pyelonephritis. Resolution was not possible in the presence of an enlarged prostate and an indwelling catheter as reinfection of the kidneys was inevitable. Cases 3, 5, 11 and 12 survived because the urinary obstruction was removed and the drainage tubes discarded. Infection recurred in Cases 3 and 5 because the chronically distended bladder had not had time to contract down sufficiently and completely evacuate all the urine. The small amount of residual urine acted as a sump in which the organisms thrived. Carcinoma of the prostate, small fragmented calculi, diverticulæ and bladder neck obstruction caused the relapsing infection in the remaining cases.

Summary

Chloramphenicol should only be administered when the organisms are sensitive to it and in-

sensitive to penicillin and streptomycin and the sulpha group of drugs. It is of value in urinary infections due to the colon and para-colon organisms, *Ps. aeruginosa*, *Klebsiella*, *Salmonella* and *B. proteus*. Relapses are common in the presence of growths, drainage tubes and bladder neck obstruction.

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THE MANAGEMENT OF ACUTE INTESTINAL OBSTRUCTIONS

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It is now 20 years since Harry Burgess (1929) published his well-known and often-quoted papers analyzing a very large series of cases of acute intestinal obstruction and reviewing the fatality rates compared with those of other acute abdominal emergencies. He pointed out that, although at the Manchester Royal Infirmary the mortality for acute appendicitis in 1924 was one-twelfth of that in 1900 and for gastro-duodenal perforations one-seventh, that for acute intestinal obstruction was still half the earlier figure. The figures of Souttar, which he quoted, showed an overall mortality of 26.2 per cent. Excluding all external herniae and idiopathic intussusceptions the mortality for the remaining cases in which the diagnosis was not immediately obvious was 37.9 per cent.

Why, Burgess asked, do patients die of acute intestinal obstruction? There was no adequate answer forthcoming. It was easy to see that

obstruction, if allowed to continue, killed the patient, and operation was therefore undertaken to relieve it. The precise mode of death was not, however, clearly understood and this failure adequately to analyze the lethal factors at work left a considerable gap in the knowledge of the principles of treatment, which should have been designed to counteract the secondary effects of obstruction in addition to relieving the obstruction itself.

It is clear now that the theories of the day, for instance that deprivation of bile, or toxæmia from the activity of the *Cl. Welchii*, killed the patient, were wide of the mark.

In taking stock of the position today, it is therefore right that we should go back to this point and consider pathology first, asking ourselves whether we now know the answers to these questions which eluded our predecessors in the

1920s and, in fact, have a clear picture of the precise nature of the pathological changes and lethal factors at work in the various types of acute obstructions. I propose, therefore, to approach the problem of the management of the acutely obstructed patient by discussing pathology at some length, for without a detailed knowledge of pathology no accurate plan of therapy can be put forward.

PATHOLOGY

It is convenient to describe acute obstructions under the following headings:

(1) Mechanical obstructions—(a) Simple occlusion; (b) closed loop obstruction; and (c) intestinal strangulation.

(2) Functional Obstruction.

Simple Occlusion

The effects of simple occlusion of the intestine are, very briefly, as follows:

Below the Occlusion. Collapse of the intestine which remains empty and physiologically defunctioned.

Above the Occlusion. (a) Distension with a rising intra-luminal tension.

(b) Increased peristalsis, becoming more violent and ill-co-ordinated with the passage of time, but finally giving place to inhibition of peristalsis due to gross stretching of the intestinal wall and interference with its local circulation.

(c) Increased secretion and decreased absorption of fluid.

(d) Increased content of gas, mainly from swallowed air but also from bacterial fermentation and diffusion of gases from the blood stream.

(e) An increase in the bacterial content of the intestine accompanied by a change in the character of the flora which, in the main, tend to be of heightened virulence.

(f) A variable degree of vascular damage to the intestinal wall.

The relative importance of these primary changes varies according to the site of the occlusion and the function of the intestine proximal to it.

High Occlusion of the Small Intestine

In high occlusion the picture is dominated by the loss of the absorptive function of the small intestine. Normally, some 5 to 7 litres of fluid per day (Rowntree, 1922) are poured out into the upper gastro-intestinal tract as digestive secretions, reabsorption occurring in the lower small intestine. In the presence of a high occlusion not only is the patient unable to absorb fluid taken by mouth but in addition he may also lose an enormous volume of secreted fluid, some of which is flung back into

the stomach and vomited, the rest remaining stagnant in the occluded, dilating coils of intestine.

The patient with an unrelieved high occlusion dies rapidly of dehydration and loss of chloride, which between them cause:

(a) A lowered blood volume with haemoconcentration and increased viscosity of the blood, with slowed capillary circulation and generalized anoxia, including anoxia of the myocardium, medullary centres and kidneys.

(b) A sharp fall in the chloride content of the plasma and a compensatory rise in the plasma bicarbonate, a rise in the blood urea and non-protein nitrogen due to concentration, increased production and decreased elimination.

There is no need to look any further for lethal factors in high occlusions or to incriminate some hypothetical toxin. Note also that writers who refer to patients with a high occlusion as being 'shocked' are entirely correct, for the closer one scrutinises the circulatory changes occurring in surgical shock and in acute high occlusions, and for that matter in the post-haemorrhagic state, 'burn toxæmia,' general peritonitis or the 'crush syndrome,' the clearer it becomes that they are but different facets of one problem and not entirely separate and discrete entities.

Low Small Intestinal Occlusions

In low occlusions the loss of water and salt from the circulation is less rapid and less severe. Nevertheless, although a greater length of intestine whose normal function is absorptive lies above the occlusion, this function is grossly interfered with by dilatation and circulatory impairment. Thus, although vomiting is later in onset and usually less profuse, one should not forget that fluid stagnating in a dilated, obstructed intestine whose absorptive function has been lost is just as certainly lost to the circulation as vomited fluid. All observers are satisfied that loss of fluid and electrolytes from the circulation is a very important lethal factor in low as well as high occlusions. There is no such general agreement, however, on the importance of toxic absorption, for some hold that a toxin formed in the intestinal lumen above an occlusion and absorbed into the circulation with generalized effects is a myth, while others consider it the main cause of circulatory failure in many cases.

The truth, as is so often the case, probably lies somewhere between these two extreme views. My own view is that loss of fluid and electrolytes from the circulation is the most important single lethal factor, but that toxic absorption probably does occur to a limited extent in those cases in which damage to the intestinal wall has resulted from unrelieved distension and circulatory embar-

raiment. True toxæmia then occurs and, although it may well be that the amount of toxic substance absorbed is small and possibly insufficient to bring about the failure of a normal circulation, we must remember that the circulation is not normal, but already struggling to cope with dehydration and electrolytic imbalance and may be on the verge of failure already.

Occlusions of the Large Intestine

Acute occlusion of the large intestine usually follows a stage of chronic incomplete obstruction, for by far the commonest cause is a carcinoma. The effects of the colonic distension depend largely upon the function of the ileo-caecal sphincter, which may or may not allow an early 'blow-back' into the ileum, which then dilates in exactly the same way as in an occlusion of the lower ileum itself.

Life is in danger from:

- (a) An associated ileal obstruction.
- (b) Peritonitis, which may occur in several ways: (i) The actual obstructive lesion may itself perforate, e.g. a carcinoma; (ii) gross dilatation may result in necrosis of part of the intestinal wall, often in the caecum, but it may occur anywhere above the obstruction; and (iii) contamination of the peritoneum with faecal contents may occur at operation or after it, for instance if a suture line leaks, and lead to post-operative peritonitis.

Closed Loop Obstructions

Closed loop obstruction without strangulation is of rare clinical occurrence except for two conditions: obstruction of the large intestine in the presence of a competent ileo-caecal sphincter and obstruction of the appendix. It can also occur in obstruction of a Meckel's diverticulum. Nevertheless, it is of considerable interest, for it is easily produced experimentally at various sites, and observations do shed light upon the pathology of more usual forms of obstruction.

Analysis of the relevant literature suggests that three lethal factors are of importance. Firstly, stimulation of afferent nerve endings, caused by a rise in intra-luminal tension, reflexly causes interference with peristaltic function over a wide area, resulting in colic, distension and vomiting, the picture being very like a simple occlusion at the same site. Secondly, a phase of peripheral circulatory depression occurs as a result of loss of fluid and electrolytes from the circulation into the closed loop and the intestine above it, possibly accentuated by toxic absorption from the loop itself. Lastly, perforation of the loop and peritonitis may occur. These three phases are seen clearly in a typical attack of acute appendicitis,

which is the commonest condition in which a true closed loop is encountered.

Intestinal Strangulation

Of the many varieties of acute intestinal obstruction, the most fatal are those in which the intestine is deprived of its blood supply. The term strangulation is often used to denote any case of this kind, whether the blood supply is in fact truly 'strangled' by an occluding band or the tight neck of a hernia, or obstructed by thrombosis or embolism of vessels in the mesentery.

Long and medium loop strangulations are of uncommon clinical occurrence, incidence being confined to cases of mesenteric thrombosis or embolism and of volvulus of a considerable coil of intestine.

Mesenteric Vascular Occlusion

This may be arterial, venous or both, and thrombotic or embolic.

Arterial occlusions are slightly more common than venous (Jackson, 1904). Primary thrombosis is rare, the pathology most often found being thrombosis on top of an original embolism. Aetiological factors include endarteritis, atheroma and arteriosclerosis, rheumatic endocarditis and auricular fibrillation. Although one might expect that an ischaemic infarct would follow arterial blockage, this is rare, an haemorrhagic infarct being much more common.

Venous thrombosis may occur as a result of local sepsis, cirrhosis of the liver with portal hypertension, and polycythaemia vera. An haemorrhagic infarct is the usual result, with rapid capillary engorgement and the out-pouring of a blood-stained fluid into the lumen and tissue spaces of the intestine and into the peritoneal cavity. Oedema of the intestine and its mesentery soon becomes marked and if survival continues long enough may eventually lead to a secondary occlusion of the arterial circulation. As in arterial occlusions, initial blockage of a main vessel is extremely liable to be followed by an extension of the thrombosis into the arcades and smaller veins.

Death usually occurs before gangrene has been followed by peritonitis. The cause is the loss of fluid from the circulation, which in the case of a long loop may produce a most extreme and rapid state of circulatory collapse, the clinical picture resembling one of severe internal haemorrhage, which in fact is precisely what is occurring.

Short Loop Strangulations

The commonest cause of a short loop strangulation is strangulation of an external hernia. The pathology is best considered under two headings,

changes above the strangulated loop and changes in the strangulated loop.

The changes occurring above a strangulated loop are exactly the same as those occurring in a case of simple occlusion at the same level. The changes in the strangulated loop are again similar to those described as occurring in long and medium loop strangulations, the difference being merely one of degree. In the vast majority of cases the occlusion is venous, at least to start with, and the first phase is one of increased capillary permeation with the out-pouring of a blood-stained fluid into the lumen and tissue spaces of the intestine and into the hernial sac or peritoneal cavity in which the intestine is lying. Later, gangrene and perforation of the intestine may occur. One other factor may be mentioned at this point. A short loop strangulated in a hernia in a peritoneal fossa, or having undergone torsion, differs from a loop deprived of its blood supply by mesenteric vascular occlusion in that it is also a closed loop. Stimulation of afferent nerve endings by a very high intra-luminal tension may well be a factor of some importance in the production of the vaso-vagal shock often seen early after the onset of a strangulation.

The main causes of death in short loop strangulations are loss of fluid and electrolytes from the circulation into the occluded intestine above the strangulated loop and, in later cases, peritonitis. It is doubtful what additional part is played by the absorption into the circulation of a toxic substance from the strangulated loop. Experimental evidence has certainly shown that a powerful circulatory depressant is produced by the septic autolysis of a loop of bowel strangulated and allowed to become gangrenous. It is, however, less easy to be sure that this factor is of great importance clinically. It is likely that before toxin has been produced by autolysis in any significant quantity, dehydration and electrolytic imbalance will be well established and peritonitis will have been added to this.

Functional Intestinal Obstruction

Functional intestinal obstruction means obstruction due to derangement of peristalsis in the absence of mechanical occlusion. Very occasionally functional obstruction may be spastic in character, a segment of intestine in spasm acting as an organic occlusion, but the paralytic variety is far commoner.

The causes of paralytic ileus may be classified thus:

(1) *General Causes* :

(a) Circulatory failure: (i) pneumonia; (ii) uraemia; (iii) 'shock syndrome'—dehydration; and (iv) other advanced toxic states.

(b) Neurogenic causes (splanchnic irritation or a reflex via the splanchnic nerves): (i) renal causes—trauma, renal colic, etc.; (ii) retro-peritoneal haematoma or cellulitis; (iii) acute pancreatitis; and (iv) any severe generalized trauma, particularly if painful, e.g. multiple fractures.

(2) *Local Causes* (damage to the neuromuscular structure of the intestine causing paralysis through a local effect as well as through reflex inhibition of peristalsis via the splanchnic nerves):

(a) Gross distension, stretching of the actual muscle fibres.

(b) Vascular damage to the intestinal wall.

(c) Trauma at operation.

(d) Peritonitis.

Of these the local causes are the most important and, in general, one may say that, apart from the normal temporary physiological ileus occurring after any laparotomy and lasting about 12 hours or so and perhaps up to 24 or 36 hours after a major procedure, true paralytic ileus is rare except in the presence of general peritonitis.

Nevertheless, whatever the cause of a paralytic obstruction, in the absence of peristalsis the intestine becomes filled with fluid and gas and there is a steady rise in intra-luminal tension. The resultant stretching of the intestinal wall reinforces other causes producing ileus and, just as in any other variety of acute obstruction, vascular changes soon occur, starting with capillary engorgement and oedema and ending with intra-mural thrombosis and mucosal ulceration and even spontaneous perforation of the intestinal wall. It is probable that some passage of organisms can occur through the intestinal wall even before actual perforation has taken place. In any event, the ultimate end of a patient with an unrelieved paralytic obstruction is practically every case is death from, or with, peritonitis.

DIAGNOSIS

So much, then, for the problems of pathology. Accepting the preceding section as a basis, we have some idea of the lethal factors involved and can plan our therapy accordingly. First, however, a brief summary of important points in diagnosis may be of value, for it is self-evident that early diagnosis of the presence and site of obstruction is the first essential of successful treatment.

Pain, vomiting, abdominal distension and constipation are the four most common manifestations indicating obstruction and usually present in that order. The pain is colicky and ill-localized, being situated mainly above or around the umbilicus if the small intestine is obstructed, and below it if the large intestine is obstructed. Constant pain, persisting between bouts of colic, indicates

peritonitis or tension on a mesentery, for instance by a band of volvulus. Marked abdominal tenderness with rigidity, guarding or rebound tenderness has the same significance, though some degree of tenderness can be caused by extreme intestinal dilatation in the absence of strangulation.

The type of vomiting in acute obstruction is well known, beginning with a reflex vomit of gastric contents and later becoming more profuse, containing bile and finally dark intestinal fluid.

The degree of distension is greater in low than in high small intestinal obstructions and is greater still in obstructions of the large intestine. An acute volvulus of a redundant loop of pelvic colon causes particularly gross abdominal distension.

Absolute constipation is almost invariable, perhaps after one bowel action soon after the onset of obstruction, though there are exceptions.

One or two other points may be mentioned. A dirty tongue and offensive breath are present in all but the very earliest cases. A rectal examination is essential and the hernial orifices must always be carefully examined. It is only too easy to miss a small strangulated femoral hernia in a fat patient. Peristaltic sounds heard on auscultation of the abdomen provide most important evidence. In acute organic obstructions there are frequent exaggerated sounds and the distended intestinal wall imparts to them the high-pitched 'tinkling' note which is so characteristic. Paralytic obstruction, on the other hand, can be identified by the complete silence on auscultation. Visible peristalsis and coils of intestine arranged in a visible 'ladder pattern' are only seen if the abdominal wall is thin and atrophic.

The use of enemata to demonstrate the presence of obstruction has long been recognized. If obstruction is present, a result is often obtained with the first enema, but a second and subsequent enemata are returned clear. While this method of examination may be of use in some doubtful cases, clinical and radiological evidence is in most cases sufficient to make a certain diagnosis. Further, the value of subsequent radiological examination is vitiated by giving an enema.

Radiography in Acute Obstructions

Plain radiography of the abdomen is now a standard investigation in any case where the existence or the nature of obstruction is uncertain. The physical basis of the method is that gas and liquid, recognizable in the plain film, accumulate in the intestine above the obstruction. In the erect position the association of gas and fluid gives rise to a series of fluid levels. In the supine position the film shows the amount and distribution of gas in the bowel. The pattern and

size of the distended loops confirm the presence of obstruction and indicate the site.

Those unfamiliar with the technique are apt to condemn the method as too disturbing for a patient ill with intestinal obstruction. In reality there is less upset than that entailed by the conventional double enema, and the information gained from radiographs is far in excess of that gained by a double enema.

A few films are shown here as illustrations of various types of acute obstruction.

TREATMENT

The subject of this paper is the management of acute intestinal obstructions and we seem to have been rather a long time arriving at a discussion of therapy. Nevertheless, I personally have always disliked having the treatment of some condition detailed to me with no reasons given and, to my mind, it is indeed impossible to develop a sound judgment in treating the many different varieties of acute obstruction unless the reasons behind the various elements in therapy are fully understood and based on a sound knowledge of pathology.

When an acutely obstructed patient is admitted to hospital, provided the general condition allows it, he should call on his way to the ward at the X-ray department for plain X-rays of the abdomen to be taken, unless the presence of obstruction and its nature are at once obvious, for instance in the case of a strangulated external hernia.

Once admitted to the ward, every effort should be made by clinical examination and study of the X-ray films to determine not only if obstruction is present but, if so, the site and nature of the obstructing agent. The degree of dehydration must now receive special scrutiny and by studying the tongue, state of the subcutaneous tissues, pulse rate and volume, amount, specific gravity and chloride content of the urine, and chloride level in the plasma, an assessment must be made of the amount of water and salt required to restore the patient's circulation to normal. An intravenous drip infusion of normal saline or 5 per cent. dextrose in one-third normal saline, depending upon the need for chloride, is then set up and a Ryle's tube is passed into the stomach and connected to an apparatus for continuous suction.

A decision must now be taken on whether to operate and, if so, how soon. Certain cases must be operated upon with the minimum of pre-operative delay. These include:—

1. All acute obstructions in children.
2. All acute obstructions in adults seen within the first 48 hours, except certain cases of post-operative obstruction which will be discussed later.

3. All cases in which intestinal strangulation is possible.

Even in cases falling into one of these groups one may sometimes deliberately delay operation for an hour or two if the general condition is particularly bad, in order to allow an intravenous drip and suction drainage of a grossly dilated upper gastro-intestinal tract time to bring about some improvement. If this is the case, one cannot lay down hard and fast rules about exactly how long one can afford to wait for this improvement. One can say, however, that re-assessment at least every hour is essential¹.

Morphine should be withheld until a decision has been made when to operate, and is then preferably given by the intravenous route if circulatory depression is at all marked. Pre-operative treatment may also include giving oxygen which undoubtedly has a useful effect upon the very shocked and grossly distended patient. It should not be used unnecessarily, however, for over-complicating the treatment by attaching the patient to as many tubes as possible is to be deprecated.

The Conservative Plan of Treatment

The conservative or non-operative plan of treatment is based upon the knowledge that patients with a simple occlusion of the small intestine or with paralytic ileus die because they have lost water and salt from the circulation and because their upper gastro-intestinal tract is put out of action by gross dilatation. Both of these factors can be modified or eliminated without operation—hence the conservative plan. It should be reserved for those cases in which symptoms have been present for over 48 hours, in an adult, with evidence sufficient to exclude a strangulating obstruction. It is particularly useful in dealing with obstruction caused by multiple sites of adhesion and kinking, the result, for instance, of a widespread peritonitis.

The basis of the treatment is intestinal suction drainage and intravenous provision of water and salt sufficient to correct established dehydration and then supply the body's normal needs. A Ryle's tube in the stomach is insufficient for this purpose and some form of intestinal tube, such as the Miller-Abbot, is necessary. Intubation of the small intestine with the double-lumen tube is by no means a rapid, simple process. The stomach is first emptied and then the Miller-Abbot tube passed into it, weighted with a little mercury in the balloon. The patient lies on the right side and serial X-rays are taken until the balloon is well into the duodenum. It is then inflated and suction begun, when the tip of the tube is carried down the small intestine to the site of obstruction; emptying and decompressing the small intestine

as it goes. This sounds simple and it is, once the tube is in the duodenum. Unfortunately, the tube does not pass easily through the pylorus in those cases in which intestinal suction drainage is most desirable, for although the tube will slip comparatively easily through the pylorus in a normal subject undergoing some physiological investigation, it is quite a different matter if there is a stream of intestinal fluid constantly welling back into the stomach, as is the case in a late obstruction. The tube tends to kink back on itself and lie coiled in the stomach and even tie itself in a knot. My experience with the Miller-Abbot tube has been a little disappointing because in a fair percentage of cases intestinal intubation has failed and the conservative plan has had to be abandoned. Nevertheless, increasing familiarity does bring a higher percentage of successful intubations and there is no doubt that in a certain group of cases intestinal suction drainage is more likely to meet with success than is immediate surgical intervention.

Suppose, for instance, a patient undergoes appendectomy for gangrenous appendicitis and peritonitis and after operation develops a severe general peritonitis and paralytic ileus. This functional obstruction is better treated by continuous suction of the small intestine than by operation. Indeed, there is little chance of intervening effectively by opening the abdomen. Mere drainage of the peritoneal cavity is useless or even harmful unless a localized abscess is present, while enterostomy carries a mortality of approaching 100 per cent.

Again, supposing this patient survives his severe peritonitis and after many weeks leaves hospital with coils of intestine glued together and to the abdominal wall. He may now, after one or two attacks of incomplete obstruction, develop an acute, complete obstruction. If, as often happens, he neglects this, expecting his symptoms to subside, as have his previous attacks, and arrives in hospital obstructed for several days, grossly distended, grossly dehydrated and in gross electrolytic imbalance, laparotomy carries a considerable risk to life and the division of multiple adhesions attached to the oedematous, friable, dilated intestine is technically difficult. Provided a correct diagnosis can be made and a strangulating obstruction excluded, and provided the intestinal tube can be persuaded through the pylorus, the conservative plan is much more likely to meet with success. At best, intestinal decompression cures the obstruction itself which, due in part to kinking and oedema, disappears once the intestine is deflated. Even if the obstruction itself remains unrelieved and laparotomy ultimately becomes necessary, the prognosis is very greatly improved,

for it is much safer to operate upon a patient whose dehydration and lack of chloride has been corrected and whose intestinal tract is no longer grossly distended.

It will be apparent that if the conservative plan is employed, frequent re-examination and X-ray control of the patient is necessary, to be certain that fluid and chloride balance is maintained and to follow the progress of intestinal decompression. Clinically, one must pay particular attention to pulse rate and blood pressure, state of the tongue, amount and specific gravity of the urine, chloride content of the urine and blood and the state of peristaltic activity ascertained by auscultation of the abdomen.

So much for pre-operative and non-operative treatment. Certain general remarks may now be made about operating upon the obstructed patient.

without much difficulty between small and large intestinal obstructions, between high and low small intestinal obstructions and to localize the site of large intestinal obstructions with a fair degree of a curacy.

If the abdomen is to be explored, whatever incision is employed let it be a big one. It is no economy in time or labour to try to operate with inadequate access to the site of obstruction. If a long incision is made, the time expended in sewing up the added length is amply compensated for by the time gained in performing the essential steps of the operation unhampered by mechanical difficulties which are avoidable.

Operative Technique

It is beyond the scope of this paper to enumerate the qualities demanded of a good abdominal sur-

FIG. 1.—A Miller-Abbott double-lumen tube.

The Anaesthetic

Muscular relaxation being very important, a light general anaesthetic supplemented by curare is a most satisfactory anaesthetic.

The Incision

Before embarking upon the operative relief of an acute obstruction, every effort should be made to localize the site as accurately as possible. The incision should then be placed to give the best access. When the site of the obstruction is not obvious from clinical signs, the procedure formerly advocated was to open the abdomen through a low right paramedian incision and examine the caecum, its distension or lack of it at once localizing the obstruction to the large or small intestine. In the light of the information gained by the study of plain X-rays it is now possible to distinguish

geon, but certain of these qualities are particularly desirable in dealing with the acutely obstructed patient. Of these, gentleness must be valued very highly. The patient with gross distension is particularly susceptible to trauma, to which he may respond with circulatory collapse during the operation or paralytic ileus after it.

The following points are worth stressing:—

1. Make a big enough incision in the right place.
2. Handle distended coils of intestine gently and do not pull on the mesentery.
3. After relieving the obstruction do not empty the dilated intestine by 'stripping.'
4. Be gentle with clamps. Leave them on only as long as is necessary. Clamps used to keep a segment of intestine empty should be rubber shod and closed only with sufficient tension to achieve their purpose.

FIG. 2.—Acute obstruction of the ileum. Gas shadows in the supine film are of typical small intestinal distribution and outline well the valvulae conniventes. Many fluid levels in the erect film (right).

5. Do not eviscerate unnecessarily, but if the actual site of obstruction cannot itself be drawn out through the incision and access is obscured by dilated coils of intestine, these should be allowed to prolapse out of the abdomen and be received into warm moist gauze squares and their weight should be supported by an assistant. The heavy, distended coils of intestine should not be allowed to hang over the side of the abdomen with their weight supported by the mesentery.

6. Do not employ heavy retraction. If adequate access is not possible without it, the incision is not large enough.

7. Be sparing and gentle with gauze squares. If distended coils of intestine are to be held out of the operating field, they should either be allowed to prolapse as described or be gently placed in the quadrant of the abdomen desired, covered with a moist gauze square and controlled by the gentle retraction of an assistant's fingers, not raked out of the way by a process resembling gauze dissection and then left with multiple squares interleaved among the dilated coils.

General Principles of Post-operative Treatment

Details of post-operative treatment differ considerably according to the site of the obstruction and the operative measures found to be necessary. Nevertheless, certain general observations may be made.

The post-operative management is dominated by the necessity, in the majority of cases, of employing continuous suction drainage and therefore of giving fluids intravenously. This should continue until the resumption of normal intestinal motility and absorption ensures that passive dilatation of the intestine will not occur if suction is withdrawn. Evidence that this stage has been reached is provided by the observations that:—

(a) On auscultation, peristaltic sounds can be heard.

(b) If gastric suction drainage is being employed, the fluid withdrawn by suction no longer contains bile and the fluid balance chart shows satisfactory absorption of fluid.

(c) Plain X-rays, if taken, show (i) Gas shadows

in the large intestine; (ii) No dilatation or fluid levels in the small intestine.

Particular care should be taken not to stop suction too soon in cases complicated by peritonitis or in which a resection of intestine or a short-circuit operation has been necessary.

Inhalation of oxygen in the early post-operative period is of value, particularly in those cases in

but it must be stressed that for an enema to be effective it must be the large intestine which is distended. If clinically this is in doubt, plain X-rays settle the point. The way *not* to treat a case post-operatively is to withdraw suction drainage too soon and then to treat the resultant abdominal distension and discomfort by giving purgatives, enemata, pituitrin and the like without bothering

FIG. 3.—Acute obstruction of the large intestine by a recto-sigmoid carcinoma. Gas shadows outline greatly dilated colon. Erect film shows a long fluid level in the caecum.

which gross distension or circulatory failure is a prominent feature.

Morphine is a most valuable drug particularly if given in small repeated doses of $\frac{1}{8}$ gr. to $\frac{1}{6}$ gr. 8 to 12 hourly.

The question of employing aperients or enemata is one which crops up sooner or later in most cases. Provided the small intestine is kept decompressed by suction, there need be no anxiety if several days pass without a bowel action. Spontaneous resumption of normal intestinal motility is likely and this may be accompanied by a certain amount of discomfort from distension of the large intestine with gas. An enema will then give material relief,

to find out whether it is the small or large intestine which is dilated.

Individual Varieties of Acute Obstruction

It is manifestly impossible to deal adequately with all the different types of acute obstruction and their detailed management. Certain brief observations may be made about some of them.

Strangulated External Hernia (inguinal, femoral, umbilical, ventral and other rarer types).

A strangulated external hernia must be treated by early operation if the intestine is involved. Pre-operative gastric intubation is essential and,

FIG. 4.—Acute obstruction of the large intestine and associated ileal obstruction due to incompetence of the ileo-caecal valve. Distension and fluid levels in both large and small intestine.

except in the very early case with no dehydration, an intravenous drip is advisable.

At operation the hernial sac is opened and the dark fluid exudate in it sucked or mopped out. When the loop of intestine has been released from its constriction it is drawn down so that it can be

carefully examined, particularly the two constriction grooves where the intestine has been actually compressed at the neck of the sac. The viability of the loop is then assessed with particular regard to the following factors:—

Colour	Viable	Not Viable
	Red→Dark Purple	Black→Green
State of the visceral peritoneum	Peritoneal sheen still present, though there are, perhaps, small subserous haemorrhages.	Lustreless, shaggy appearance of the intestine.
State of the mesentery.	Oedema of the mesentery but pulsation felt in the larger vessels. No major thrombosis.	Thrombosis of main vessels. No pulsation felt.
Reaction to warming in towels wrung out in warm saline.	Colour improves from plum colour to red. Peristaltic wave seen to pass along the loop.	Colour fails to improve. Still no pulsation in the main vessels. No peristaltic wave.

FIG. 5.—Volvulus of the caecum. A greatly dilated caecum lies in the centre of the abdomen.

FIG. 6.—'Closed loop' obstruction in the jejunum. This was caused by a war wound (note retained shell fragment) and a band across the bowel. The Miller-Abbott tube could not enter and decompress the loop, which was released by operation.

In the case of small intestinal strangulations, if it is decided that the loop of intestine must be removed, resection and anastomosis is the method of choice and should only be abandoned in favour of exteriorization if the desperate condition of the patient dictates a rapid termination of the operation. Occasionally invagination of a doubtful constriction groove is advisable if the rest of the loop is clearly viable.

If the large intestine is strangulated, which is rarely the case, the principles of treatment are similar, but their application is modified by two important facts about the large intestine.

1. Resection and primary anastomosis of the large intestine in the presence of acute obstruction is notoriously a dangerous procedure.

2. Invagination of a doubtful constriction groove by a layer of Lembert sutures is not safe on account of the degree of distension which the large intestine commonly exhibits after operation, which is much

greater than that seen in the small intestine and not under the control of suction drainage.

The treatment, then, of a coil of strangulated large intestine after its release is to return it to the abdomen if there is no doubt about the viability of the coil or the constriction grooves. If, however, the viability is in doubt, a resection of the Paul type should be carried out, leaving a double-barrelled colostomy through a separate incision in the flank, which is closed later.

Obstruction by Bands and Adhesions

Cases fall roughly into two groups. In the first variety of case, the onset is abrupt and a diagnosis of acute obstruction can be made with certainty within a few hours of the onset of symptoms. In a fair proportion of cases, signs suggesting a strangulating obstruction appear and laparotomy is clearly necessary. In the second class of case, vague abdominal colic has been present for a day

or two, accompanied by growing abdominal distension and sometimes vomiting. Advice may be sought at this stage, before the condition becomes truly acute, but often on this stage complete obstruction supervenes, with increasing colic, vomiting, distension and dehydration, but no sign or symptom at all suggestive of an intestinal strangulation.

Of these two clinical types, the first is usually due to obstruction by a band or to a volvulus associated with a band or adhesion, though multiple adhesions may be present in addition. The second is due to many adhesions with multiple sites of partial obstruction, one or more of which become complete.

It is important to recognize these two types of case, for the treatment is not the same. If the onset is sudden with nothing suggestive of previous partial obstruction, the likelihood of a strangulating obstruction being present is sufficiently high for expectant treatment to be unjustifiable. Early laparotomy must be carried out. If acute obstruction supervenes upon a stage of subacute, partial obstruction, particularly if there is a history of premonitory minor attacks, if careful clinical examination reveals no sign or symptom of strangulation and if the total duration of acute symptoms is not less than 48 hours, the conservative plan of treatment may be employed.

Volvulus

Any coil of intestine with a mesentery may undergo rotation, with intestinal obstruction and strangulation of its blood supply. Parts of the intestine not normally on a mesentery, such as the caecum, may also be affected if a congenital error in the development of the peritoneal attachments leads to an abnormal mesentery being present. The danger of gangrene dictates in all cases an early operation, at which the intestine is untwisted and returned to the abdomen if viable. If not, resection and anastomosis is carried out for a volvulus of the small intestine or a Paul's colectomy for an acute gangrenous volvulus of the large intestine, almost always affecting a redundant sigmoid colon.

Intussusception

Intussusception is not initially a variety of intestinal obstruction. Indeed, the ideal of treatment is to reduce the intussusception before obstruction has time to occur. Early diagnosis is thus all-important, particularly in the idiopathic intussusception in babies, for whom delay may spell disaster. Two points in diagnosis not sufficiently stressed in some textbooks may be mentioned. Firstly, an early vomit is almost invariable. Secondly, the passage of blood per

rectum does not occur until considerable vascular damage to the intestine has taken place. One should try to make a diagnosis early enough to intervene before this stage is reached. Operative reduction of the intussusception is then carried out. If it is already irreducible, resection or exteriorization is performed, either carrying a very high mortality.

Acute Obstruction by a Neoplasm

Acute obstruction by a growth nearly always affects the large intestine rather than the small, and follows a period of increasing partial colonic occlusion due to a stenosing carcinoma, usually of the left colon.

It is generally accepted that resection of the growth itself in the presence of acute obstruction is bad treatment. Certainly resection and anastomosis is unsafe and even a resection of the Paul variety has disadvantages, for in the presence of colonic distension, which is always marked in an acute large intestinal obstruction, the mesentery is shortened and oedematous and this undoubtedly makes it difficult to remove enough of the lymphatic drainage of the part to be satisfied that one has done a good cancer operation. One should therefore treat first the acute obstruction and then, later, the carcinoma, and be satisfied with a proximal defunctioning colostomy, with a spur, performed preferably without exploration of the area of the growth. Not everyone, of course, agrees entirely with this, but I feel very strongly that little can be gained by a full abdominal exploration. Certainly it is not possible to assess local operability with any accuracy, for a fixed, apparently inoperable growth may very easily become mobile and removable when a proximal colostomy for two or three weeks has allowed local oedema and inflammation to subside. Furthermore, it is by no means unknown for the first insertion of an exploratory hand to rupture the softened, distended colon, an accident which is usually fatal.

The stages in the treatment of acute large intestinal obstruction by a carcinoma are, therefore as follows:—

1. Clinical and radiological evidence must exclude a strangulating obstruction or exploration is obligatory.
2. Plain X-rays must be studied, if the growth itself cannot be palpated, and the site deduced.
3. If this is the rectum or pelvic colon, a transverse colostomy is done through the upper right rectus.
4. If the site is in the left transverse colon, splenic flexure or descending colon, the ascending colon is used, after mobilization.
5. If the growth is in the caecum, ascending

colon, hepatic flexure or right half of the transverse colon, it is short-circuited by ilio-transverse colostomy and later resected.

Intra-Luminal Impaction

Gallstone obstruction is notoriously diagnosed late, for the stone may become impacted and dislodged several times in its journey down the small intestine before it reaches its final site of irremovable impaction. Obstruction may thus be intermittent to start with and lead to unfortunate delay in instituting treatment. Plain X-rays often help in the diagnosis, demonstrating obstruction of the small intestine, gas shadows in the biliary tree and, possibly, the stone itself.

Treatment consists of pre-operative intubation and correction of dehydration, followed by enterotomy and removal of the stone. Owing to incarceration in the wall of the bowel it is not always possible to push the stone up into a segment of intestine undamaged by pressure necrosis, as advised in some textbooks. In either event, the incision in the bowel wall should be made longitudinally and sewn up transversely, to avoid the formation of a stricture from the contracture of scar tissue.

Mesenteric Vascular Occlusions

Pre-operative diagnosis can often be made, particularly if obvious aetiological factors are present, such as cirrhosis of the liver or auricular fibrillation. Signs of obstruction are accompanied

by severe peripheral circulatory failure, abdominal tenderness, guarding and rigidity, rebound tenderness and ascites. Treatment consists of transfusion with whole blood and laparotomy, gangrenous or devitalized intestine being resected. Post-operatively, anticoagulants are used to prevent spread of thrombosis into uninvolved vessels.

If the whole small intestine is involved by a venous thrombosis, the patient probably has a better chance if heparinized than if the whole small intestine is resected, provided that actual gangrene has not already occurred.

Infarction of the large intestine is particularly lethal, though fortunately rare. It is treated by resection or exteriorization according to the site and the condition of the patient.

It is impossible to do justice to the many very different varieties of acute intestinal obstruction in a paper of this length. It is hoped, however, that it will provide some pointers to the way one should start thinking about obstructions and the important lethal factors one must consider. As a final word, I would reiterate the following two important considerations:—

1. If the blood supply of the intestine is in jeopardy, early operation must be performed. Gangrene of the intestine must not be allowed to occur.
2. If this is not the case, pre-operative correction of dehydration and electrolytic imbalance and decompression of the upper gastro-intestinal tract by suction greatly improve the prognosis.

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