sutures may then be inserted at various points, or a continuous Lembert's suture may be used all round if that be thought necessary. It is wise to scarify the peritoneum of the intestines with a needle for about half an inch round the seat of union. This scarifying promotes the exudation of lymph, which acts as a callus (just as in a fracture of bones) round the ends to be united. Especially should this be done if no Lembert's sutures have been employed. The intestines when united cannot move in either direction, for the sutures, which are tied up tightly at the narrowest part of their

FIG. 6.

respective cones, naturally cannot slip up to any broader portion; moreover, the bobbin itself cannot possibly alter its position either upwards or downwards.

Murphy's button and Mayo Robson's button are sometimes blocked by solid faeces which have to travel through them for some distance (about an inch). Now, since my bobbin is shaped like an hour-glass, there is an extremely small surface to obstruct the passage of solid matter; this may be represented as being merely the extent of a line—i.e., at the meeting of the apices of the cones. This is quickly passed, and then the cones grow wide once more.

FIG. 7.

Simplicity is, I think, the characteristic of the bobbin. No complicated suturing is required, nothing being needed except the continuous sutures—one round one end of the divided intestine, and the other round the other end—or, in a similar manner, round the incisions, one in one viscus and the other in the other. Hence it can be applied very rapidly. Further, it cannot slip, and in consequence of its shape the parts to be joined are brought closely together. Lastly, it is absorbed in time and therefore cannot act as a foreign body. I have experimented many times with the bobbin on the dead body and find it is most easy of application, and the operation rapidly carried out. At present I have had only one opportunity of using it upon the living subject, and in this case with excellent results.

Grosvenor-street, W.

HEAVY FINES FOR ADULTERATING MILK.—At Southwark Police-court, on Tuesday, Aug. 27th, David Jenkins, of Guy-street, Bermondsey, and Joseph William Bramley, of Weston-street, Bermondsey, were summoned under the Sale of Food and Drugs Act, at the instance of Mr. Henry Thomas, inspector to the Bermondsey Vestry, for having unlawfully sold milk adulterated with added water to the extent of 19 and 18 per cent. respectively. The cases having been proved, Mr. Fenwick fined Jenkins, against whom there were previous convictions, £20 and 12s. 6d. costs, and Bramley £3 and 12s. 6d. costs.

SPINAL SOFTENING LIMITED TO THE PARTS SUPPLIED BY THE POSTERIOR ARTERIAL SYSTEM OF THE CORD.

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THE spinal cord is supplied with blood at its anterior part chiefly by a long narrow artery, the anterior spinal, and at the posterior parts by two long narrow vessels, the posterior spinal arteries. These arteries are reinforced by numerous small arterial twigs coming from the intercostal lumbar and sacral arteries, which enter the spinal canal by the intervertebral foramina and run along the anterior and posterior nerve roots. A number of small arteries, derived from the vessels on the surface, enter the substance of the spinal cord: the largest of these are the anterior median arteries, which penetrate the anterior median fissure. The exact distribution of these branches within the cord is described in most anatomical text-books. Now the blood supply of the cord may be divided broadly into the anterior and posterior arterial systems; and a transverse section of the cord may be divided roughly into two districts or areas—one supplied by small arteries coming from the anterior arterial system and the other by arteries from the posterior system.¹ The blood supply to the interior of the cord by the anterior spinal arteries is more important than that by the posterior arteries. The distribution of the lesions in a number of spinal diseases is related to the vascular supply, as I have pointed out elsewhere.² A spinal cord which I have recently examined furnishes a beautiful example of the localisation of the lesion to the part supplied by the posterior arterial system. During life the patient was under the care of Dr. Graham Steell, to whose kindness I am indebted for the opportunity of making the pathological examination.

A man fifty years of age, suffering from paraplegia, was admitted as an in-patient to the Manchester Royal Infirmary on Aug. 29th, 1894. His previous health had been good until three weeks before admission, when he suddenly lost the use of his left leg immediately before getting into bed in the evening. He had just passed urine and was pulling off his stockings when he found that he was unable to raise the left leg. He thought that the right leg became paralysed soon afterwards. There was no pain in the back or limbs at the onset, but the legs felt numb. He stated, however, that he could feel when anything touched his legs. Very soon after the onset of the paralysis the legs became painful, especially the left, and there had been dull, aching pain, with occasional sharp, shooting pains, in the legs ever since. Retention of urine had been present from the onset and a catheter had been passed daily. The legs had remained completely paralysed from the commencement. The arms had not been affected, and there had been no pain in the back. There is no history of syphilis or gonorrhoea or rheumatism. With the exception of two attacks of pleurisy (one eight years and the other twenty years before admission) the previous health has been good, until one month before the onset of the paralysis. At that date he began to feel languid, but was able to attend to his work quite well.

On admission to the infirmary the patient was somewhat wasted. Both legs were completely paralysed, and the feet and toes were "dropped." The legs were flaccid; the knee-jerks were present; there was no ankle-clonus; and the plantar reflex was present on the left side, but was not obtained on the right. The abdominal, epigastric, and cremasteric reflexes were not obtained. A light touch with the head of a pin was not felt on the right leg; a pin prick also was not felt. On the left leg both the point and head of a pin were felt and localised. On the abdomen the head and the point of a pin were not felt on the lower part—up to a point midway between the pubes and umbilicus; above this level both were felt distinctly. There was no zone of hyperaesthesia just above the anaesthesia. The patient could

¹ See Marie: Diseases of the Spinal Cord.

² See the Medical Chronicle, Dec., 1894, and Jan. and Feb., 1895.

distinguish hot and cold test tubes on both legs. The bladder was distended; there was retention of urine, with dribbling. There was also incontinence of fæces. There was no pain in the back, but the patient had considerable pain in the legs, especially when moved. The first lumbar vertebral spine was very slightly prominent, but there was no tenderness on percussion at this region. The arms were unaffected. There was no paralysis of the facial, ocular, of tongue muscles. When the edge of a silver coin was drawn firmly over the skin in any part of the trunk or limbs a peculiar bluish-black mark remained, and the patient's initials could be written quite easily on the chest and abdomen. The pulse was 105 and intermittent; one beat was missed in about every 10. The tension was normal, and the artery not atheromatous. There was a short systolic murmur limited to the apex, but the cardiac dulness was normal. Physical examination of the lungs and abdominal organs revealed nothing abnormal. The urine was dark in colour, ammoniacal, and very foetid, the specific gravity being 1017. It contained a small amount of albumin and a deposit of triple phosphates, a few pus cells, mucus, and a few red corpuscles. By the end of October the anæsthesia had increased, and both legs were equally affected. The anæsthesia was most marked on the outer side of the leg, but, owing to the patient's dull mental condition, it was not possible to map it out accurately. At a later date the results of examination of the cutaneous sensibility were quite unreliable. Soon after admission a sacral bed sore developed and the general condition of the patient became gradually worse. He complained most of pain in the legs, which was very severe when the limbs were moved. The doubtful prominence of the first lumbar spine did not increase, and spinal tenderness did not develop. The reflexes remained the same as on admission, but the bladder symptoms and the bedsores became much worse, and the patient died on Jan. 2nd, 1895.

Post-mortem examination.—The body was found to be greatly emaciated. There was an extensive sacral bed sore. The pericardium was normal. Fibrous pleuritic adhesion was present on the left side. There was slight thickening of the cusps of the mitral valve. Tuberculous nodules were scattered throughout the right lung. The stomach, intestines, liver, kidneys, and spleen presented no important changes. The bladder showed well-marked signs of cystitis. The spinal column presented no signs of angular curvature or caries. There was no evidence of tumour growth in the vertebræ. The spinal cord appeared to be normal to the naked eye. On section the grey matter in the lumbar and lower dorsal regions was seen to be congested. The spinal dura mater was adherent in the lower dorsal

Section of spinal cord (Weigert's stain). Black dots and lines=healthy nerve fibres; the blank area=softening and degeneration.

and lumbar regions to the posterior surfaces of the vertebræ; otherwise the spinal meninges were normal. After hardening in Müller's fluid, the cord, on transverse section, presented to the naked eye well-marked degeneration in the posterior and lateral columns of the lower dorsal region for about two inches. These columns were much lighter in colour than the rest of the transverse section. Above and below the region just mentioned the usual secondary degeneration could be detected. The whole of the lower two inches of the dorsal region of

the cord was cut into small pieces, which were embedded in celloidin, and sections made. Every third or fourth section was mounted and examined microscopically. Practically, therefore, the whole of the cord at the seat of the lesion was examined microscopically. The sections were stained according to the methods of Weigert and Pal, and also with logwood and eosin and with aniline blue black. The figure represents the appearance of a transverse section (stained according to Weigert's method) through the lowest dorsal region, at the part where the lesion was most intense. The black dots represent healthy nerve fibres in the white matter, the fine lines represent the small nerve fibres in the grey matter, and the blank part corresponds to the area of degeneration in which nerve fibres are very scanty or altogether absent. (In the sections stained according to Weigert's method this blank area is light brown.) In the figure nerve fibres are represented (by fine black lines) in the posterior horns of grey matter, but in a good number of sections the posterior horns were completely degenerated, and their black-stained nerve fibres absent. The degeneration affected the posterior columns, median and external, with the exception of a narrow zone, close to the grey matter of the commissure and to the neck of the posterior horn; it affected the region of the lateral pyramidal, the direct cerebellar, and the antero-lateral ascending tracts, and also extended a little farther forwards on the surface of the cord. Under a high power the part where the changes were most intense—i.e., the whole of the posterior columns, with the exception of the narrow zone adjacent to the grey commissure and to the neck of the posterior horn—was seen to be densely infiltrated with round cells—large mononuclear cells, compound fat granular cells, and leucocytes. The nerve fibres were absent, and only a few irregular fibres of neuroglial tissue were seen amongst the cell infiltration. The microscopical appearance was that of spinal softening. The bloodvessels were dilated and their walls studded with nuclei, but the vascular changes were certainly not so marked as is usually the case in so-called myelitis. A large portion of the degenerated area in the lateral columns presented similar appearances of softening (cell infiltration, &c.). In some of the sections these changes had extended into and destroyed the posterior horns of grey matter. In other sections at this level of the cord the changes were more chronic than those just described, but involved the same area in the posterior and lateral parts of the cord. The nerve fibres were scanty, or absent altogether; the neuroglia was greatly increased and presented round or oval spaces, from which the nerve fibres had disappeared; and the changes then resembled those met with in secondary degeneration or sclerosis. The grey matter of the anterior horns and the intermediate grey matter appeared to be quite normal. The ganglion cells and fine nerve fibres were brought out quite distinctly—by different methods of staining. The whole of the anterior white matter was normal. In some sections small ante-mortem blood-clots were found in the veins on the posterior surface of the cord, and in a few sections they were also found in the veins near the anterior median fissure. The clots in all cases consisted of numerous leucocytes with a few red corpuscles, and were always separated by a small space from the vessel walls. No clots were found in the spinal or meningeal arteries. The walls of the bloodvessels appeared to be normal except at the seat of the changes in the posterior and lateral columns. Above the seat of the lesion just described there were the usual ascending changes in the posterior columns (secondary sclerosis), and below there was the usual descending sclerosis in the lateral pyramidal tracts. Only in the lower two inches of the dorsal region of the cord were the above-described suffering and degeneration found in the posterior and lateral columns.

Remarks.—The changes at the seat of the lesion I have described as spinal softening rather than myelitis, because the former term does not necessarily imply an inflammatory origin. The case is of interest on account of the localisation of the changes just to that part of the cord which is supplied by the posterior arterial system. If we compare the above figure (showing the distribution of the lesion) with that given by Marie (*Diseases of the Spinal Cord*) to show the distribution of the anterior and posterior arteries of the cord, we shall find that the blank area of degeneration corresponds almost exactly with the part of the cord which Marie maps out as that supplied by the posterior arterial system. The very sudden onset of the symptoms is also very suggestive that the lesion was of vascular origin.

Manchester.