Remarks.—I think the partial success of this case was mainly due to the fact that the girl was lying in bed when rupture took place, and that she remained on her back until after the distension of the intestines had shut off most of the extravasated matter from the lower peritoneal cavity. Some food undoubtedly escaped into it, and there was evidence of mild peritonitis there, both before and after the operation. As bearing on this point, I will mention a case which came under my observation, in which a woman was walking when rupture of a gastric ulcer took place, and great difficulty in diagnosis arose from the fact that, notwithstanding my leading questions, she persisted in her statements that the seat of first pain and of greatest pain and tenderness was in the left iliac region. There was no history of gastric trouble beyond slight dyspepsia of an indefinite character, and there was the misleading positive history of a lump situated in the left iliac region, which had been noticed to "gradually increase in size until it reached that of an orange." I had made preparations to cut down upon this part with vague ideas of finding a ruptured abscess or ovarian cyst (though the possibility of its being due to ruptured gastric ulcer was still entertained), when the patient got beyond all help and died rapidly. Indeed, Mr. Truman, who kindly acted on my responsibility, was actually giving chloroform just before death took place. She was then fully conscious, and her pulse was 150. I thought the anæsthetic might diminish the effect of the shock, but it had no such result. I am glad, however, to see that my action in this case has its precedent, for Mr. Taylor of Birmingham¹ operated with success upon a patient who was apparently sinking, the lips being livid, the extremities cold, and the pulse uncountable. After death I found an ulcer on the lesser curvature, but the site of intensest peritonitis was in the left iliac fossa, and it was evident that as the woman was upright when rupture had taken place, the contents of the stomach had fallen to the lower part of the abdomen.

I could not account for the parovovitis, for it was not suppurative, it was bilateral, and there were no other signs of septic poisoning, except perhaps the temperature, which remained over 100°. But Dr. Moody-Ward referred me to an interesting paper by Mr. Stephen Paget,² in which he shows that parovovitis is a rare complication of pelvic and abdominal disease, and may occur independently of septic causes. Mr. Taylor's is the first successful case recorded of operative treatment of ruptured gastric ulcer. The incision was from the umbilicus to the pubes, and the stomach was left alone. The patient survived, not only the operation, but septic pneumonia, subdiaphragmatic and pleural abscesses, and septic intoxication from insanitary surroundings, and died ultimately of intestinal obstruction, the result of the rupture, just two months afterwards. Mr. Bartlett,³ of Birmingham, operated upon a case of Dr. Saundby's, but without success, but a successful case has recently been recorded by Mr. Gilbert Barling.⁴ Dr. Sinclair's⁵ case died on the sixth day after an operation by median incision below the umbilicus. Dr. Dreschfeld⁶ recommends a preliminary puncture with a Southey's trocar, as a means of diagnosis, to ascertain by its odour if the extravasated gas comes from the stomach or intestines. He points to the fact that vomiting is almost invariably absent in this form of perforation, and mentions a case of Czerny's⁷ in which the operation was performed by median epigastric incision five days after perforation, and death resulted from fresh peritonitis in four days. Mikulicz⁸ reports an extraordinary case in which gas, smelling strongly of alcohol, escaped through a cannula which was pushed into the abdominal cavity for diagnosis, and the abdomen was opened first from the navel to the symphysis, and the incision was afterwards extended to the xiphoid cartilage. A clean rupture was then found along the lesser curvature, apparently the result of the bursting of an overdistended stomach, without previous ulceration. Death took place in three hours. Billroth⁹ performed gastrorrhaphy for gunshot wound of the stomach, but death ensued in twenty-

eight hours. Dr. Parsons¹⁰ stitched the margins of a ruptured ulcer to his abdominal incision. Death resulted in six days. He mentions two cases of traumatic perforation of the stomach in the practices of Professor Kocher and Dr. Ball respectively, in the former of which recovery took place.

THE DIRECT PYRAMIDAL TRACTS OF THE SPINAL CORD.

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THE BRITISH MEDICAL JOURNAL of April 5th, 1890, contains a leading article on the functions of the above tracts, and also the notes of a most interesting case, recorded by Dr. Macewen, which he regards as giving some support to the theory that the function of the direct pyramidal tract is to convey motor fibres to the arms and probably to the intercostal muscles.

In Professor Macewen's case "the intrinsic muscles of the forearm, wrist, and hand were absolutely paralysed." There was paresis of the supra- and infraspinati, deltoids, biceps, brachiales antici, and supinators. The legs were not affected. Macewen localises the lesion in the cord at the region of the seventh and eighth cervical and first dorsal, and suggests that the lesion was one implicating the anterior (or direct) pyramidal tracts at this region, and that the crossed or lateral pyramidal tracts were unaffected. He thinks the symptoms may be explained by accepting the theory that the direct pyramidal tracts are chiefly related to the innervation of the arms.

The direct or anterior pyramidal tracts (columns of Türek) consist of fibres which have not decussated at the medulla; the fibres, which have decussated passing down the crossed pyramidal tracts. The direct pyramidal tracts gradually diminish from above downwards, and as a rule cease about the middle dorsal region, but sometimes pass down to the lower dorsal region. Hence, as pointed out by Tooth, this tract must have some other function in addition to the innervation of the arms. Macewen points out "that the direct tracts generally continue down the cord to the middle of the dorsal region, where the anterior tracts would terminate did they supply the principal respiratory intercostals." He suggests that in addition to the innervation of the arms they may also innervate the intercostals.

Some time ago, through the kindness of the late Dr. Ross, I had the opportunity of observing clinically and of making the pathological examination in a case of spinal cord disease, in which the direct pyramidal tracts were markedly degeneration at the part at which Macewen placed the lesion in his case, and yet the arms were not paralysed.

J. T., aged 39, was admitted under the care of Dr. Ross at the Manchester Royal Infirmary, July 19th, 1890.

Previous History.—About two years before admission the patient first noticed slight numbness in the fingers of both hands, then the legs became weak; they gradually became worse, and five or six months before admission he was compelled to give up work on account of loss of power in his legs. There was no history of cramps in the legs, pains in the back or limbs. The bladder and rectum had not been affected. There was no history of alcoholic excess, syphilis, or diphtheria.

State on Admission.—There was a little œdema in both legs. The patient was quite unable to stand. He was unable to raise the legs in the extended position from the bed. He was able to flex and extend the legs at the hips and knees very slightly. There was only the slightest movement when he attempted to dorsiflex the feet. The knee-jerks were absent, and there was no ankle clonus. The plantar reflexes were feeble. There was no rigidity of the legs, and no pain in the legs except when they were moved. He was able to feel and localise the touch with a pin's head (if the pressure were moderate), and was able to distinguish between the head and the point of a pin on both legs. A very slight touch with a pin's head was often not recognised. The patient was able to perform all the movements of the arms with the exception of abduction at the right shoulder. This he was unable to perform on account of pain in the right shoulder-joint. He was able to feel and localise the touch of a pin's head on both hands and arms. The diameter of the pupil on the right side was slightly greater than on the left; both reacted quite well to light. Nothing of importance was detected on examination of the heart, lungs, abdomen, and urine.

At a later date there was incontinence of urine, and this continued until his death. Cystitis occurred, and a small bed sore formed over the sacrum. The paralysis of the legs persisted, and slight anaesthesia occurred in the legs, but there was no paralysis of the arms. The knee-jerks were absent up to the last. There was no ankle clonus, and the plantar reflexes were absent. The patient became delirious, and passed into a semi-conscious condition. Death occurred on October 12th.

BRITISH MEDICAL JOURNAL, 1886, vol. i, p. 183.

² *Birmingham Medical Review*, 1888, p. 155.

³ *Birmingham Medical Review*, 1888, p. 183.

BRITISH MEDICAL JOURNAL, ii, 1892, p. 1011.

⁵ *Med. Chron.*, 1889, May.

⁶ *Med. Chron.*, 1886, November.

⁷ *Berlin. klin. Woch.*

⁸ *Volkmann's Sam. klin. Vort.*, p. 2, 310, 1885.

⁹ *Magenoperationen*, 1880 85.

¹⁰ *Dub. Med. Journ.*, vol. ii, p. 26, 1892.

The friends gave permission for *post-mortem* examination of the spinal cord only. The spinal canal was normal; there was no evidence of caries or of any cause of compression of the cord. The spinal dura mater was normal. The upper dorsal region of the cord was softer than the other parts. No other changes were detected on naked-eye examination. After hardening in Müller's fluid, pieces of the cord were embedded in collodion, and the sections stained according to Weigert's method, and with aniline blue black and logwood. Microscopical examination showed an area of transverse myelitis in the upper dorsal region of the spinal cord. The white matter was chiefly involved. Above the lesion, in the uppermost part of the dorsal region and in the cervical region, was the usual ascending degeneration in the direct cerebellar and posterior median columns. In addition to these usual tracts of degeneration, there was degeneration of each anterior or direct pyramidal tract for about 4 inches above the lesion (Fig. 1). In the upper third of the cervical region, the direct pyrami-

Fig. 1.

dal tracts were not degenerated, but commencing about the junction of the upper and middle third of the cervical region there was marked degeneration of the direct pyramidal tracts. This extended down to the area of transverse myelitis in the upper dorsal region. There was marked degeneration of these direct pyramidal tracts, therefore, in the lower two-thirds of the cervical and in the upper dorsal regions. Above the transverse myelitis, in the uppermost dorsal and in the whole of the cervical region, there was no affection of the crossed (lateral) pyramidal tracts. The degeneration in the direct pyramidal tracts could be detected quite readily on naked-eye examination of the cord after hardening in Müller's fluid, this and other degeneration tracts being of a paler colour than the rest of the white matter. It was seen best in sections stained according to Weigert's method; it was also seen in sections stained with aniline blue black and logwood. The degenerated tracts showed the usual excess of neuroglia connective tissue and the disappearance of nerve fibres. The degeneration was very well marked, and quite as distinct as that in any other part of the cord. Below the area of transverse myelitis there was

Fig. 2.

the usual descending degeneration in the crossed pyramidal tracts; there was also degeneration in the direct pyramidal tracts down to the lower dorsal region (Fig. 2).

In the posterior median and posterior external columns there were numerous small irregular patches of degeneration, most marked just below the transverse lesion, and gradually diminishing towards the lower part of the cord. They extended to the upper lumbar region.

At the uppermost part of the degeneration in the direct pyramidal tracts. At the junction of the upper and middle thirds of the cervical portion of the cord the changes extended laterally into the white matter, a little beyond the limit of these tracts. It seems probable that at this level there was originally a small patch of myelitis at the anterior part of the cord, just in the region of the direct pyramidal tracts, and that this had been followed by descending degeneration in the direct pyramidal tracts. Any such area of myelitis must have been very small, and strictly limited to the region just mentioned. There was no descending degeneration in the crossed pyramidal tract of the cervical region. That the direct pyramidal tracts had their normal position is shown by the fact that they were degenerated in the usual way below the area of (dorsal) transverse myelitis down to the lower dorsal region.

In this case the direct pyramidal tracts were markedly degenerated from the junction of the upper and middle thirds of the cervical region down to the lower dorsal region, and yet the arms were not paralysed: destruction of the fibres of these tracts in the parts just mentioned was, therefore, not followed by paralysis of the arms.

PRELIMINARY NOTE UPON A NEW METHOD OF PREPARING THE SPINAL CORD FOR MICROSCOPICAL EXAMINATION.

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In attempting to prepare sections from the fresh spinal cord for microscopical examination I have hitherto met with disappointment. As far as my knowledge goes, attempts of the kind have always failed. My own experience is that sections can be made from the ether-frozen fresh cord, and floated on water with comparative ease, but that they become quickly spoiled by further manipulation. If they are treated after the method employed for the fresh cortex cerebri¹, the tissue composing the white matter is thrown into a number of folds, and the sections are thereby rendered useless. But a still graver defect, and one which effectually condemns this procedure when applied to the spinal cord, is the complete absence of fixation of the elements of the white substance. The ordinary appearance of nerve tubes in transverse section is wanting; in place of it one sees a tangled skein of connective tissue fibres and axis cylinders, the latter especially being twisted and corkscrew shaped. There are literally no nerve tubes to be seen. As regards the grey matter, the nerve cells are well stained, and considerably larger than in a hardened specimen, but other tissue elements appear but indifferently.

The following are briefly the steps of the method now adopted:—

1. Remove spinal cord from a recently-killed animal.
2. Place a portion 6 to 8 millimetres high on the ether-freezing microtome; freeze and cut. The precautions used in freezing the brain cortex to be observed here also.
3. Float the section on to water. It should be quite free from wrinkles; if allowed to sink beneath the surface they appear at once.
4. Take the section up as soon as possible with a perforated lifter, drain off excess of water on blotting paper, and float the section on pure piridin,² kept at hand in a watch glass; it may presently be pushed beneath the surface of the liquid. Hitherto I have kept sections in the piridin for at least one hour, usually several hours. In one case one quarter of an hour sufficed to fix the tissue elements, so that the time above mentioned is, perhaps, unnecessarily long.
5. Wash well in water.
6. Stain.
7. Dehydrate and clear in piridin (possibly desirable to pass through piridin diluted with water into the pure reagent).
8. Mount in balsam suitably thinned with piridin.

In this way sections free from wrinkles are obtained. It certainly may occur that out of a large number of sections some present a slight fold at the point of exit of one of the nerve roots, but most obtained are quite free from wrinkle. Such sections are instructive, even unstained, examined simply in water upon the slide. The horns stand out in relief against the darker white substance, and the nerve roots are clearly seen, sweeping outwards in broad bundles. Within the horns appear large colourless ganglion cells, and an intricate meshwork of fine nerve tubes. All these structures approach very nearly the natural size. Such fresh specimens would probably show tract degenerations well under the low power. In stained specimens the transverse sections of the nerve tubes in the white substance are brought out exceedingly well; the contour of the tubes is sharp, the axis cylinders are well stained. The appearance of the white columns is such as one is accustomed to see in sections from hardened cord; the effects of corrugation are, however, absent, and the wealth of structure is greater than in the latter case. Any of the dyes ordinarily used for the purpose may be employed for staining the axis cylinders and connective tissue of the white matter, but I have mostly used anilin blue-black ($\frac{1}{4}$ per cent. aqu. sol.), followed by picrocarmine, as I find the combination more effectual than any single stain in bringing out all the structures in the section (grey and white matter). Kernschwarz, diluted to one-third its original strength (5 to 10 minutes), is an excellent stain for the axis cylinders; nerve cells it stains rather too uniformly. By using subsequently the Biondi-Ehrlich stain (10 minutes) they are brought out better. An entirely satisfactory staining of the grey matter is yet to be obtained. By

¹ Bevan Lewis.

² First recommended, I believe, by De Souza (*Zeitschr. f. wiss. Mikroskopie* Bd. v, H. 1.) as a hardening reagent for brain substance.