

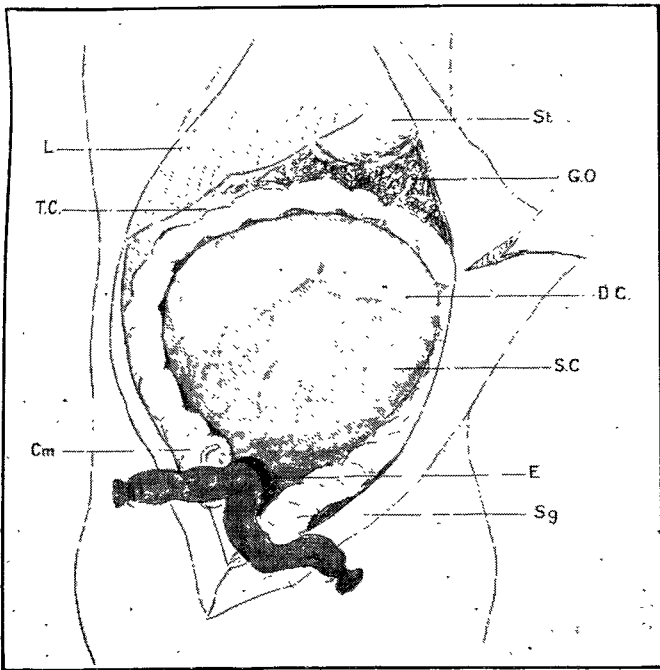
STRANGULATION BY VOLVULUS IN AN ABNORMAL SAC LYING WITHIN THE GENERAL PERITONEAL CAVITY.

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THE subject of the condition described below was a man, aged 46, who was admitted to the Leeds General Infirmary on August 3rd, 1908, in a moribund condition, and who died without any operation having been performed. He had been under the care of Mr. W. W. Walker of Batley with symptoms of acute intestinal obstruction which had begun five days before, and had resolutely refused to come into the infirmary till the day of admission. There had been no preliminary warnings of intestinal trouble previous to the sudden onset of acute symptoms.

When the abdomen was opened at the necropsy a very unusual and interesting state of its contents was disclosed. Occupying the mid-region of the cavity was a large rounded cyst or sac, through the semi-transparent wall of which distended coils of bowel could be seen. The upper coils were large and pale, and had a somewhat transverse direction; the lower were black, as if the subject of strangulation, and had an obliquely vertical arrangement. Below the cyst, the lower portion of the abdomen, which was little more than the pelvis, was occupied by coils of black small intestine which concealed the terminal portion of the large gut. Above the cyst, and encroaching on its shelving upper surface, lay the liver, and in the left hypochondrium the stomach. The great omentum was huddled up into a mass which fitted into the space between the cyst and the stomach. The colon was not visible, but it was found to encircle the cyst, except at its lowest part, and to be attached to its wall in a manner subsequently to be described. (Fig. 1.)

FIG. 1.



Appearance of sac before the parts were disturbed. L., Liver. T.C., Transverse colon. Sg., Sigmoid. E., Entrance to sac. S.C., Strangulated coils showing through sac wall. D.C., Distended coils inside sac. G.O., Great omentum. St., Stomach. Cm., Caecum.

To permit of the further investigation of parts the strangulated small bowel occupying the area below the cyst was removed. It was a single loop some three or four feet long and the two ends of the loop passed into the cyst through an aperture on its lower surface. The two ends were tied a few inches from the aperture, as represented in the illustration. The whole loop was deeply congested, of a purplish-black colour, and infiltrated with blood. It was not constricted at

the neck, and the finger could be introduced alongside the gut through the aperture into the cyst. The entrance to the sac looked downwards and was on the level of the promontory of the sacrum. It was large enough to admit three fingers. The cyst, which will now be referred to as "the sac," occupied the small intestine area within the arch of the colon. To continue the examination it was opened by a central vertical incision through its whole length which terminated by dividing the boundary of the aperture.

For the sake of simplicity the cause of the strangulation will be dealt with at once. The two cut ends of the strangulated loop were continuous with similarly affected coils within the sac. There had been no strangulation by the neck of the sac, and no signs of constriction were presented by the portions of bowel that occupied it. The greater part of the ileum was in this deeply congested state which was evidently due to a twist of the lower portion of the mesentery, but the exact mechanism was not made out, as owing to the inexperience of the manipulator the relations were disturbed and lost, and the previous removal of the loop outside the sac added to the difficulties of its demonstration. The jejunum was distended but not strangulated.

The small intestine was now removed from the upper end of the jejunum to the lower portion of the ileum. By this the whole cavity of the sac was displayed. On its posterior surface was the quite normal attachment of the mesentery. It lay entirely within the sac. At the upper part the third and fourth parts of the duodenum were in their natural position and covered as usual by peritoneum. The terminal portion of the ileum passed out of the opening to join the caecum, which lay just outside and to the right of the mouth of the sac. The ileum at this point was attached by its mesentery, which blended with the margin of the aperture on the right side. (Fig. 2.) The condition may be fairly

FIG. 2.

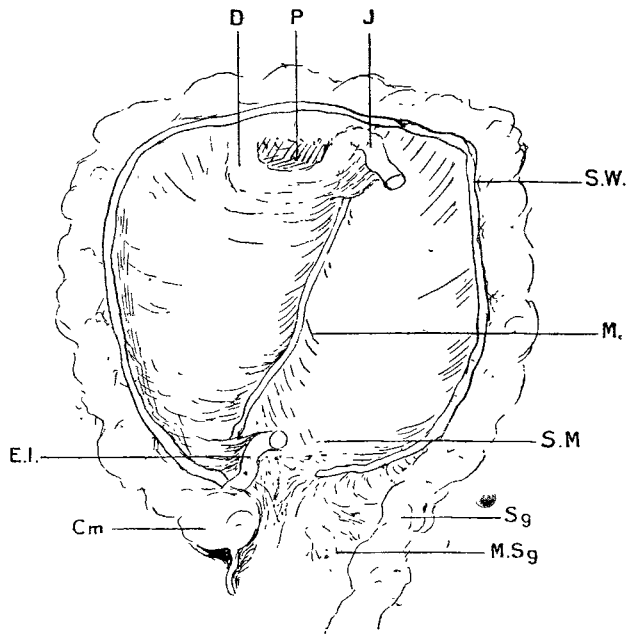


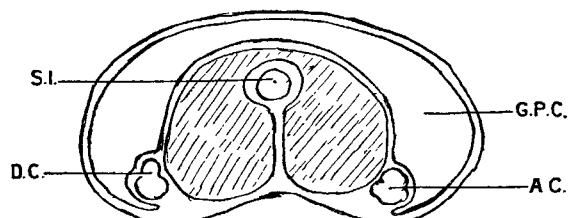
Diagram to show the posterior wall of the sac. The small intestine with its mesentery has been cut away as well as the anterior wall. J., Jejunum. P., Pancreas. D., Duodenum. E.I., End of ileum, with its mesentery. Cm., Caecum. M.Sg., Mesosigmoid. Sg., Sigmoid. S.M., Mouth of sac. M., Cut attachment of mesentery. S.W., Sac wall.

described as a second and abnormal lesser peritoneal sac, whose natural contents were the small intestine and its mesentery. Some of the contents had escaped through the orifice into the general peritoneal cavity.

The actual anatomical disposition of the peritoneum can most easily be explained by the help of diagrams. Tracing the peritoneum in a horizontal direction (Fig. 3) about the middle of the sac and beginning with the outer layer of its anterior wall it could be followed to the descending colon, which it invested without forming a mesocolon. It was then reflected on to the anterior abdominal wall and lined the parietes till it reached the ascending colon. After investing this—also without forming a mesocolon—it was continued on the anterior wall of the sac to the point from which a start was made. On the inside of the sac the serous

membrane could be traced from the inner surface of the anterior wall to the right till the posterior abdominal wall was reached, but just before this the ascending colon could be seen lying upon its exterior. Passing to the spine the small intestine was invested and the mesentery formed. The posterior abdominal wall was then covered on the left side,

FIG. 3.

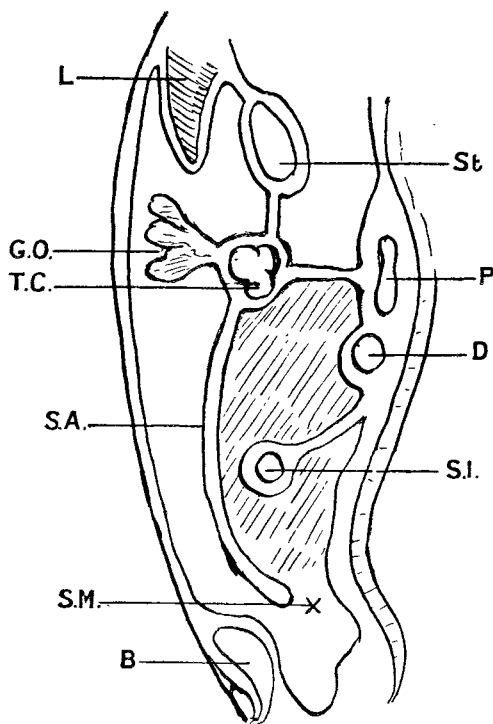


Diagrammatic horizontal section showing relation of the sac to the general peritoneal cavity. The sac is shaded. D.C., Descending colon. S.I., Small intestine. A.C., Ascending colon. G.P.C., General peritoneal cavity.

and as the peritoneum was reflected to the anterior wall of the sac to reach the point from which it started it skirted the descending colon which lay on its outer surface.

A study of Fig. 2 will make plain the way in which the terminal part of the ileum was tethered to the margin of the neck of the sac by the two layers of its mesentery which diverged to become continuous with the peritoneal covering of the aperture. A vertical section through the mouth shows how the two layers of peritoneum by which the anterior wall was formed blended at the free margin and also how the posterior peritoneal lining of the sac was continuous with the peritoneum lining the pelvis. (Fig. 4.) The neck of the

FIG. 4.



Diagrammatic vertical section passing through the aperture in the sac. The sac is shaded. L., Liver. G.O., Great omentum. T.C., Transverse colon. S.A., Anterior wall of sac. S.M., Mouth of sac. B., Bladder. S.I., Small intestine. D., Duodenum. P., Pancreas. St., Stomach.

sac contained no large vessels, but it was slightly rounded and thickened. The anterior sac wall was also destitute of vessels of any size.

It is evident that this unusual condition was in no sense a hernia, and that consequently, notwithstanding a certain suggestive similarity in appearance, the pathology must be quite different from that of the various forms of internal abdominal herniæ. I have discussed the case on several occasions very carefully with Professor J. Kay Jamieson, who has referred me to a paper by Dr. J. Cameron and Dr. J. D. Lickley,<sup>1</sup> in which is described an abnormal peritoneal sac very similar to this one, but without any aperture; and also

to the report of another case by Professor Clelland (mentioned in their paper) which would appear to have some resemblance to it. I had hoped that Professor Jamieson would have joined me in this communication and given a description of the way in which such a condition might arise in the process of development, but at the present time he feels unwilling to commit himself to an explanation which subsequent research may show to be incorrect. As the whole subject is one in which he is much interested it has been decided that the case shall be published now, and at some future date he may in a separate communication deal with the condition from the anatomical standpoint.

Personally I believe that the abnormal sac is a consequence of an irregularity occurring during the evolution of the large intestine. With a piece of rubber tubing to represent the bowel and a towel to constitute the mesentery, it is quite easy by imitating the normal rotation of the colon and the passage of the cæcum to the right side to produce a sac, such as existed in this case, either by the formation of a fold in the mesocolon and its adhesion to the posterior parietes, or by adhesion at some point of the right peritoneal layer of the mesocolon. But I must content myself with this simple statement, for the description of the manner of its production—in such a way that it could be understood—is beyond me.

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## BRADYCARDIA IN ASSOCIATION WITH URÆMIA.

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It is well known that an infrequent pulse is occasionally associated with some forms of toxæmia, particularly those which accompany catarrhal jaundice and chronic nephritis. An infrequent pulse may be due to three different causes: (1) the regular occurrence of extra-systoles not perceptible to the finger on the pulse (coupled rhythm); (2) heart block, partial or complete; and (3) slowing of the whole heart. It is only since the introduction of the graphic method of studying heart affections that it has become possible to distinguish certainly between these distinct conditions. It has been ascertained that the slow pulse of jaundice is most often due to a coupled rhythm, much less commonly to a slowing of the whole heart. As regards the pulse in nephritis, little is at present known either as to its nature or its cause. It is not yet considered as proved to be due to uræmic poisoning, and Vaquez and Esmein,<sup>1</sup> in their report on Bradycardia to the French Medical Congress last year, state that the question needs further research.

The patient, a man now aged 42 years, was first under my care six years ago with an attack of nephritis. When first seen he had already had considerable dropsy of the legs for three weeks. The urine contained a considerable quantity of albumin, granular casts, and some blood. At the time it was hoped that it was a first acute attack, but the subsequent progress makes it more probable that it was really an exacerbation of an already chronic disease. This attack was of four months' duration, and at the end of it the urine still contained a large quantity of albumin. In spite of many weeks' rest in bed, careful dieting, and the usual remedies the cedema was very persistent. Finally, he improved considerably on a more generous diet and the free administration of iron, and he was able to go into the country in April, 1906. On his return he went back to work and remained fairly well and able to perform his duties until March, 1908. Then he had an attack of "influenza" and the cedema returned. This time the disease was still more resistant to treatment than before, and he was in bed for over eight months. In spite of all the usual remedies the amount of urine often fell very low and the cedema gradually spread upwards, fluid collected in the abdominal and pleural cavities, and the breathing became at times very distressed, particularly at night. At times there was almost total suppression of urine, and for many weeks the onset of severe uræmia was feared. The exclusion of salt from the diet appeared to have little effect as regards producing diuresis. It may be mentioned here that the patient was extremely fond of

<sup>1</sup> Journal of Anatomy and Physiology, vol. xli., p. 88. The reference to Clelland's paper in this article does not seem to be correct.

<sup>1</sup> Vaquez and Esmein: Archives des Maladies du Cœur, Paris, December, 1910, p. 732