Two cases of varicocele with undeveloped testicle : with remarks on the nature of varicocele ; and, A case of antiseptic osteotomy of the tibia in which rapidly fatal carbolic intoxication occurred / by A. Pearce Gould.

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TWO CASES OF VARICOCELE WITH UNDEVELOPED TESTICLE; WITH REMARKS ON THE NATURE OF VARICOCELE.

Read November 26, 1880.

CASE I.*—G. W., æt. 18, single, a waiter by trade, came under my care among the out-patients of the Westminster Hospital on Nov. 3, 1880, suffering from gonorrhœa. It was noticed that there was a large left varicocele, having the usual characters and forming a rounded mass below the testicle as well as behind and above it. The left testicle is of normal shape and about half the size of the right, or rather less, but of nearly, if not quite, the same firm consistence. The man has noticed the scrotal swelling about four or five months, but it has not occasioned any pain or other trouble, and it may have existed longer. The right testicle is of normal size and consistence. The testicular sense is slightly more acute on the left side, but of normal character. The man is tall, well formed, florid and healthy looking, has no other varicose veins visible; he has not had any injury or inflammation of either testicle, and he believes they used to be of the same size. He denies having practised masturbation, has not been continent for four years past, does not suffer from seminal emissions.

Case II.*—W. B., æt. 17, a porter by trade, came under my notice at the Westminster Hospital on Nov. 17, 1880. He was then suffering from an inflamed inguinal gland. Through the left side of the scrotum was plainly seen a

* The cases were shown at the Meeting.

large varix, the large twisted veins of which were prominent under the skin. There is nothing peculiar about the varicocele save its somewhat unusual size. The left testicle is less than half the size of the right; it is firm to the feel, and the testicular sense is slightly more acute than in the right. The right testicle is of normal size and consistence. The patient suffers no pain or inconvenience from the varicocele. He has noticed the swelling in the scrotum for six or seven years, and he does not think it has increased or altered at all during that time; is quite sure it was not there before that time. He believes the testicles used to be of equal size. The man is in robust health, with a strong muscular frame, is free from piles, or other visible varices. He has never had any affection of the genital organs, or, so far as he knows, injury to or inflammation of the testicles. He has not masturbated, and does not have frequent seminal emissions.

As there is such a marked difference of opinion among equally high authorities as to the influence of varicocele on the nutrition and function of the testicle, I have thought it worth while to bring forward these cases in the hope of throwing some light on a subject which is evidently obscure.

I will quote only the two most prominent living English authorities on this subject, Sir James Paget and Mr. Curling.

Sir James Paget* writes: 'No varicocele, to the best of my belief, ever did cause impotence or waste of testicle,' and again, 'I do not believe that it ever produced wasting of a testicle, or impotence, or any such thing.' Mr. Curling,† on the other hand, states, 'When highly or rapidly developed, the dilatation of the veins interferes so much with the nutrition of the gland as to occasion wasting. A softening and partial atrophy of the gland, co-existing with varicocele, has come under my notice in numerous instances; indeed, in nearly all the cases in which there was a decided dilatation of the spermatic veins on one side only, the testicle of that side was the smaller of the two.' 'We have evidence too, that the secreting powers of the gland are impaired, and sometimes even destroyed, by this disease.' Mr. Curling gives three examples in which the fluid of an emission was found, on examination, not to contain spermatozoa—one of the cases being Gosselin's.

* 'Clinical Lectures and Essays.'

† 'On Diseases of the Testis.'

Sir Astley Cooper* was of the same opinion as Sir James Paget, but agreeing with Mr. Curling we have Landouzy,† Richter,‡ Erichsen,§ Gross,‖ Gant,¶ Holmes,** Humphrey,†† Hurgeston,‡‡ Bumstead and Taylor,§§ Pott,‖‖ Will,¶¶ and others. It is of course impossible to discuss or criticise the negative evidence of Sir Astley Cooper and Sir James Paget. On the other hand, when we come to examine closely the cases recorded as instances of atrophy of the testicle from varicocele, we find that several of them are of no value whatever for that purpose, as both the varicocele and the wasting of the testicle followed an injury to the scrotum of greater or less severity, and even should other facts lead us to suppose that the two conditions are related to each other as cause and effect, we must put such cases out of view altogether in our present investigation.

The three cases recorded by Pott*** are for this, among other reasons, valueless in our present enquiry, and so also is the case recorded by Dr. Will, and possibly the others which he mentions having seen, as he incidentally remarks that 'in all the cases where the varicocele has been a large one, the history has pointed to a traumatic origin.' We must exclude Key's case recorded by Sir Astley Cooper, because there had been previous inflammation, and in fact, all cases where the varicocele has resulted from a blow, or followed an inflammation; atrophy of an organ after injuries or inflammation is so common that I need not justify this exclusion. When we have done this, we have several cases remaining in which there is no precise history given, and of which therefore we must speak with some hesitation. They arrange themselves into three groups:—In the first I would place the cases under consideration. I think that it is hardly possible to controvert the view that in these men, G. W. and W. B., the small size of the left testicle is in some way connected with the associated varicocele, and not a mere coincidence.††† But it is just as clear that the testicles are *small* and *not wasted*; they are of their natural shape and firmness, and preserve their peculiar sensibility,

* 'Observations on the Structure and Diseases of the Testis.'
† 'Du Varicocèle.' ‡ 'Observationes Chirurgicæ.'
§ 'Science and Art of Surgery.' ‖ 'System of Surgery.'
¶ 'Science and Practice of Surgery.' ** 'Surgery, its Principles and Practice.'
†† 'A System of Surgery,' edited by Holmes.
‡‡ *Medical Times and Gazette*, 1852, vol. 2. §§ 'Venereal Disorders.'
‖‖ 'The Chirurgical Works of P. Pott,' edited by Earle.
¶¶ *Lancet*, 1880, vol. 1. *** *Loc. Cit.* ††† Appendix A.

and present all the characters of the healthy testicle just before its full development at puberty,—*they are undeveloped testicles*—and I would submit that these cases are examples of the effect of varicocele in preventing the full development of the testicle at puberty. The one lad, æt. 17, had noticed the swelling in the scrotum six or seven years, and although the other, æt. 18, had only been aware of the varicocele for a few months, as it had not occasioned any pain or inconvenience, and was only noticed accidentally, it is quite open to us to believe that it has really existed longer. That varicocele is met with, and is even rather common before puberty, is certain; and it is not at all surprising that its existence or formation should in some way interfere with the important developmental changes occurring at that time.

2. In the second group I would place the cases believed to be quite common, where the testicle on the side of the varicocele is said to be not quite so large or firm as the other cases of very slight atrophy. I have already quoted Curling's reference to these cases. I believe that I have met with some examples, but it is difficult to estimate with certainty the size of the testicle when surrounded by dilated veins. I am sure, however, that I have never seen the left testicle larger than the right—its normal relation—when its pampiniform plexus was markedly varicose.

3. The third group includes the cases of marked unmistakable atrophy of the gland. Examples are recorded by Curling, by Richter, who speaks of having seen the testicle 'a mere rudiment,' by Landouzy who speaks of one case where the testicle was very soft and not so big as a large hazel nut; by Bumstead who writes that the testicle is 'in some cases reduced to the size of a pea, and sometimes it seems wholly absorbed,' and by Gross who says the 'testicle is liable to become soft and shrunken.' I have never met with a clear and marked case of true atrophy of the testicle which was certainly due to a varicocele, and I know of no case on record of which the description is so precise that it puts it beyond question that the small testicle was not either an undeveloped gland, or one wasted from injury or previous disease. While, however, we have no conclusive evidence on this head, it appears to be hardly justifiable to speak dogmatically in the negative, for we are quite unable to state that in many of the recorded cases there had been an injury or inflammation. And there is so

much *à priori* reason to lead us to expect such a result that it is easier to receive than to reject such evidence as we have. That a bad varicocele may induce sterility, the cases carefully investigated by Curling and Gosselin surely prove. Cases are on record where the radical cure of the varicocele has been followed by a growth of the so-called wasted testis,* (Curling, Barwell).† Thirteen months ago I operated on a man with varicocele, who was suffering from a good deal of pain in the part, with a sense of 'emptiness of the belly' after copulation, and the left testicle was slightly smaller than the right. He has lost all pain, is 'much stronger' sexually, and his left testicle is firm and plump, and if anything, larger than the right organ.‡

The much greater frequency of varicocele on the left than the right side has been variously attributed to the greater length of the left spermatic vein, to its passing beneath the sigmoid flexure, and to its mode of entrance into the renal vein. An examination of these explanations makes it appear probable that there is some other factor present. The difference in length of the two veins is slight, and does not exceed that between the two iliac veins, which has not led to a similar disproportion in the occurrence of varicosity in the veins of the lower extremities. The influence of a loaded sigmoid flexure has never been demonstrated. In only a small proportion of cases of varicocele can any history of chronic constipation be elicited, and the proportion is not greater than in men of the same age and class who are not suffering from varicocele; and on the other hand, I do not think that varicocele has ever been found to be a frequent concomitant of constipation. I recently operated on a gentleman who assured me that his varicocele always troubled him most when his bowels were confined. This is far from proving that the constipation caused the varicocele.

The difference in the mode of ending of the two spermatic veins is more generally held to account for the fact we are discussing. The left spermatic vein most frequently enters the renal vein at right angles, and in all the cases I have yet examined, where this has been the case, the opening has been guarded by a single or double valve, which I demonstrated to be capable of completely preventing the

* Mr. Curling's work on 'Diseases of the Testis.'

† After the reading of this paper Mr. Bryant mentioned a very excellent example (*vide Lancet*, Dec. 1880).

‡ This patient was shown at the Meeting.

passage of any fluid from the renal into the spermatic vein. It is not at once apparent how this valve acts; no doubt it aids and does not retard the circulation. As the circulation in the renal vein in health is practically equable, not being exposed to muscular pressure or other causes of temporary obstruction to the on-flow of blood, or of increased blood pressure, it appears probable that the valve has a constant, not an intermittent action. But if it be held that the use of the valve is to prevent regurgitation of blood, evidently it can only succeed in this when quite closed, and in that condition the flow of blood in the spermatic vein must be stopped entirely, there being no anastomosing channels along which the blood can pass; by how much is this better than regurgitation? There are valves lower down in the spermatic vain as well as at its mouth, and if there were obstruction at its mouth, the valve next below would be closed, and the first part of the vein to suffer from the obstruction would be the upper intervalvular segment, but the dilatation of varicocele is an affection of the scrotal portion of the veins and not of the upper part at all.

The lower valves can evidently have a different purpose, for where there are two or more veins, when one is closed to the blood, it can traverse the other or others, but where the vein is single and without anastomoses this is evidently impossible. I would suggest that the valve at the orifice of the vein may be of use by being partly opened, and in such a manner as to convert the direct aperture into an oblique opening directed upwards and to the right. In confirmation of this view I may mention that in a case where this valve was absent, the upper one and a half inch of the vein took an oblique course to the right, as if it thus compensated for the absence of the valve. By thus narrowing the aperture, the aspiration of the blood in the renal vein as it flows over it would be increased, and some compensation afforded for the vertical course of the spermatic vein. There are, however, several examples of veins opening at right angles into others, and without any ill effects upon the circulation in them; it is seen in the termination of the circumflex iliac veins, the lumbar veins, the renal veins, and in some of the intercostal veins, and in the vena azygos major.

Were this anatomical arrangement the main cause of varicocele, we ought to find the affection even more common than it is, and as the obstruction, if any, is constant and but little varying during health, we ought to witness serious

effects upon the testicle from the impeded return of blood—œdema, loss of function. Nor must we forget that such an explanation supposes a condition unique in the human body, the anatomical relation and structure of a part the direct cause of its disease; we are quite familiar with many examples of the manner in which anatomical arrangement and structure *modifies disease*, but in no other case, I believe, do we find it the *exciting cause* of disease. Still further in opposition to this view stands the fact that those diseased conditions which greatly increase the blood pressure in the renal vein, even to the extent of producing hæmaturia, and therefore (according to this view) the obstruction to the emptying of the spermatic vein, have not been noticed to induce varicocele. Has obstructive disease of the heart, or lungs, or pressure upon the vena cava, either from cancerous nodules in the liver or ascitic fluid, been found to produce varicocele? M. Brixoux* had a case in which a cancerous mass pressing upon the spermatic vein caused a large varicocele, showing that the disease can be produced by such damming back of the blood. When also we remember that one occasionally successful form of treatment of varicocele is the pressure of a truss applied to the cord over the external abdominal ring, we have still further reason for doubting whether the ending of the left spermatic vein has any but a very slight influence upon the production of varicocele, even if we can fairly attribute to it any at all.†

In comparing varicocele with varices of the lower extremity, there are some important differences between them which are sufficient to suggest that they may differ in nature more widely than their names imply. Varicocele certainly is not a disease of late middle life and commencing old age; it does not become frequent when the arterial pressure is lessening, and the difficulty of the venous circulation increasing. But it is most often noticed just when the testicle is passing through its most important development, or even before that, and exists during the period of its early activity. It is well to remember that varicocele may remain long unnoticed by its subjects, and that although only noticed in early manhood may really have developed much earlier. I have met with several instances of varicocele developed before puberty, and I have detected others of the existence of which the subjects were quite unaware. Although vari-

* Landouzy 'Du Varicocele.'

† Appendix B.

cocele very often develops between puberty and twenty-five, I believe that, could we get accurate information, we should find that it most commonly commences just before and at puberty.*

Other rather peculiar features of this affection are that it often remains stationary for years; that it has been noticed to undergo spontaneous cure at even so late an age as thirty, and that even when of large size it may produce no symptoms whatever, no pain, no sexual debility, no wasting of the testicle. It is rare for large varices of the lower leg when quite unsupported—and a varicocele is commonly peculiarly unsupported by the loose scrotum—not to give rise to any nutritive or nervous disturbance, and to undergo spontaneous cure.

Another point of difference between them is the fact that the veins of a varicocele, although enormously dilated, are not subject to thinning of their walls and ultimately to rupture as frequently occurs in varices elsewhere; this may be held to be connected with the different time of life and nutritive condition at which they occur. Mr. Erichsen mentions two cases of hæmatocele following varicocele, but in each case it was excited by a blow.†

Varicocele often reaches a large size, and occasionally is so immense that it proportionately far surpasses the development of other varices—a case is mentioned by Landouzy‡ where the tumour it formed was as large as the head of an infant at birth. Heredity is a point difficult to trace in this disease; the affection is not obvious, and is not one the existence of which in the father would be likely to be known by the son: but M. Blandin§ has recorded a family in which the father and three sons each had varicocele, and Landouzy speaks of a similar case. Gross says 'it occasionally occurs in several members of the same family.'‖

Putting all these facts and considerations together, I would submit that at any rate some varicoceles are not the result of a yielding of the veins to internal pressure, but are produced by a primary growth of venous tissue—are in fact venous hypertrophies. The normal pampiniform plexus is a striking arrangement, and the developmental activity in the veins forming it remarkable; it surely shows some special tendency to develop veins just at this spot in the embryo.

* Appendix C.

† Mr. Bryant recorded two similar cases when this paper was read. *Loc. Cit.*

‡ Landouzy, 'Du Varicocele.' § *Loc. Cit.* ‖ *Loc. Cit.*

And if so, what more likely to happen than that at puberty, when the testicle is undergoing its full development, or should do so, the veins should again take on the same active growth and overstep the limit between the normal and the deformed. If this view be correct, we may perhaps represent the cases shown to-night as resulting from a transference of the developmental stimulus of puberty from the testicle to the veins; while those who see in continence the cause of varicocele, and in marriage its cure, will find this a simple pathological explanation of their view.

There are other examples of such primary venous hypertrophy passing under the name of varicose veins. I refer to the hypertrophies of one or other of the saphena veins, in part or all their extent, met with in young robust men, frequently hereditary, or attacking more than one of the same generation in a family, producing no symptoms, no disturbance in the nutrition of the limb, and differing in all but name from the varicose veins coming on later in life and causing so many and serious troubles.

Appendix A.—It may be worth while to state here, the reasons that led me to think the view expressed in the paper the only tenable one. They are: (1.) That atrophied testicles are soft, flabby, misshapen, without testicular sense, and with a peculiar sensitiveness in some cases, and with wasted cord. Nothing of the kind existed in these cases. (2.) That varicocele is not a frequent sequel to atrophy of the testicle where that has been noticed to follow mumps, etc. (3.) The effect upon the testicle of the cure of the varicocele in similar cases.

As showing how the growth of the testicle at puberty may be impeded, and so having some bearing upon these cases, I quote the following passage from Curling's work on 'Diseases of the Testis:' 'In cases of congenital inguinal hernia, the testicle, even in its natural situation, was not of its proper size at the period of puberty, so that when the infirmity existed on one side only, the testicle was not more than one-half to two-thirds the size of the other gland.'

Appendix B.—In reference to the occurrence of varicocele most often on the left side, it is well to remember that the same is true of some other diseases, and notably diseases of the veins—venous thrombosis, phlegmasia alba dolens.

Appendix C.—Landouzy speaks of one case in which the varicocele had existed from infancy, and of another where it

came on at the age of seven. Mr. Bryant (*loc. cit.*) has seen three or four cases of varicocele before puberty, one in a boy of seven. I have had one case in a boy of four, and another in a boy aged eleven.

Landouzy gives the following statistics:—

In 13	cases	first noticed the varicocele	between	9 and 15 years.
In 29		„ „		15 and 25 „
In 3		„ „		25 and 35 „

Curling's statistics are:—

2	cases	between 10 and 15 years of age	when they came under notice.	
26	„	15 and 25	„	
14	„	25 and 35	„	„
5	„	35 and 45	„	„
3	„	45 and 65	„	„

Gross says: 'It often begins very early in life'; 'now and then as early as eleven.'

A CASE OF ANTISEPTIC OSTEOTOMY OF THE TIBIA, IN WHICH RAPIDLY FATAL CARBOLIC INTOXICATION OCCURRED.

Read May 13, 1881.

J. T., æt. 8, was admitted into the Westminster Hospital under my care, on October 27, 1880. He was a country lad, who had been under treatment for ricketty deformity of the legs at the National Orthopædic Hospital for some time, and he was sent to the Westminster Hospital, to have osteotomy performed. He came to the Hospital from the country, not after a stay in London. He was a ruddy healthy-looking lad, well nourished, strong in limb, and with the exception of deformity of the bones, apparently in perfect health. There was a marked curve inwards and forwards in the lower third of each femur, and the lower end of each tibia was curved outwards and forwards to a still greater extent; this curve was most marked on the left side. The boy stated that his legs had been bent as long as he could remember, and that he had worn irons and splints for years, but without any benefit. For three days the boy appeared in perfect general health, cheerful, free from pain, ate and slept well.

On October 30 he was placed under the influence of chloroform, and I divided his left tibia with McEwen's chisel and mallet, using Listerian precautions. The bone was very hard indeed, and the chisel was introduced into the wound three several times before the fracture could be completed. There was no notable hæmorrhage, the limb was put up with

the curve straightened out, and retained in splints. The spray was carbolic lotion and steam 2½ per cent.; the part operated upon, the instruments used, and my hands were washed in carbolic lotion 5 per cent., and each time the chisel was removed from the wound it was replaced in a dish of this lotion. A double layer of good 'protective' supplied to me by Martindale was placed over the wound, outside that a few folds of carbolised gauze wrung out of carbolic lotion 2½ per cent., and over all the usual carbolised gauze dressing, fastened with bandages of a similar material.

The operation was performed at 2 P.M., and lasted in all about 20 minutes. The lad quickly recovered from the effects of the chloroform, and was restless, crying out with pain until 8 P.M. He then fell asleep, and slept well all through the night. Next morning, October 31, he was free from pain, bright and cheerful, made his usual breakfast of bread and milk, and expressed himself as quite well; his temperature at 9 A.M. was 98·4°. He continued in this state until 11 A.M. (21 hours after the operation), when he vomited some yellowish fluid. At 1 P.M. he took 4 ounces of beef tea, and vomited it at once; at 3 P.M. he passed a loose orange-coloured fluid motion; at 4 P.M. he passed another similar motion, and vomited some milk and watery fluid. At 7 P.M. temperature 100·2°, he was violently sick and became very rigid and cold; he was then ordered ʒij. of brandy every half-hour, milk and soda water, and hot bottles in the bed; he vomited every dose of brandy and milk immediately. At 8 P.M. he became very restless, throwing himself about and screaming, and he was given ʒj. of brandy every ¼-hour, and soon afterwards became quiet. At 9 P.M. he was almost unconscious, speaking when roused but incoherently; he rallied after a mustard poultice applied to the left side of the chest. At 10 P.M., I saw him, and found him lying flat on his back in bed, conscious, not complaining of any pain, his face was pale, and with his arms and hands rather cold; his body and lower extremities warm, temperature in axilla 100·2°. His breathing was very hurried but not difficult, 44 per minute, air entered both lungs freely. Pulse very small and rapid, and hardly to be counted at the wrist; no murmur with the heart sounds. The tongue had a thin white fur on it; half an hour before my visit he had vomited about a pint of almost clear watery fluid; no tenderness in belly. Pupils small. He had not passed water since the early morning, and could not pass any. That

passed earlier had been unfortunately thrown away, the nurse did not notice any peculiarity in it. I ordered turpentine stupes to the chest, iced beef tea, and white of egg, and brandy. He was very restless up to 12 midnight, calling out wildly for people and things and trying to get out of bed; he took the white of an egg and an ounce of brandy and did not vomit. Between 12 and 2 A.M. he took 3 ounces of beef tea, and 2 ounces of brandy. At 2 A.M. temperature 99·6°, he was quite unconscious and sinking, at 2.35 he screamed and appeared to be choking, was raised in bed and gasped for breath and died at 2.45, exactly 36¾ hours after the operation. The bowels acted at the time of death, no urine passed, no vomiting after 9.30 the previous evening. The wound was not examined after the operation, there was no pain after the first few hours, no hæmorrhage.

Autopsy 35 *hours after death.*—Rigor mortis well marked; a good deal of post-mortem congestion reaching up to the nipple on the chest, and all over the belly. Body well nourished. *Heart* 4½ ounces; right cavities filled with soft black coagulum loosely adherent; left cavities empty and contracted; no staining of endocardium or petechiæ on surface. *Blood* everywhere black, in venæ cavæ and largest branches partly fluid, partly in very soft coagula, elsewhere entirely fluid; no staining of lining of vessels anywhere. *Lungs* Rt. 8 ounces, Lt. 7½ ounces: no petechiæ; fluid blood in pulmonary vessels; mucous membrane of bronchi congested, no excess of mucus in tubes, lung tissue apparently healthy with the exception of slight congestion. *Trachea* intensely congested, with excess of tenacious mucus on the surface. *Spleen*, 2¼ ounces, firm, unusually mottled. *Liver*, 23½ ounces, a few pale mottlings on the surface of right lobe, on section the lobules beneath for ¼ to ¾ inch were yellowish white in colour and bloodless. *Alimentary Canal* apparently healthy in the whole length, no congestion or ulceration noticed. *Brain* 49 ounces, vessels on surface full, puncta cruenta numerous and distinct. *Kidneys*, no change to naked eye. *Bladder* empty, a catheter was passed soon after death, but I only drew off two or three drops of urine. *The Wound*: a small blood clot lay in wound, none in medullary canal, no splintering of bone, cut surface of which was nearly even; tibial vessels uninjured, no thrombosis. No appearance of admixture of fat with the blood.

Portions of all the organs except the intestine were prepared for microscopical examination by my friend Dr. Gibbes.

The spleen, liver, brain and pons do not show any departure from the normal.

In the *Lungs* the changes seen are twofold : 1. Thrombosis of the minute vessels ; in some sections examined every vessel visible is seen to be plugged with blood-clot. 2. Small areas of inflammatory round-cell infiltration, some of these are very minute, others from the extent and intensity of the change might almost be called minute abscesses.

Kidneys.—Many sections show nothing at all abnormal if we exclude a rather deep staining with hæmatoxylum of the nuclei of the tubular epithelium. In other sections minute spots of inflammation are seen, consisting of masses of small deeply-stained round cells. They are generally to be traced in close relation with vessels ; some of the smaller and less marked ones are fusiform in outline and extend along a vessel, but the more marked ones are globular. Close to one of the largest of these inflamed spots is seen a good sized vein filled with clotted blood, and one artery can be seen plugged in a similar manner, but the other vessels are empty, or with only fragments of clot in them.

Stomach.—The most marked changes are in the stomach. The sections are through the lower end of œsophagus and adjacent part of stomach. The œsophagus is healthy. The proper glandular structure of the mucous membrane of the stomach is nearly entirely destroyed by cellular infiltration, evidently inflammatory in nature. At places, some traces of the glands are still to be seen, while in other parts these structures are entirely destroyed. The exuded cells are mostly round, some oval or oblong, and all stain deeply. The infiltration extends the whole depth of the mucosa and submucosa, and in some places reaches even far into the muscular coat of the stomach in an intense form, but for the most part it can be seen to be much less in degree as it continues down between the bundles of muscular tissue. There are one or two spaces in this inflamed mucosa suggesting that here the process has gone on to suppuration. There is no distinct evidence of thrombosis ; the condition may fairly be called *Acute Croupous Gastritis.*

There is no appearance of fat embolism in any organ examined.

Remarks.—In November 1878 Mr. Barker read before this Society a paper on a case of antiseptic osteotomy of the internal condyle of the femur, in which death occurred very quickly from septic pneumonia. If further evidence

DESCRIPTION OF PLATE.

FIG. 1.—Section of the cortex of the kidney, showing inflammatory exudation. *a*, renal tubes; *b*, malpighian body; *c*, artery; *d*, compressed renal tube.

FIG. 2.—Section of a portion of lung, showing inflammatory exudation. In the centre the process is so intense that some of the cells have been lost from softening of intercellular material; near this, is a small thrombosed vessel.

FIG. 3.—Part of a section of the stomach, showing the mucous membrane, the glands entirely destroyed by a small-celled, inflammatory, exudation, softening down in the centre.

The sections were magnified by Hartnack, eye-piece No. 3, obj. No. 8.

Fig 1

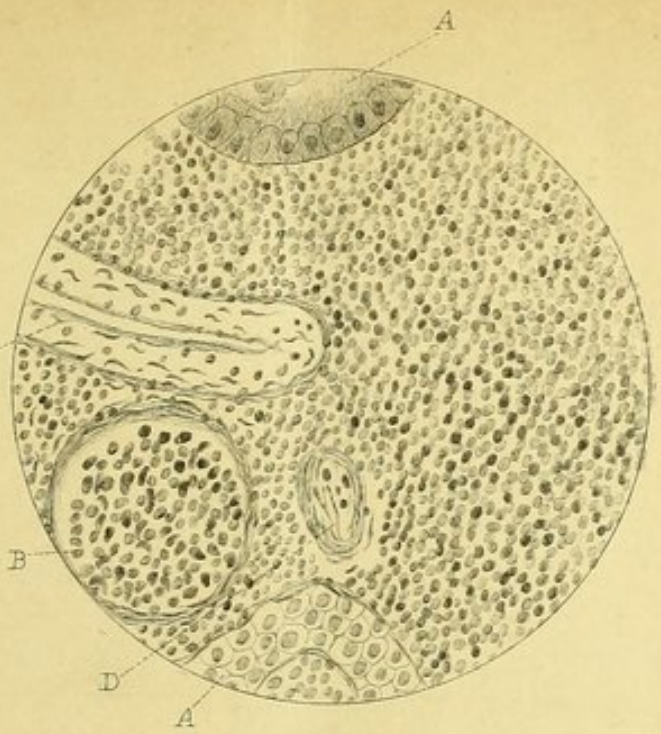

Fig 2

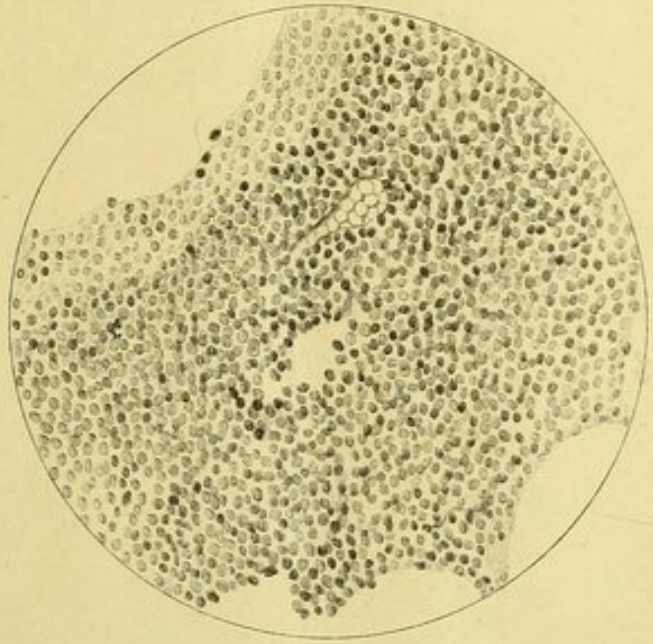

Fig 3

Montern Bros Lith

were needed to show that osteotomy, even when performed with our present antiseptic precautions, is not devoid of danger and that to life, my case affords it. And this reason, and the fact that the cause of death is either obscure or else a condition not generally sufficiently recognised, and the remarkable pathological changes found, have induced me to bring the facts under the notice of this Society.

We may at once exclude as the cause of death shock, the immediate and remote effects of the anæsthetic, either of the acute specific diseases, erysipelas, pyæmia, and fat embolism. This last was especially considered, but there was no crushing of the medulla of the divided bone such as would give rise to it, nor was there any sign of this condition in the vessels visible to the naked eye nor in microscopic sections, while such an accident would not explain the extraordinary pathological conditions present. As regards the different forms of septic poisoning, and adopting the classification of the Committee of the Pathological Society, it is evident that time alone excludes septic infection and pyæmia, not to mention the absence of sufficient primary and secondary changes. On the other hand, the vomiting, diarrhœa, and collapse are symptoms present in septic intoxication, but this supposition is negatived: 1st, by the presence of true inflammation of the stomach, lungs and kidneys; 2nd, by the absence of general visceral congestion and capillary hæmorrhage, especially beneath the pleura, pericardium, and endocardium; 3rd, by the absence of any source of such intoxication, for which a large dose of the poison rapidly introduced is necessary; 4th, by different clinical signs, notably the low temperature, and death from the failure of respiration rather than of the heart.

As regards the ingestion of any irritant poison, I have ascertained that this patient did not take anything but the simple food given by the nurse in charge, which several other patients shared without any ill result; he was under the care of an extremely careful and efficient nurse, so that we may with certainty exclude this cause. But there evidently was some irritant poison circulating in the blood, and the question arises whether it may have been carbolic acid absorbed from the wound. The answer to the question naturally divides itself into a consideration: 1st, of the symptoms; 2nd, of the pathological changes found after death.

The most constant and best known of all the symptoms of carbolic intoxication is a peculiar condition of the urine,

causing it to assume an olive-green colour either when passed or after exposure to the air. The urine passed the morning after the operation appeared normal, and was unfortunately not preserved, and none was afterwards excreted. This suppression of urine has not hitherto been recorded in carbolic poisoning, but is not inconsistent with our knowledge of the known influence of this poison upon the kidney, while the minute changes found in the kidney, coupled with the copious and oft repeated vomiting, not only of the food taken but of large quantities of watery fluid as well, afford some, though not an entirely satisfactory, pathological explanation of the occurrence. Next to this peculiar change in the urine vomiting is the most characteristic symptom of the toxic action of carbolic acid, and the vomited matter is abundant and watery, as was the case with this patient. Diarrhœa has been less often noted, but the fact of the passage of loose orange-coloured stools in cases of poisoning by the mouth has been noted.

The nervous symptoms, viz. the pallor, collapse, muscular rigidity, restlessness, loss of consciousness, and delirium, are just those observed in cases of carbolic poisoning; there was no twitching as has been sometimes noted, and the coma did not come on so rapidly as in many cases. The effect of the poison upon the cardiac and respiratory functions was very marked and special. The pulse was quickened and weakened, but under active stimulation the heart recovered its power to some extent, and the empty condition of the left side of the heart shows that death did not occur from failure of cardiac action. On the other hand, respiration was very rapid and distressed, although air entered both lungs freely and the patient lay flat on his back; and this was unaffected by stimulating treatment; death was preceded, however, by choking, gasping respiration, and the post-mortem evidence is convincing that the immediate cause of death was asphyxia. In artificial carbolic intoxication in animals Husemann and Küster have observed that the animals died asphyxiated, and that when the thorax was opened directly after respiration ceased the heart was found to be still contracting. The pupils have been noticed contracted in some cases, in others dilated, and Küster states that the characteristic symptom is a more or less complete loss of mobility of the iris—whether this was so in the present case was unfortunately not observed. The temperature was normal before the onset of the acute symptoms,

and then quickly rose to 100·2°, and remained at that height until just before death, when it fell to 99·6°. Küster found that in animals carbolic acid caused a rise of temperature followed by a fall just before death, except where the quantity of the poison was so great as to produce almost instant death, when no initial rise was noted. Sweating and ptyalism are the only other symptoms observed in carbolic intoxication; they were neither of them present in this case. In the main, then, the symptoms correspond closely with those recognised as characteristic of carbolic intoxication from external application of the acid.

In turning to a consideration of the pathological changes, we are met with the difficulty that, excluding the direct effect of the poison upon the stomach, &c., when taken by the mouth, nothing is known of its effects except the naked eye changes upon the blood. In recorded cases the viscera appeared healthy as in this instance, but, unlike it, were not examined microscopically, so that we can draw but few comparisons. The blood in carbolic poisoning has been described as dark in colour and all fluid, or but very imperfectly coagulated. These facts were noted in the case under consideration. But as the boy died asphyxiated there is nothing peculiar in the colour of the blood, and it is quite possible that the deficient coagulability of the blood and the great congestion of the trachea and bronchi, with excess of mucous secretion, are to be attributed to the asphyxia rather than to any special action of the poison. The thrombosis of the minute pulmonary vessels, and of a few of those of the kidney, as well as the inflammation of the stomach, lungs, and kidneys, has not been recorded before, but its absence has never been proved, for no microscopical examination of the apparently healthy organs has been made. But there are some facts that may with advantage be referred to here, as they give some support to the view that similar changes have been produced in other cases of undoubted carbolic intoxication. The first is the frequent occurrence of vomiting, dysphagia, and furred tongue; indeed, several observers have stated that these symptoms were explained by supposing that the acid caused gastric catarrh, although Küster opposes this view and attributes them to a central nervous influence. A similar chain of reasoning applies to the respiratory difficulty. But more to the point are the facts that in a case of Dr. Machin's (*British Medical Journal*, 1868, I.), where the acid was accidentally

applied to the skin, symptoms of congestion of the lung were noticed before death (no autopsy was made), and that pneumonia has occurred in several cases of poisoning by the mouth; of course the latter cases are open to the objection that the pneumonia resulted from inhalation of the irritating vapour. The special influence of carbolic acid upon the urine has long been known, and an action upon the kidney is not surprising, but more than that, Salkowski found albumen and renal epithelium in the urine in two undoubted cases of carbolic intoxication, while the urine has been found albuminous and even bloody in rabbits poisoned by the acid, and such a fact is almost as good as a microscopical demonstration of the inflammation of the kidneys. From a consideration of the pathology it seems right to affirm that some of the changes found coincide with those hitherto described, while the others harmonise with our knowledge of the effects of carbolic acid. It might be urged that the quantity of carbolic acid absorbed must have been too small to produce such effects as we have described. It must be remembered, however, that children are especially intolerant of carbolic acid, and that individual peculiarity plays a far larger part in the effects produced by a dose of carbolic acid than is the case with most poisons, and we have no evidence to show the minimum fatal dose of the acid in man. As typical examples of the peculiar susceptibility to the toxic effects of the acid in some people, I may note the case of a lady who was repeatedly made to vomit by dressing a small ulcer of the leg with carbolic lotion; a case of an abscess dressed with carbolised oil (1 to 8), where discolouration of urine, vomiting, and dysphagia occurred after each dressing (Wallace, *British Medical Journal*, 1870, I.), and Küster mentions a case where the temperature rose after each dressing. It may be asked why the symptoms did not come on earlier. The answer is that in all probability the absorption occurred from the skin rather than from the wound; a longer interval still has been noticed in a case of excision of the elbow dressed with carbolic lotion 1 to 50 on bandages, where the symptoms, which were very characteristic, did not come on for 72 hours. (Lightfoot, *British Medical Journal*, 1870, I.)

I would submit therefore that death must be attributed in this case to the poisonous effects of carbolic acid absorbed by the skin from the gauze dressing round the leg: 1st, because of the absence of any other cause of the symptoms; 2nd, because the symptoms and pathological changes, with

but few exceptions of no great moment, were those observed in other cases of carbolic intoxication; 3rd, because there are no facts to negative entirely this supposition.

Although the minute changes observed in the stomach, lungs, and kidneys, require independent confirmation, they suggest certain points in relation to this form of intoxication. I think we have decided evidence that carbolic acid, even when absorbed through the skin of the most distant part, has yet a tendency to act upon the mucous membrane of the stomach and set up more or less severe catarrh, in this respect being allied to arsenic. It seems to negative Küster's view that the vomiting, dysphagia, &c., are entirely due to central irritation—more probably one element, at least, in their causation is the gastric catarrh. Küster has explained the respiratory difficulty as due to irritation, and then paralysis of the respiratory centre in the medulla oblongata. Others have maintained that the increased frequency of respiration is in part caused by irritation of the terminals of the vagus, and this view is confirmed by finding minute inflammatory changes in the pulmonary parenchyma. That the fatal result was due to the influence of the acid upon the nervous system, and in particular upon the respiratory centre in the medulla oblongata, I suppose cannot be denied, though the evil effect of the extensive capillary thrombosis in the lungs must have been immense.

The proper treatment for the case would have been to remove the dressing and wash the limb thoroughly immediately the vomiting occurred, and on looking back it appears more than probable that had that been done it might have been successful in saving life.

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