aged 49. On the tenth day tetanus supervened, which proved fatal in 48 hours.

Incisions:

Gastrotomy.—On a man, aged 38, for malignant stricture of the Œsophagus. Peritonitis. Death.

Ovariotomy.—(See Appendix I).

Miscellaneous Operations:

Trephining.—In all cases for compound depressed fracture of the vault of the skull.

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	1	2	1	..	..	..	1	4	5	2	2	2	4	2	3	..	..	..	..	..	17	12	1	..
Occurring in Hospital	..	..	..	..	..	..	3	1	1	1	4	..	3	1	..	1	..	..	..	..	11	4	3	..
„ after Operations	..	..	..	..	..	..	..	3	2	1	1	..	..	1	2	1	1	..	..	..	6	6	1	..
Phlegmonous Inflammation—																								
Admissions	..	1	2	..	1	..	2	1	5	1	7	1	8	3	2	1	1	..	..	..	28	8	4	1
Occurring in Hospital	..	..	1	..	..	..	..	3	3	..	..	..	..	1	..	..	..	..	..	..	4	4	1	..
„ after Operations	..	..	..	..	..	..	1	..	1	..	..	..	1	..	1	..	..	..	..	..	4	..	2	..
Diffuse Cellulitis—																								
Admissions	..	..	..	..	..	..	..	1	3	..	..	..	1	..	..	..	..	..	..	..	4	1	1	..
Pyæmia and Septicæmia—																								
Admissions	..	..	..	..	1	..	..	1	1	1	1	..	1	..	..	..	..	..	..	..	3	3	2	2
Occurring in Hospital	..	..	..	..	..	..	1	..	1	..	..	..	..	1	..	..	..	..	..	..	2	1	2	..
„ after Operations	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	2	..	2	..
Phagedæna—																								
Admissions	..	..	..	..	..	..	2	2	..	..	..	..	..	..	..	..	..	..	..	..	2	2	..	..

TABLE OF AMPUTATIONS WITH THE PERCENTAGE OF DEATHS DURING THE TEN YEARS *from* 1867 *to* 1876, *inclusive.*

OPERATIONS.	CASES UNDER TREATMENT.										PERCENTAGE OF DEATHS.										Total Number of		Average Percentage of Deaths.
	1867	1868	1869	1870	1871	1872	1873	1874	1875	1876	1867	1868	1869	1870	1871	1872	1873	1874	1875	1876	Cases.	Deaths.	
PRIMARY.																							
Thigh	..	..	1	..	2	3	2	..	1	1	..	..	100·	..	50·	33·33	..	..	100·	..	10	4	40·
Leg	3	3	1	3	1	..	..	..	..	3	33·33	100·	..	33·33	..	..	..	..	..	66·66	14	7	50·
Ankle-Joint ..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Shoulder-Joint ..	..	..	..	..	..	..	..	1	..	1	..	..	..	..	..	..	..	100·	..	..	2	1	50·
Arm	1	2	1	2	1	1	3	3	3	2	..	..	..	50·	..	..	..	..	33·33	..	19	2	10·52
Forearm	1	7	4	1	3	5	..	..	2	2	..	14·28	..	..		..	..	..	..	50·	25	2	8·
SECONDARY.																							
Thigh	2	1	1	2	1	1	..	1	..	2	50·	100·	..	..	..	..	..	..	..	50·	11	3	27·27
Knee-Joint	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..
Leg	1	5	1	..	3	..	1	1	2	..	..	60·	..	..	66·66	..	..	..	..	..	14	4	28·57
Arm	..	..	..	2	..	2	..	..	1	..	..	..	..	..	..	..	..	..	..	..	5	..	..
Forearm	1	..	1	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	4	..	..

OPERATIONS.	CASES UNDER TREATMENT.										PERCENTAGE OF DEATHS.										Total Number of		Average Percentage of Deaths.
	1867	1868	1869	1870	1871	1872	1873	1874	1875	1876	1867	1868	1869	1870	1871	1872	1873	1874	1875	1876	Cases.	Deaths.	
FOR DISEASE.																							
Hip-Joint	..	..	..	..	..	..	1	1	2	1	..	..	..	..	..	..	..	100·	..	100·	5	2	40·
Thigh	9	13	10	9	18	15	12	19	18	9	44·44	53·84	20·	11·11	22·22	13·33	..	10·52	27·77	11·11	132	28	21·21
Knee-Joint	2	..	..	..	..	..	2	..	..	..	50·	..	..	..	..	..	..	..	..	..	4	1	25·
Leg	2	10	8	5	4	5	7	7	9	2	50·	30·	25·	40·	..	..	14·28	..	..	50·	59	10	16·94
Ankle-Joint	4	6	4	..	2	8	8	9	7	6	25·	..	25·	..	..	12·5	..	11·11	..	16·66	54	5	9·25
Shoulder-Joint ..	..	..	1	2	1	..	..	..	..	1	..	..	..	..	100·	..	..	..	..	100·	5	2	40·
Arm	3	3	1	1	..	1	..	..	2	4	..	..	100·	100·	..	100·	..	..	..	25·	15	4	26·66
Forearm	1	2	..	1	3	2	1	4	3	2	..	..	..	100·	..	..	..	..	..	..	19	1	5·26

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