Autopsy.—The thymus was enlarged, the uterus atrophic, and the ovaries consisted of fibrous tissue only. The spleen, liver, pancreas, kidneys, and suprarenals were normal. The pituitary showed some changes thought to be secondary to inanition. "The small intestine showed marked post-mortem changes; the villi showed no structure and most of the surface portion of the mucosa had disappeared. Marked catarrh of the intestine was present, desquamated cells and mucus lay on the surface, and most of the cells of the glands showed goblet formation. The condition, in fact, was that which Herter surmised must exist." A microphotograph of the colon is reproduced.

Comment.—The author states that "the apparently primary pathological change in this case was catarrh of the colon, and probably also of the small intestine, and in consequence one may regard the case as one of undoubted intestinal infantilism." But it is not easy to accept this view. Clinically we have in this case, as I understand it, a fatty diarrhoea lasting for 13 years, and so severe as to produce a practical cessation of growth for eight years; post mortem there are found intestinal changes which are too acute and trivial in type to account for so chronic and severe a condition, and, moreover, they affect chiefly the colon, which has no part to play in fat-absorption. It is, I think, very much more plausible to regard the intestinal condition as not the primary cause of the fat-wastage, but as a secondary condition arising from result of the excess of fat in the intestinal contents and the infection by whip-worm.

If this view be accepted the origin of the excessive fat-loss in this case remains, as in my own, undiscovered by the post-mortem examination.

Conclusion.

A detailed consideration of these three autopsies in which intestinal lesions were found shows that they fail to prove that the excessive fat-loss in coeliac disease is due to an enteritis. This view is supported by other considerations. The excessive fat-loss in the stools is the essential and characteristic feature of coeliac disease. It is the first diagnostic symptom to appear, and is of severe grade (up to 57 per cent. of the faeces may consist of fat). It is altogether out of proportion to the disturbance in protein digestion. In all these features coeliac disease is unlike an enteritis.

The following recent case seems definitely to prove that the essential fault in fat-absorption is not merely the result of an enteritis.

G. W., female. At 2 years developed very offensive diarrhoea with enlargement of abdomen. Did not walk until 4. Ceased growing from 7 to 10. Then symptoms ceased and growth restarted. I first saw her at 10½ years, when she was 6 in. below normal height. For 11 months, during which she grew 1½ in., I watched her, awaiting a return of the diarrhoea. She was taking ordinary diet and no special medicine. As no further diarrhoea developed I had an analysis made of her faeces in order to test the reality of her "recovery." The stool was dark brown, formed, hard, and appeared normal. Nevertheless, when dried it was found to contain no less than 39·6 per cent. of fat (neutral fat, 7·0 per cent.; fatty acid, 28·3 per cent.; soaps, 64·7 per cent. of total faecal fat). No excess of protein was found microscopically.

Again, so far as symptoms denoting enteritis are present they are usually late in the disease, as Still¹⁴ observes. We do not in a typical coeliac case watch an enteritis becoming so severe as to cause conspicuous fat-loss in the stools; rather we seem to see a case essentially one of defective fat-digestion in which mild symptoms of intestinal catarrh may develop. That this is the generally accepted view is shown by usual treatment advised—namely, a fat-free diet.

On the other hand, the autopsy which I have reported here confirms the absence of organic disease which was emphasised by the original writers on the affection. If this be taken as a correct view, it follows that the defective fat-absorption in coeliac disease must be due to a fault of digestive secretions, either of the pancreatic juice or of the bile. Of these it is practically certain that the pancreas is not to blame,¹ and there is some evidence that the essential fault in coeliac disease is concerned with the bile-salts.

Summary.

1. Details have been given of a fatal case of coeliac infantilism in which post-mortem examination failed to show any chronic changes in the pancreas, liver, intestine, or elsewhere to account for the prolonged failure in fat-digestion.

2. The only autopsies hitherto recorded in cases of at all a similar nature have been discussed in detail; and

it has been argued that the intestinal changes found in them do not prove that the excessive fat-loss in coeliac disease is due to an enteritis.

3. Other reasons against the view that coeliac disease is essentially an enteritis have been given.

4. From these arguments it is concluded that coeliac disease is independent of organic changes and thus must be due to a digestive fault (probably a defective action of the bile on fat-absorption).

References.—1. Miller, R., Webster, J., and Perkins, H.: Coeliac Infantilism, *THE LANCET*, 1920, ii., 894. 2. Miller, R.: *THE LANCET*, 1920, ii., 1166. 3. Poynton, F. J., Armstrong, R., and Nabarro, D. N.: A Contribution to the Study of a Group of Cases of Chronic Recurrent Diarrhoea in Childhood, *Brit. Jour. Child. Dis.*, 1914, xi., 145 and 193. 4. Wells, H. G.: Chemical Pathology, third edition, Philad., 1918, 398. 5. Gee, S.: On the Coeliac Affection, *St. Bart. Hosp. Rep.* 1888, xxiv., 17. 6. Gibbons, R. A.: The Coeliac Affection in Children, *Edin. Med. Jour.*, 1889-90, xxxv., 321 and 420. 7. Cheadle, W. B.: On Acholia, *THE LANCET*, 1903, i., 1497. 8. Herter, C. A.: Infantilism from Chronic Intestinal Infection, N.Y., 1908. 9. Freeman, R. G.: Intestinal Infantilism of Herter, *Amer. Jour. Dis. Child.*, 1911, ii., 332. 10. Bramwell, B.: A Case of Pancreatic Infantilism, watched since 1901, *Edin. Med. Jour.*, 1915, i., 323. 11. Bramwell, B.: A Case of Infantilism, *Clin. Stud.*, Edin., 1902-3, i., 157. 12. Still, G. F.: On Coeliac Disease (Lumleian Lects.), *THE LANCET*, 1918, ii., 163, 193, and 227. 13. Moorhead, T. G.: Infantilism—Pancreatic and Intestinal, *Dub. Jour. Med. Sci.*, 1920, cxlix., 1. 14. Still, G. F.: *Loc. cit.*, 193.

RECURRENT HERNIA: THE OPERATIONS FOR ITS CURE.

BY A. W. SHEEN, C.B.E., M.S.LOND., F.R.C.S.ENG.,
VISITING SURGEON, MINISTRY OF PENSIONS HOSPITALS, ORPINGTON
AND SHEPHERD'S BUSH; CONSULTING SURGEON, KING
EDWARD VII.'S HOSPITAL, CARDIFF, ETC.

I HAVE operated recently on a number of cases of recurrent inguinal hernia, a few recurrent for the second time. The previous operations have been done in hospitals of various kinds—large civil hospitals, army hospitals, and smaller hospitals often described as "V.A.D.," "auxiliary," or "country house." It may be presumed that the experience of operators varied considerably. The histories given by the patients suggest that in nearly all the cases the hernia was quite small at the original operation; the recurrence also has been, almost invariably, small. Several times I have diagnosed the "recurrence" rightly as direct hernia. In only one case has there been evidence of wound infection; operators have been able to maintain asepsis and recurrence has not been due to weak post-suppurative scars. The original operation incisions have been short, not extending far out; the musculo-aponeurotic structures have shown evidence of repair, but the region of the "neck" of the sac has remained apparently virgin ground. The joining of the conjoint tendon to Poupart's ligament has been often shown by the "pegging" of muscular fibres into the ligament. The cord has in a few cases been superficial to this—i.e., a Bassini operation has been done, but much more frequently behind, in its normal position. In some cases possibly the tendon-ligament junction has given way and the cord has sunk back. In one case the vas alone was subcutaneous and might easily have been cut, the other structures of the cord being normally placed. In another a hydrocele of the cord was found, which appeared to be due to leaving the distal part of the sac. In another the original operation was for strangulated hernia. In some cases a direct hernia was present, although whether the original operation was for direct or indirect hernia it was impossible to say. In one case there was a double direct hernia. I have not seen direct evidence of ligation of the sac-neck, but sometimes there has been much scarring on the inside or outside of the sac. Neither have I seen gliding hernia or hernia demonstrable as being within the one horn left of an original bifid sac. It has often been puzzling or impossible to determine exactly what the original operation was. The matting of the parts frequently renders the operation difficult and tedious.

Causes of Recurrence.

The conclusions I have arrived at are: that in indirect hernia the usual cause of recurrence is non-removal of the deeper part of the sac; that an

occasional cause may be a small sac not having been found; that other recurrences are those of direct herniæ; that in a few cases an original indirect hernia has been cured, but a direct hernia has been present at the original operation or has developed subsequently. I have seen a number of cases of recurrent hernia in which the patients, naturally disappointed, have refused further operation and asked for trusses.

There must be an enormous number of hernia operations; operators count their cases by hundreds, sometimes by thousands. No major operation is, I imagine, more frequently performed. The percentages of recurrences I do not know; recently working in two hospitals I have encountered a number; every hospital surgeon must be operating on them, so that the total number is great. The recurrence in my cases after an original operation for small hernia is to be noted; these should be the most successful cases.

What can be Done to Lessen the Number of Recurrences?

In the first place, it must be recognised that a well-performed hernia operation demands a high degree of technical knowledge and skill, and that no one should attempt it who has not been thoroughly trained by actual operative experience on the living body. I remember—almost with horror—many years ago being interviewed by a candidate for a certain hospital appointment, who most ingenuously said that he would like to have a “slap at a hernia”! Those of us with larger experience should recognise that the deepest part of the sac right down to the parietal peritoneum must be removed to effect a cure. Recurrences—particularly immediate—may be due to the treatment of an indirect and the overlooking of a direct hernia. Sometimes a hernia operation only reveals a very tiny indirect sac beneath the internal oblique margin, the class of sac which is frequently found incidentally in a very high operation for varicocele; the operator should then be especially careful not to miss a direct hernia.

The Operation for Recurrent Hernia.

In operating for recurrence, whether I think the hernia is indirect or direct, I always investigate both possibilities. I first seek for the sac in the region of the internal ring, as that is the easiest place to find it. After reaching the external oblique aponeurosis I incise it from the external ring for 3-4 inches and turn back the flaps. I then open the cord coverings close to where the internal oblique edge crosses over the cord and divide the internal oblique in the line of the canal outwards for about an inch, this being often done between two pairs of pressure forceps placed on the muscle, as there is an artery in the muscle which is thus conveniently controlled. A sac, if present, is found and the outermost part of its neck reached without any retraction. The sac is then followed down and isolated by splitting the coverings and dissecting them off. The sac is then opened and the finger passed inside and hooked round on the peritoneal surface and its point pushed forward against the peritoneum and transversalis fascia on the inner side of the deep epigastric vessels, thereby demonstrating a direct hernia if present and its size, and feeling the width of the gap between the conjoint tendon and Poupart's ligament. The appendix can now often be removed on the right side through the sac opening. Lipomata are removed. Where there is an indirect and no direct hernia the operation is continued by removing the sac flush with the parietal peritoneum (taking care to go deeper than any circular white thickening on its interior wall), and closing the peritoneal wound. Then the cut internal oblique and the coverings of the cord are sutured, the internal oblique and the conjoint tendon drawn down to Poupart's ligament as an additional safeguard, and the external oblique closed. The cord is as little interfered with as possible, and remains in its natural position.

The Bassini Operation.

Where there is a definite direct hernia, with or without an indirect, or where there is a large gap between conjoint tendon and Poupart's ligament, then, after treatment of any direct sac present as necessary, an operation of the Bassini type is performed, the cord

eventually lying subcutaneous. I have sometimes found it necessary to put in a wire filigree lying on the transversalis fascia. I think the Bassini type of operation unnecessary in ordinary cases, the small herniæ which, nowadays, form the majority of cases operated on. Neither am I enamoured of the disturbance of the cord nor of its being placed between sutured planes or subcutaneously. Still, I think the operation should be that of choice for large musculo-aponeurotic weaknesses and for direct herniæ.

The operative procedures I have described are the ordinary ones I use for hernia. The division of the internal oblique I regard as essential in adults to get at the “neck” of the sac properly. It is not, however, necessary in babies, where the tiny overlap of this muscle can be ignored, and where the operation can be simplified to a complete removal of the sac in a way that has often been described.

An Illustrative Case.

I conclude with an illustrative case, selected because I made full notes of the operation immediately after its performance.

W. W. S., aged 36, formerly a private in the Labour Corps. A right hernia appeared in 1915 and increased in size until it became scrotal. Two years later he went into hospital for operation on it. Strangulation occurred there; immediate operation. Now there is a short linear scar over the external ring, with slight forward bulging beneath it. He is a fat man.

Operation March 21st, 1921. Long incision. The external ring was enlarged, with bulging below it, in which a few scattered cremaster fibres were seen. A small incision through this thin scar tissue opened an intimately adherent sac. The thin external oblique aponeurosis was incised for four inches and turned back, thereby disclosing a fat-overlaid internal oblique. The aponeurosis was scarred in its lower part. The internal oblique was cleared on its deep surface and divided for one and a half inches between two forceps; its section showed much fat intermingled with the muscle. The “sac” was then laid freely open and found to end blindly above and below, forming a large cyst without fluid in it, lined by shiny peritoneum, through which vas and veins could be seen. The cyst extended below into the upper part of the scrotum, but was separate from the tunica vaginalis; above it ended near the internal ring. Projecting into its upper end from the internal ring was the true sac, the size of a pigeon's egg, on the wall of which at its upper margin the cyst ended. Two layers of peritoneum had therefore to be gone through to reach the interior of the hernial sac. I cleared this sac near the ring, the rest of it being free, and continued the separation half an inch beyond the commencement of the parietal peritoneum, dividing deep to its neck a knot of scar tissue, probably the place where the strangulating stricture was divided. The sac had been opened during these manipulations; non-adherent omentum occupied it. The finger was passed within it and inwards, demonstrating that there was no direct hernia, and that the parietes were firm, the conjoint tendon coming close down to Poupart's ligament. The sac was removed and the peritoneal wound closed. The large cyst was removed. A large lipoma running along the cord was removed, being tied at the level of the peritoneal wound in two sections. The vas and other cord structures were freed and held aside. All fragments of cremaster, other coverings, and scar tissue were cleared off conjoint tendon and Poupart's ligament. The internal oblique and conjoint tendon were joined to Poupart's ligament, two stitches going external to the cord. The external oblique was closed. The cord was laid back and the subcutaneous fascia and skin united over it.

Comments.

At the operation for strangulation the external oblique was opened for an inch or more from the ring, the sac opened, the stricture divided, the contents reduced, and the wound closed, healing per primam. Adhesions closed the neck of this original sac and a second sac was invaginated into it from the top, originating either from the deepest part of the original sac beyond the closing adhesions or as a new sac. No “radical cure” was done. It is advisable that it should be done in operations for strangulation; usually there is no additional risk. The second operation shows that conditions present can best be elucidated by approaching the neck of the sac first. The Bassini type of operation was done although there was no great musculo-aponeurotic weakening because of the fatty condition of the muscles and the weak scar in the external oblique.