3. Adrenal rests^{26 27} originally or by metastasis from the hypernephroma which was present. A hypernephroma is itself a distinctly rare tumour. Its presence may have been a pure coincidence. Doran,²⁰ Grafenberg,²¹ Guthrie and d'Este Emery,²² and Monsarrat²³ record cases of metastasis from hypernephromata. Lubarsch²⁴ found metastasis 20 times out of 29 cases in various organs, including the pancreas.

The microscopical findings do not accord with the suggestion that the cysts arose as retention cysts, caused by obstruction to the outflow from the small ducts, as (a) by a pathological secretion from the zymogenous cells, analogous to the ascribed cause of congenital cystic disease of the kidney, or (b) by chronic interstitial pancreatitis (see Robson and Moynihan²⁵). For it should be noted that in interstitial pancreatitis the islets of Langerhans usually escape atrophy, at all events until a very late stage (Robson²⁷), and there was no inflammatory process.

A feature of the case was the complete transformation of the pancreas into cysts. It would have been quite impossible to have enucleated the tumour or any part of it from the pancreas, as appears to have been done in some of the recorded cases (Dunning's case¹⁰).

Briefly, from a clinical point of view, the case was of interest because it showed the effect upon the economy of gradual loss of function of the pancreas in an otherwise healthy person, the outstanding features of the terminal part of the illness being the gradual wasting²⁶ of the body, especially of the muscles, unto death and the warding off of glycosuria until the end. The urine removed from the bladder after death contained sugar. It had always been free from it during life.

REFERENCES.

- ¹ New York Med. Journ., 1882, xxi, p. 46; and Med. Rec., 1882, xxi, p. 286. ² Arch. f. klin. Chir., 1885, xxii, p. 994. ³ Zeitschr. f. Heilk., 1886, vii, p. 19. ⁴ Inaug. Diss., Breslau, 1887, p. 77. ⁵ Virchow's Archiv, 1890, cxx, p. 230. ⁶ Prag. med. Woch., 1891, xvi, p. 365. ⁷ Bull. de la Soc. Anat., 1892, lxvii, p. 310. ⁸ Gaz. des Hôp., 1896, lxxix, p. 347. ⁹ Amer. Journ. Med. Sci., vol. cxx, 1900, p. 184. ¹⁰ Amer. Journ. Obstet., New York, 1905, li, p. 101. ¹¹ Ann. Surg., 1905, xxxvii, p. 226. ¹² Adami refers to Ransohoff, Amer. Med., 2, 1901, p. 138. ¹³ Unable to find Vankreef's case. ¹⁴ Cent. f. d. Grenzgeb. de Med. u. Chir., 1903, vi, pp. 490, 529, 573, 619, 654. ¹⁵ Congrès franc. de chir., 1891, p. 618. ¹⁶ Path. Soc. Trans., 1885, p. 17. ¹⁷ Phil. Trans., ser. B, vol. cxvii, p. 25-46, London, 1904. ¹⁸ BRITISH MEDICAL JOURNAL, 1909, vol. ii, pp. 681, 1381, 1652. ¹⁹ Proc. Path. Soc., June 2nd, 1906. ²⁰ Journ. Obstet. and Gynaecol. of the British Empire, June, 1907. ²¹ Virchow's Archiv, vol. xciv, 1908. ²² BRITISH MEDICAL JOURNAL, vol. i, 1910, p. 141. ²³ BRITISH MEDICAL JOURNAL, vol. ii, 1909, p. 1800. ²⁴ Virchow's Archiv, Bd. cxxxv, p. 104. ²⁵ System of Medicine, vol. iv, Part I, p. 428. ²⁶ Diseases of the Pancreas and their Surgical Treatment, 1902, pp. 193, 228. ²⁷ BRITISH MEDICAL JOURNAL, 1908, vol. i, p. 1163.

Hospital, during the last three and a half years, in the practice of all the surgeons, 190 cases have been admitted. If strangulated external hernias of all kinds are excluded the number falls to 83. These figures exclude all cases in which the large intestine was obstructed, a fairly numerous class. The mortality in all cases of acute obstruction when they receive proper surgical treatment is given by Treves¹ as 50 per cent. The more recent statistics of Elsberg² give 30 to 40 per cent. as the correct mortality. In the herewith collected cases it was 19 per cent., but if the cases of external hernia are not considered, it rises to 24 per cent.

THE CAUSES OF OBSTRUCTION.

The following table shows the conditions leading to obstruction which were met with, the number of cases in which gut was found gangrenous, and the mortality in the several classes:

	No. of Cases.	Gangrenous Gut.	Deaths.
Inguinal hernia ...	50	6	5
Femoral hernia ...	46	11	7
Umbilical hernia ...	7	1	3
Ventral hernia ...	3	1	1
Obturator hernia ...	1	0	0
Intussusception ...	35	4	6
Adhesions not elongated into bands ...	22	4*	5
Bands ...	14	1	2
Meckel's diverticulum ...	3	0	0
Gall stones ...	1	0	1
Volvulus ...	1	1	1
Stricture ...	1	0	1
Hole in mesentery ...	1	0	0
Pressure by an abscess ...	1	0	1
Mesenteric thrombosis ...	2	2	2
Cause undetermined... ..	2	0	2
Total	190	31	37

* Actual gangrene was not present in these four cases, but the gut was so damaged that resection was deemed necessary.

External Hernia.

Under this heading few remarks need be made except to call attention to the fact that a femoral hernia, if not cured by operation, is more likely to become strangulated than an inguinal hernia. In spite of the much greater relative frequency of inguinal hernia, the number of strangulated cases dealt with is only slightly greater than the number of strangulated femoral hernias. This is because of the unyielding character of the margin of the crural ring. In inguinal hernias obstruction is most often caused by fibrosis round the internal ring at the upper extremity of the canal.

The number of cases of strangulated umbilical hernia is not large, but only those cases which had small intestine in the sac are included. The omentum is the first structure to enter into the sac of an umbilical hernia, then this drags in the transverse colon. Usually only after this has happened does small gut enter the sac.

Of the 3 ventral hernias 2 occurred through old laparotomy scars; the other one was in the middle line below the umbilicus. No operation had been performed, but there was some divarication of the recti muscles. The aperture leading into the abdomen was small and had hard margins. A loop of small intestine was strangulated to the point of gangrene.

Adhesions.

Next to external hernia and intussusception as a frequent cause of obstruction of the small gut, comes local peritonitis leading to the formation of adhesions. In the table the cases of obstruction due to adhesions which had not elongated to form bands are placed separately. Where definite bands were present, the cases have been grouped with cases of obstruction due to bands of other origin than

A Lecture

ON

ACUTE OBSTRUCTION OF THE SMALL INTESTINE.

DELIVERED AT THE MEDICAL GRADUATES' COLLEGE AND POLYCLINIC

By CHARLES A. PANNETT, F.R.C.S., M.D.,

SUPERNUMERARY OUT-PATIENT SURGEON TO ST. MARY'S HOSPITAL; ASSISTANT SURGEON, ROYAL FREE HOSPITAL.

Few serious abdominal conditions present more difficulties in diagnosis and treatment than obstruction of the small gut. Yet diagnosis must be early and treatment prompt if the conduct of the case is to lead to a successful issue. In the beginning signs and symptoms may be inconclusive; there is only a probability of obstruction being present. To wait until this probability becomes a certainty will, in many instances, cost the victim his life. If the possibility of intestinal obstruction were more obtrusive in the practitioner's mind when confronted with the complaint of abdominal pain, early diagnosis would be arrived at more often. But he can scarcely give the disease a greater prominence in his mentality because of the rarity of the condition in comparison with the multitude of other diseases with which he has to cope. In a hospital where the number of acute abdominal cases admitted is large, where such cases concentrate from an extensive surrounding district and from the practice of many men, the numbers do not reach a high figure. At St. Mary's

the elongation of adhesions. It is right to make this distinction, because adhesions not elongated into bands cause simple obstruction of the lumen of the gut (obturation), whereas bands usually cause, in addition, interference with the blood supply (strangulation).

Obstruction of the small intestine due to adhesions occurs most often in the lower part of the ileum, and the adhesions are situated in the pelvis or right iliac regions. In the pelvis they result from operations upon, or disease of, the female internal genital organs. Appendicitis is a frequent cause of adhesions in the right iliac fossa, but other forms of local peritonitis also occur here, notably peritonitis around glands in the lower part of the mesentery. Tuberculous peritonitis is another well-known cause. Of the 22 cases, 8 were due to appendicitis and followed appendicectomy, 3 followed hysterectomy, 1 followed an operation for extrauterine gestation, 1 was due to adhesions around an inflamed ovarian cyst, 1 to tuberculous peritonitis, and 1 was found where a length of gut in a post-operative ventral hernia had become coiled up and matted into an impassable mass, and had subsequently become reduced into the abdomen, no fixation to the sac having occurred. There were 7 cases of obstruction of the small intestine in the right lower abdomen, where the origin of the adhesions was not determined at operation. When obstruction followed an operation, the interval elapsing before obstructive symptoms came on varied very much. Thus, in the 8 cases of appendicectomy, for example, the longest interval was two years and the shortest seven days.

The ways in which adhesions produce obstruction are said to be by (a) the formation of a perimural stricture, (b) fixation of the walls of the gut, or (c) kinking. The exact mechanism of obstruction when the latter two conditions are present is not always clear. A length of gut

Fig. 1.—A length of gut matted into a fused mass, the result of inflammation in the sac of a ventral hernia.

may be coiled up and its component parts fused into a formless mass. Peristalsis of the affected segment may be so interfered with in such a case that the contractions of the gut above are not sufficiently powerful to force the contents through the intricacies of the canal in the coiled-up portion, even though there be no actual mechanical block. The ventral hernia above mentioned was such a case. Afferent and efferent sections of small intestine were attached to a rounded structure about the size of an orange, the architecture of which could not be determined by external inspection. (See Fig. 1.) Even after excision it was most difficult to trace the lumen of the gut through all its windings.

Adhesions over a limited area are said by Treves to interfere with normal intestinal movement, just as peristalsis is abolished in a strangulated partial enterocoele. The cases do not seem to be strictly comparable; in the latter condition of sudden traumatism inhibition is probably reflex, even as such stoppage of movements occurs after any manipulation of the bowel, as the researches of Cannon and Murphy³ demonstrated. Obstruction due to adhesions over a limited area should, more probably, be relegated to the third class—obstruction by kinking. This last class is very interesting, especially when it is remembered that very acute bending must be present to hinder the fluid contents of the ileum in their onward passage. Most surgeons must have opened the abdomen for obstruction, and found an adherent loop, which certainly was not occluded, and yet the freeing of this loop resulted in the disappearance of all the symptoms and a prompt recovery. Figs. 2 and 3 are an attempt to explain this. Fig. 2 represents a caecum and neighbouring ileum after an appendicectomy, and shows also that the terminal loop of ileum has become fixed by local peritonitis. Under normal circumstances, in the absence of adhesions, if for any reason the proximal limb of this loop should become unusually quickly filled with fluid or

gas, this distension in itself would lead to a straightening out of the bend, just as the kinked finger of a rubber glove will straighten itself if the mouth of the glove be blown

Fig. 2.—Lowest coil of ileum fixed by adhesions after appendicular inflammation.

Fig. 3.—Obstruction owing to inability of coil to straighten itself.

into. But if, as depicted, the limbs of the loop be fixed and the distension take place rather rapidly, the septum between the two limbs will be pushed over, closing the lumen of the distal limb and acting as a valve, as seen in Fig. 3. This condition of affairs might be perpetuated if the intra-abdominal pressure were increased so that surrounding coils of gut from all sides were thrust against the loop in question. Such pressure would become greater from accumulation of gas the longer the obstruction remained. Under such circumstances merely opening the abdomen and allowing some coils to escape from the wound might be sufficient to allow the patency of the distal limb to be restored. When the surgeon arrives at it there is no longer obstruction, yet he very rightly frees the coil. Quite limited adhesions at the lower end of the ileum, where the gut is more or less fixed, are sufficient to bring this mechanism into play.

Bands.

When bands give rise to obstructive symptoms, in the vast majority of cases a loop of small intestine is strangulated under a short band attached at each end.

In 7 such cases the method of formation of the band was not determined more definitely than that it was a stretched adhesion the result of a local peritonitis. One case occurred four years after a partial excision of the colon. In 2 cases omentum formed the band; in the one its free extremity was attached to a tuberculous mesenteric gland; in the other the omentum passed completely round the loop of small gut employed to effect an ileo-sigmoidostomy, and its free extremity had become adherent to the abdominal scar. In 2 cases the band connected gut to a tuberculous gland in the mesentery. In 3 cases the origin of the band was a previous appendicitis.

Other bands may be produced when the appendix vermiformis, a Fallopian tube, an appendix epiploica, or a persistent Meckel's diverticulum becomes adherent at its free extremity. The first three causes were not met with in this series, but in 3 cases Meckel's diverticulum was the cause of the obstruction. It is stated by Nothnagel⁴ that when a Meckel's diverticulum is simply adherent to the umbilicus it is never a cause of obstruction, but in one of these cases such a condition of affairs was present, and Halstead⁵ has found this to be so in 15 out of 69 cases of obstruction due to a persistent diverticulum. In another patient the tip of the process was adherent to a lower mesenteric gland, and under the short loop thus formed a coil was strangulated. In the third case a chronic obstruction ended acutely, and at operation it was found that the diverticulum was surrounded by adhesions, whilst there was a stricture of the gut at the origin of the process.

Gall Stones.

A gall stone caused an obstruction in one case; it was 1 in. in diameter, and lodged in the upper part of the ileum. It had ulcerated from the gall bladder into the duodenum, but the adhesions between these two viscera had broken down, leading to leakage peritonitis, which caused the patient's death.

Stricture.

A fibrous stricture occurred just above the ileo-caecal valve in one case. It was due to cicatrization of a tuberculous ulcer.

Volvulus.

The one case of volvulus occurred in a patient who, two weeks before, had had an abdominal operation for the release of a partial enterocele. *Post mortem* the twisted coil, whose two limbs had become approximated by a cicatrizing process in the mesentery, was found in the pelvis.

Mesenteric Thrombosis.

There were two cases of thrombosis of tributaries of the superior mesenteric vein. In only one was a *post-mortem* examination made, but no cause for the thrombosis was found. There were adherent *ante-mortem* clots in the portal vein itself.

Mesenteric Aperture.

One case occurred where the bowel was strangulated by passing through a congenital hole in the mesentery.

Appendic Abscess.

Pressure by an appendicular abscess was found to be causing obstruction in one case.

CLINICAL FEATURES OF ACUTE INTESTINAL OBSTRUCTION.

The symptoms are: (1) Pain, (2) shock, (3) vomiting, (4) constipation, and finally (5) collapse.

The first symptom is always pain. It begins suddenly, and is very acute; it is constant, but paroxysmal exacerbations occur. This is very characteristic. In obstruction of the small intestine it is referred to the umbilicus, unless the obstruction is very high up, as when a gall stone ulcerates into the duodenum, or very low down, the terminal portion of the ileum having become adherent to the parietal peritoneum. In such cases the pain may be referred to the site of the lesion. The pain is made worse by food or by enemata, both of which stimulate peristalsis. Towards the end, in the state of collapse, the pain abates, and may disappear. Shock, occurring at the onset, is not always present. It is dependent upon the suddenness of the blockage, the tightness of the constriction, and the length of gut affected. The absence of shock in some cases is to be borne in mind.

Treves remarks that "vomiting in obstruction is characterized by its early onset, persistence, and its copiousness." This in the main is true, but every surgeon must be able to recall cases in which there has been a single act of vomiting, or perhaps two, only, during the first twenty-four hours, although there has been complete obstruction. Too much reliance must not be placed upon vomiting as a prominent symptom. If the condition is allowed to go on until the vomit becomes faeculent, the prospect of recovery is much lessened. When there is profuse vomiting, thirst may be a very prominent and distressing symptom.

As a rule constipation is absolute. In spite of the fact that at the onset the colon may contain much faecal matter, it is reflexly paralysed by the injury to the small gut. But in some cases the bowel below the blockage is emptied soon after the onset. Barnard's plan of giving two turpentine enemata within half an hour of one another, in order to decide whether obstruction is present or not, is excellent. The first one empties the bowel below the obstruction; the second flows away clear—indeed, it cannot enter the lumen of the tonically contracted bowel below the block.

The collapse which comes on towards the end is due to toxæmia combined with excessive loss of fluid by vomiting.

The physical signs of obstruction are (1) constitutional and (2) abdominal.

The pulse is unchanged at first. Only when peritonitis comes on and collapse sets in does it become rapid and weak. The temperature remains low. Even the onset of peritonitis does not usually raise it. The urine is partly or completely suppressed. Indicanuria occurs in obstruction of the small gut; it is usually absent when the colon is affected. The terminal stage of collapse is characterized by cyanosis, pinched features, sunken eyes, extreme muscular debility, and weak voice.

With regard to the state of the abdomen, it may be said that, with some reservations, it is not rigid, not tender, not distended, and there is no visible peristalsis. The flaccidity of the abdominal wall is striking; the onset of rigidity indicates the development of peritonitis. The presence of tenderness has the same significance. During the early and most favourable stage for operative treatment distension is absent, but later meteorism occurs. It is markedly increased with the onset of peritonitis. If acute obstruction supervenes on a chronic condition distension is present from the beginning.

It is usually stated that in primarily acute obstruction visible peristalsis is always absent, that only coils of small intestine hypertrophied as the result of chronic obstruction can, by their powerful contractions, render themselves visible through the abdominal wall. But peristalsis in acute obstruction may be visible through the thin abdominal wall of a child or through the lax abdominal wall of a multiparous woman. In such cases within twenty-four hours distended coils of small gut may stand out. When the peristaltic waves cannot be seen they can sometimes be felt. A coil of gut hardening under the fingers whilst palpation is being performed is a very valuable sign, and accompanied by a paroxysm of pain is absolutely diagnostic of obstruction. Not infrequently, through the stretched abdominal wall of a multiparous woman, coils of intestine can be seen to stand out, and also to contract when there is no obstruction present. These normal peristaltic movements are apt to be misleading, but doubt as to their nature will disappear if it be remembered that they are unaccompanied by pain and that they are slower and less tumultuous than the violent contractions in obstruction.

Occasionally, in occlusion due to some cause other than intussusception, there is a tumour felt in the abdomen. In a case where there was a loop of gut strangulated under an adherent Meckel's diverticulum, a distinct tumour (the distended loop) was felt in the right iliac fossa, so that the case was sent in with the diagnosis of appendix abscess. In another case an ill-defined resistance, sub-resonant on percussion, was found to be a collection of collapsed coils below the obstruction. Rectal examination will exclude a carcinoma recti, and perhaps reveal an intussusception.

DIAGNOSIS IN RELATION TO PROGNOSIS.

Every hour's delay before instituting treatment renders the prospect of recovery for the patient less likely, even though the appropriate surgical measures be taken. There is a point in the course of the disease beyond which all known surgical measures are powerless to avert the fatal issue. At present, while the actual cause of death in acute obstruction is still undetermined, there is no way of telling when this stage has been reached. It is therefore important to make the diagnosis early and to proceed to treatment forthwith. As shown above, in early stages many of the signs attributive to an acute abdomen (rigidity, tenderness, distension) are wanting. A conclusion must be arrived at without them. The characteristic pain and the repeated vomiting (which may, however, fail), together with the result of two test enemata, suffice for the diagnosis. Always is it to be borne in mind that the pulse-rate and temperature are not disturbed. If gut becomes gangrenous the prognosis is much worse (53 per cent. mortality in this series, as compared with 19 per cent. taking all cases together). Gangrene may set in very early; in 4 cases it was present within twenty-four hours, and in 5 others within forty-eight hours.

If, in addition to the diagnosis of obstruction, it can be settled where the obstruction is situated, surgical manipulations may be much expedited with a bettering of the prognosis. The examination of the hernial apertures and the rectum should never be omitted or valuable information will be lost, and the same may be said of the urine. The onset of uræmia may strangely simulate obstruction.

THE INFLUENCE OF EXPERIMENTAL OBSERVATIONS UPON
THE SURGICAL TREATMENT.

The problem of the treatment of acute obstruction does not resolve itself merely into the development of an efficient technique for the restoration of the continuity of the lumen of the bowel. Many cases will die in spite of this procedure being executed in an expeditious and

skilful manner. Indeed, some surgeons (Elsberg), maintain that it is not the most essential step in operative treatment, and, in fact, is frequently an unnecessary one.

Of the three alleged causes of death in acute obstruction—namely, (1) reflex nervous influences, (2) secondary peritonitis, (3) acute autointoxication—the first two have been shown not to be the real causes in the majority of cases. It is true that, if a case is allowed to pursue an uninterrupted course, organisms may eventually traverse the intestinal wall even without there being any gangrene present, and set up peritonitis, but this is unusual. Most cases die without any peritoneal infection having occurred. Absorption of the toxic material found in the lumen of the gut is to be regarded as the most potent element leading to dissolution. Murphy and Vincent's⁶ experiments support this view, and show how the rate of absorption and so the prognosis depend upon the anatomical conditions present. In simple obstruction absorption is much slower than where there is interference with venous return, in which case the lymphatics absorb abundantly, and, moreover, the lining intestinal epithelium is so damaged that it cannot perform its selective absorptive action. In simple obstruction Hartwell and Hoguet⁷ attribute great importance to the excessive loss of fluid by vomiting, and have been able in dogs to greatly prolong life by saline injections. As a preliminary to surgical treatment it is a good plan to infuse saline subcutaneously, and the administration should be continued during the operation. It may be advisable to wash out the stomach.

FINDING THE SITE OF OBSTRUCTION.

Subject to certain reservations made as the result of experimentation, the affected segment of gut should be reached and the obstructing agent removed with the least possible delay. This presents some difficulties when the actual site of the obstruction has not been determined before operation. A median subumbilical incision is made. Many surgeons—for example, Alexander Miles⁸—recommend that this incision should be only large enough to admit two fingers, and the greatest care be taken not to allow the intestines to escape from the wound; that exploration should be made with the two fingers in the abdomen only; that if large lengths of small bowel have to be examined, this viscus should be brought out of the wound a loop at a time, and the examined loop replaced as the next loop is removed. This advice can be followed, with the best results to the patient, by none but the most expert and experienced surgeons; the dexterity necessary for its performance can be acquired but slowly, because no surgeon operates upon a very large number of such cases within any one year. It is much better at once to make a large incision, a procedure recommended by Legg and Edmunds.⁹ It will be impossible to prevent distended coils escaping from the wound without using harmful force and manipulations. They are to be allowed to escape. The experiments of Cannon and Murphy⁵ in this connexion are important. They show that local post-operative paralysis is largely due to manipulation of the intestines, and is more marked the rougher such handling is. What is more interesting still, they show that cooling of the intestinal coils, exposure to air, and prolonged ether anaesthesia have no appreciable paralysing effect. To secure gentleness of manipulation is far more essential than to prevent prolapse of the gut, or even to save considerable time over the operation. The escaped coils should be covered with towels wrung out of warm saline solution. If the surgeon accepts Yandell Henderson's theory of acapnia shock,¹⁰ these towels should be made of rubber tissue enclosed in a double layer of cloth. The caecum should be sought first: its collapsed condition settles that the obstruction is in the small bowel. All the hernial apertures are then to be explored from the interior of the abdomen. If the obstruction is not discovered by these means, one of two procedures may be followed:

(a) The collapsed gut below the obstruction may be sought for, and this gut traced up to the obstruction. It is, however, not always easy to find collapsed gut; the contracted coils frequently get thrust deep down into the pelvis. They should be sought for here.

(b) What appears to be the most dilated coil may be traced downwards to the obstruction. In the supine position the most dilated coils rise into contact with the anterior

abdominal wall, and so are the first ones to be encountered when the peritoneum is opened. The coil immediately above an obstruction is more dilated than coils higher up. It is easy to determine which way to trace a selected coil of gut, if any twisting of the mesentery is undone, and it is remembered that the right surface of the mesentery is also its upper surface, and that the mesentery runs from above downwards and to the right.

TREATMENT OF INJURED GUT.

The obstruction having been found and removed, a decision must be possibly arrived at as to the viability or non-viability of a strangulated coil. Two points must be very strongly emphasized. First, if there exists the smallest doubt as to viability, a resection is imperative; and, secondly, if a resection is performed, the patient stands a greater chance of recovery if a large section of gut be removed than if only a small segment be taken away. It is better to resect gut that is badly damaged but viable, than to return it to the abdomen. Murphy and Vincent's experiments show that in gut which is strangulated and in the inflamed damaged gut immediately above an obstruction, the absorptive mechanism of the intestinal mucosa is deranged. Such gut, after the obstruction has been removed, may absorb a fatal dose of toxin from its lumen. Life may be saved by removing a sufficient length of such damaged but still viable gut.

THE VALUE OF ENTEROTOMY COMBINED WITH EVACUATION OF THE INTESTINAL CONTENTS IN THE RELIEF OF THE OBSTRUCTION.

The toxic material in the gut is not absorbed by uninjured intestinal mucous membrane, as the researches of Whipple, Stone, and Bernheim¹¹ and of Wilkie¹² have shown. The flooding of the gut below the obstruction with toxic fluid from above, when the obstruction has been relieved, is not fraught with danger, and if the gut is not too seriously damaged, it is probably not wise to empty it by enterotomy. If practised it should be done as Moynihan¹³ recommends. It is to be remembered, however, that such emptying involves a great deal of manipulation of the bowel—in itself an event to be avoided as far as possible. Emptying of the distended gut may be absolutely necessary to enable closure of the abdomen to be carried out.

Hence, enterotomy and evacuation of the gut is to be recommended (1) if the intestines are so distended that it is impossible to replace them in the abdomen unless their inflation be diminished; (2) if, whether or no a resection has been done, it is believed, from the damaged appearance of the gut above the obstruction, that the mechanism by which a normal mucous membrane is prevented from absorbing toxins has been interfered with.

THE VALUE OF ENTEROSTOMY AS AN ADJUVANT WHEN THE OBSTRUCTION HAS BEEN RELIEVED.

Enterostomy above the site of obstruction, after the block has been removed, has been strongly recommended and has been said to reduce the mortality. The present series (7 cases with 4 deaths) does not bear this out, but the number is too small to generalize from. In such cases it is a common experience to find that, after the primary gush of intestinal contents, nothing escapes from the opening for twenty-four to forty-eight hours. The enterostomy has not led to evacuation of toxic material when it is most needed, because peristalsis has been in abeyance. As soon as the patient's condition improves and intestinal movements recommence, the contents escape from the ostium, but they would do equally little harm if they were passed into the healthy bowel below the obstruction. It would appear that if enterostomy is to be done it should be performed at some distance above the obstruction, to prevent the toxic intestinal contents coming into contact with the injured mucous membrane of the gut lower down. Also it would not appear logical to make a small lateral opening in the gut, leaving the normal channel almost unencroached upon. The diversion of the toxic stream should be complete, or the object for which the enterostomy is done will be frustrated. Nutrition may be seriously interfered with by such an operation, and the patient escape his obstruction only to be faced by starvation. It would seem to be very rarely indicated.

ENTEROSTOMY AS THE SOLE SURGICAL PROCEDURE.

Enterostomy without search for the obstruction is only performed by most surgeons in the very desperate cases, but it has been strongly advocated by Elsberg² as a normal procedure under a large number of circumstances. His argument is this:

Gangrene occurs in 15 per cent. of all cases of acute obstruction. If cases of intussusception and volvulus, which are usually diagnosable before the abdomen is opened, are excluded, gangrene only occurs in 5 per cent. The performance of enterostomy evacuates the gut above the obstruction. The removal of the distension may allow strangulated coils to recede from their incarcerated position, or may relieve strangulation from kinking. The operation can be performed rapidly, under local anaesthesia if necessary, and with very little shock. At a second operation the obstruction can be removed and the enterostomy opening closed.

The arguments against adopting this line of action are obvious. Statistics in this series give 6 per cent. of gangrene in such cases, and this agrees with those cited by Elsberg. But there is a fallacy here. In all these cases the obstruction was removed; if this had not been done it is fair to conclude that other cases, where strangulation was present, would have gone on to gangrene. If the enterostomy made be situated high up the small gut, then the patient will suffer from loss of nutrition, which, following the toxæmic condition the result of obstruction, brings him into a very unfavourable state for his second operation. This second operation has to be performed in the presence of infection and often through a septic wound.

RESULTS OF OPERATIVE TREATMENT.

Inguinal Hernia.—Resection was done in 6 cases with 2 deaths, one of which occurred on the fourteenth day after operation, from pneumonia, in a patient aged 75. Enterostomy was combined with resection in one of the fatal cases. The other three deaths occurred from paralytic ileus (enterostomy proving unavailing), from collapse, and from the inhalation of the septic vomit.

Femoral Hernia.—Resection was done in 8 cases with 4 deaths. In two cases an enterostomy only was performed and both died. In one case the perforated gut was freely opened but left *in situ*, whilst a lateral anastomosis was done from above between the afferent and efferent limbs of the strangulated loop. This patient also died.

Umbilical Hernia.—Resection of both large and small gut was done in one case with fatal result; two other cases died although no gangrene was present.

Ventral Hernia.—There was one resection, with death on the third day.

Intussusception.—Resection was performed 4 times with 3 deaths. The patient who recovered was 4 months of age. One of the resections was done in a case which recurred twenty-four hours after operation. Recurrence happened in one other case on the fourth day. It was successfully operated upon. Two other cases died although gangrene was not present and the intussusception was reduced. Appendicectomy was found necessary, in addition to reduction, in two cases, one of which was a fatal case.

Adhesions.—Resection was performed in 5 cases with no deaths. In one it was combined with enterostomy. An ileo-transverse colostomy was done in two cases of obstruction after appendicectomy with no deaths. Enterostomy alone was done 5 times with 3 deaths. In two other cases the obstruction was relieved, but death followed.

Bands.—Resection was only performed once; the patient recovered. Enterostomy alone was performed once, and the patient died.

Meckel's Diverticulum.—There were no deaths. Resection was performed once, but only for the closure of an enterostomy which was made twenty-four hours after the relief of the obstruction.

Gall-stone Impaction.—This patient died from leakage peritonitis from the adhesions between the gall bladder and the duodenum breaking down. The gall stone was removed by enterotomy.

Volvulus.—This case died without operation.

Stricture.—Enterostomy above the stricture only was performed. This patient died.

Mesenteric Thrombosis.—Resection was done in these two cases, but the patients died.

Obstruction of Unknown Origin.—In two cases the cause of the obstruction was not determined; enterostomy was done in both. They both died, but no *post-mortem* examination was made.

I have to thank the members of the surgical staff of St. Mary's Hospital for their courtesy in allowing me to add the records of their cases to those of my own.

REFERENCES.

- ¹ Treves, *Intestinal Obstruction*, 1899. ² Elsberg, *Ann. of Surg.*, xlvii, 738. ³ Cannon and Murphy, *Ann. of Surg.*, xliii, 512. ⁴ Nothnagel, *Diseases of the Intestines and Peritoneum*, English edition, 1907. ⁵ Halstead, *Ann. of Surg.*, cxii, 471. ⁶ Murphy and Vincent, *Bost. Med. and Surg. Journ.*, vol. clxv, 1911, p. 684. ⁷ Hartwell and Hoguet, *Journ. Amer. Med. Assoc.*, July 13th, 1912. ⁸ Alexander Miles, article in *Choyce's System of Surgery*, 1913. ⁹ Cheyne and Burghardt's *Manual of Surgical Treatment*, vol. iv, 1913. ¹⁰ Yandell Henderson, articles in *Amer. Journ. Phys.*, 1908-1911. ¹¹ Whipple, Stone, and Bernheim, *Journ. Exper. Med.*, 1913, xvii, 286, 323. ¹² Wilkie, *BRITISH MEDICAL JOURNAL*, October 25th, 1913. ¹³ Moynihan, *Abdominal Operations*, 1906.

PITUITRIN IN POST-OPERATIVE INTESTINAL STASIS.

By NORMAN PORRITT, M.R.C.S., L.R.C.P.LOND.,

CONSULTING SURGEON, HUDDERSFIELD ROYAL INFIRMARY.

In the two following cases pituitrin was a valuable auxiliary to other measures for supporting vital power and overcoming intestinal torpor, especially as the cases were dealt with in a hillside cottage with no other nursing than that of a neighbour and the visits of an excellent district nurse.

CASE I.

The first case was that of a girl, aged 9, whose appendix was removed a week after the subsidence of an acute attack. Eight days after the operation the wound was reopened to ascertain the nature of a painful swelling in the right iliac fossa attended with abdominal distension and vomiting suggestive of obstruction. A deep abscess was found and drained. The distension increased and persistent vomiting of dark green fluid came on. Calomel given by the mouth was rejected, and turpentine enemata failed to bring away any flatus. On the night after the draining of the abscess the patient was actively delirious, picking at the bedclothes and stripping the paper from the wall at the head of the bed; the distension was increasing, the vomiting of green liquid was incessant, and she had a shabby pulse of 130; Parke, Davis's pituitrin 0.50 c.cm. was injected subcutaneously. An hour later the pulse had fallen to 115, was fuller and steadier and flatus began to be expelled per rectum. An asafœtida enema then caused the expulsion of much flatus. After this the dangerous symptoms subsided and the patient made a good recovery.

CASE II.

A woman, aged 43, was seen on April 1st, 1914, when she had the typical signs of acute appendicitis. Immediate operation was advised, but as there was no other woman in the cottage it was impossible to do anything that day. The next day the abdomen was markedly distended, and the acute tenderness in the right iliac fossa had given place to general abdominal tenderness. On opening the abdomen the same day sero-purulent fluid free in the abdomen and an acutely inflamed appendix, adherent to the side of the pelvis, but with no other limiting adhesions, were found. Near the end of the appendix was a perforation from which thick odourless pus was trickling into the abdomen. The appendix having been removed, a stout tube was passed down to the stump, and another into Douglas's pouch. The morning after the operation there was much serous discharge on the dressings and in the tube going to the appendix stump, but from the tube in Douglas's pouch thick grumous pus was sucked. No flatus had passed, and the bowels had not acted, though 5 grains of calomel had been given five hours after the operation. Three grains of calomel immediately, to be followed by a grain every hour till the bowels moved, were ordered. Next day the distension was worse, and green liquid was vomited, though nothing was being taken by the mouth. The bowels had not moved, and neither turpentine, asafœtida, nor claret enemata brought away either faeces or flatus. One c.cm. of Parke, Davis's pituitrin and $\frac{1}{16}$ grain of strychnine were given on the second morning after the operation. Within an hour some flatus was expelled per rectum. At night $\frac{1}{32}$ grain of strychnine and 1 c.cm. of pituitrin were given. In half an hour a free expulsion of flatus took place, followed soon afterwards by a copious liquid motion. The after-progress of the case was uneventful and satisfactory.

THE Royal County Hospital for Children, Heswall; the Children's Convalescent Home, West Kirby; the Liverpool Convalescent Institution, Woolton; and the St. Paul's Eye Hospital are among a large number of charitable undertakings each of which receives a bequest of £1,000 under the will of the late Mr. Septimus Brocklehurst of Sefton.