I am aware that even more extensive plastic operations have been described in the past, but these I regard as applicable only to such exceptional circumstances that they do not come within the scope of this survey. I am also aware that some surgeons still advocate the use of foreign materials such as silver filigrees for inguinal hernias and iron staples for femoral hernias; but although these methods may give excellent results in some hands, I believe that they are contrary to the general trend of modern surgery, which prefers to discard such artificial aids when cure can be obtained without the introduction of any foreign materials.

ACUTE INTESTINAL OBSTRUCTION DUE TO INTRA-ABDOMINAL CAUSES.

BY

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In the earlier years of my surgical experience there was a very decided prejudice against the employment of surgical measures in the treatment of acute intestinal obstruction. This prejudice was, in a measure, not to be wondered at, because the results of surgical intervention at that time, when surgery was a "last resort," were extremely depressing.

In those days, judging from my experience in Scotland, there were, relative to the number of operations performed, a good many more cases of acute intestinal obstruction compared with the number encountered in more recent years. A considerable proportion of cases occurred as a sequel to abdominal operations. That number was greatly reduced owing, I think, to more rational and careful methods of conducting such operations and their after-treatment. For example, one important item in preventing adhesions and band formation is strict attention to the exclusion of raw surfaces by "peritonealization" of pedicles and careful suturing of the abdominal wall. The irritation of rubber tubes and other stiff material used as intra-abdominal drains stimulates adhesions, from the formation of fibrous tissue, and predisposes to obstruction from kinks or bands. I have for many years almost entirely given up the use of such intra-abdominal drains as a routine, except after operations on the biliary passages, liver, or pancreas.

All cases of intestinal obstruction are urgent—some more so than others. This urgency is dependent on the age and general condition of the patient, on the actual cause of the obstruction, and on its site. Very young and very old individuals, or patients enfeebled by concomitant disease of vital organs, are likely to go under rapidly. They cannot endure the intense pain, and may quickly succumb to shock. If pain is not typically severe in such a case the attendant may be tempted to procrastinate, but it must be remembered that the effects of toxæmia are apt to be more intense and rapid in them and to be less easily combated than in more robust individuals.

The effects on the bowel itself vary greatly according to the cause. Slight constriction or simple kinking may at first merely prevent passage of intestinal contents. Later on, increased pressure of these contents, and the congestion due to the thin-walled veins being more readily occluded than the arteries, may ultimately bring about exactly the same state of affairs in this primarily simple occlusion as in the more dangerous condition of strangulation, whether caused by band, volvulus, or intussusception. The degree of interference with the circulation of the bowel, with the nerve supply, with the mucous membrane, and the lymphatics, and with the walls of the gut as a whole, influences the rapidity with which symptoms appear and their severity.

A tight snare caused by a band over or around the bowel may affect the whole of its constituents so much, from the onset, that gangrene will occur rapidly, and be followed by peritonitis. In some cases the snare may merely be tight enough to cause, at first, occlusion only of the veins of the bowel, but this brings on congestion and distension, so that

the snare gradually becomes tighter, with involvement of other structures and corresponding increase in the severity of all the symptoms. The ultimate result of embolism or thrombosis in the mesenteric vessels is practically identical.

Apart from the purely mechanical factor in acute intestinal obstruction, there is another and almost equally important one—the absorption of toxic material from the bowel. This material is derived either from bacterial decomposition of the bowel contents above the obstruction, or from imperfect metabolism of protein contents (producing proteoses), which may have been swallowed, but more likely are secreted or exuded directly into the bowel through its mucous membrane. Toxins of the second class are more dangerous and rapid in their action. Their effect is best seen in obstruction high up in the small bowel, where bacterial decomposition is not prominent. The jejunum has been called the "poisonous proteose level" in obstruction. In these cases toxæmia becomes evident very soon, producing definite prostration or symptoms of actual shock. Persistent vomiting is established at an early stage, and is probably dependent as much on the toxæmia as on mechanical causes. Both symptoms pass off just as quickly as they appeared, after the contents of the bowel are drained away through an enterostomy. The origin of these toxins, however, is still a matter of debate. Apparently the presence of bacteria is necessary before they can provoke a fatal issue. Vomiting and toxæmia may be long postponed when the obstruction is low down in the bowel. A third important factor is the rapid depletion of the body fluid as a whole and of some of its constituents in particular. This has often a marked effect on the general circulation. In some cases the sunken eyes, pallid or "blae" facies and hands, and shrunken limbs are striking features. But these are symptoms of advanced cases of obstruction, which, in ordinary circumstances, at least of town practice, should not be allowed to occur.

ETIOLOGY AND PATHOLOGY.

In infants and young children intussusception is by far the most frequent condition which produces obstruction of the bowels. Improper feeding, causing inco-ordinate and excessive peristalsis, as well as swelling of the lymphoid accumulations which are prominent anatomical features in the wall of the bowel at this time of life, are probably the most frequent causes of this. During ordinary peristalsis, as opposed to segmentation, a distal part of the intestine dilates while the part immediately proximal to this contracts and forces the bowel contents onwards. If the contraction is excessive, the contracted part may be forced into the dilated part, which, in turn contracting, tends to carry this apex of the intussusception still further. This is apt to occur more easily in a child, in whom the equilibrium, so to speak, of the bowel is not so highly developed as in an adult, and these lymphoid swellings are apt to be caught and forced onward by the peristaltic wave. As the process develops, the bowel becomes more and more infolded, causing congestion, swelling, occlusion of the gut and its vessels, and traction on nerves, with attendant hæmorrhage and shock. In early stages the symptoms may abate because the intussusception may recede. But later on it remains strangulated and will speedily become gangrenous. In such cases the symptoms are severe and persistent, until their severity is masked by the exhaustion of the bowel and of the patient.

Children with complete obliteration or high-grade stenosis of a segment of bowel are often in such poor condition that surgical intervention is out of the question. If, however, there is no doubt of an obstruction, as revealed by persistent vomiting, local distension, and possibly by x-ray evidence, it may be worth while to give an otherwise healthy child its only chance. A rapid short-circuiting operation is usually the best procedure. Obstruction at this age may be due to a diaphragmatic hernia or to strangulation through a hole in the mesentery.

Intussusception in older children and adults is usually due to some accompanying pathological condition, most frequently a polypus, and more rarely a stricture. Intussusception or, as it is more often called, prolapse of the sigmoid or rectum, may occur without such accompaniment, and may be predisposed to by a curious ballooning of this

part of the bowel which occurs in some cases and which is not satisfactorily explained. The ballooned condition may have a distant relationship with the equally unexplained condition called Hirschsprung's disease—an idiopathic and apparently congenital condition of the large bowel, in which the lower part is most severely and most frequently affected.

Intussusception in adults is, however, comparatively rare. At this age the most usual causes of intestinal obstruction are fibrous bands, internal hernia, Meckel's diverticulum, volvulus, impaction of concretions, sudden blockage of a chronic stenosis brought on by tubercle or cancer, or pressure from a tumour outside the bowel, and chronic or recent adhesions causing acute angulation of the bowel. The last is usually the cause of obstruction accompanying acute peritonitis from appendicitis or salpingitis, or after an abdominal operation. In inflammatory cases the symptoms of obstruction may mask those of the cause; the case usually becomes much more urgent when obstruction supervenes.

In all these cases there need not be at first any actual microbial inflammation of the bowel wall, so that, with careful coaxing, the bowel usually recovers, after being released. I should like to point out, however, that strong purgative medicines given by the mouth, and endocrine or other selective remedies, such as pituitrin and eserine, administered subcutaneously, sometimes defeat the object with which they are given. Immediately after operation the bowel may not be in a condition to respond to drastic stimulus. The so-called adynamic or paralytic ileus may possibly occur after any abdominal operation, but especially if the bowel has required much handling, and where over-distension has existed previous to operation.

The most frequent and serious form of paralytic ileus is that caused by peritonitis affecting the lower half of the abdomen, which is occupied by both small and large intestine, both of which become affected by the peritonitis—hence Sampson Handley's term "ileus duplex." This term indicates that both large and small bowel are affected and must be treated, in my opinion by caecostomy and jejunostomy; Sampson Handley recommends a jejuno-colostomy. In such cases stagnation occurs in the distended, sodden, flabby loops of intestine, and the poisonous contents of the bowel, acting on the intestinal wall, tend to keep up the paralytic condition. Unless the vicious circle can be broken, either by emptying the bowel above the obstruction as much as possible at operation, or, better, by making one or more openings in the bowel through which its contents gradually drain away, the patient will die.

With reference to the acute toxæmia which occurs especially when the obstruction is situated high in the small intestine, it appears that the poisons absorbed from the upper jejunum, and especially from the duodenum, exert profound metabolic changes, which can be easily appreciated in the blood, and are evidenced by great diminution in the chloride content, increase in blood urea, and in the capacity of the blood for carrying CO_2 . Such changes are early appreciable in paralytic ileus. In the later stages this toxæmia exerts an evil effect upon the kidneys. These organs often undergo acute inflammation, which shows as changes in the tubular epithelium or as a more diffuse nephritis. The urine may contain granular and hyaline casts, albumin, and even occasional red blood cells. It usually becomes normal again rapidly when the obstruction is relieved and the fluid deficiency of the blood is made up. These changes in the blood, especially the deficiency in chlorides, give valuable indication for treatment. The chloride deficiency can be remedied by giving ordinary or hypertonic solution intravenously. J. W. Ross of Toronto¹ has suggested the use of intravenous infusion of hypertonic saline solution (60 to 75 c.cm. of 20 per cent. sodium chloride for an average adult). This suggestion was based on the observation of Hughson and Scarff² that violent peristalsis immediately followed the injection of this solution and persisted for an hour. With more dilute solutions there was a latent period and less active response. They found also that absorption of toxic products from the gut was delayed. Thus the administration of hypertonic saline has three beneficial effects: (a) replenishment of depleted

chlorides, (b) retardation of absorption of toxic bowel contents, and (c) stimulation of active peristalsis. After the hypertonic solution has produced peristalsis, ordinary isotonic solutions may be required to replace lost body fluid.

SYMPTOMATOLOGY.

As has already been indicated, the clinical symptoms of obstruction may vary considerably, chiefly according to the site and the cause of the obstruction. At the same time there are certain phenomena which are almost invariably found, forming quite a characteristic symptom-complex. Over a period of days or even of weeks the patient may be subject to occasional recurring colicky pains, generally in the same part of the abdomen. These pains may gradually or suddenly increase in severity with development of the typical syndrome. Such patients are usually the subjects of adhesions or of growth.

The symptoms, however, usually appear with dramatic rapidity. If a loop of the bowel suddenly becomes acutely strangled, severe local irritation and pain are caused, resulting in variable manifestations of shock or, as it is called by some, peritonism. The patient may become more or less collapsed, with feeble pulse, clammy skin, drawn features, and intense pain in the abdomen. He may vomit owing to the shock, and the lower bowel may respond to the irritation by evacuating its contents. After this preliminary storm may come a more or less pronounced calm, although there may persist considerable steady pain in the abdomen, usually referred to the umbilical region when the small intestine is the seat of trouble, and to the hypogastric region when the large bowel is affected. In some cases the respite may be so definite that the patient drops off to sleep. Very soon, however, the pain becomes more intense, and is accompanied by spasms of colic, which become so severe that the patient writhes in agony. These spasms occur more frequently and are more intense when the small intestine is affected. When the large bowel is blocked the spasms are bad, but, while they may make the patient sweat with pain, he does not usually cry out as does the sufferer from colic of the small intestine.

Persistent vomiting soon sets in. At first any food remaining in the stomach is evacuated, then bilious material, then more or less clear watery material as the contents of the small intestine regurgitate into the stomach, and finally, as congestion and decomposition increase, the vomit becomes brownish—it is often described as being faeculent in character. At this stage vomiting is frequently "forcible," "pump-like," strikingly different from the overflow, in mouthfuls, which characterizes the act when peritonitis and paralysis of the bowel are present and cause steady regurgitated accumulation in the stomach. When the latter type of vomiting threatens there is urgent need for high enterostomy—one or several. The higher the situation of the obstruction in the small intestine, the more severe usually is the persistent pain, the more frequent is the colic, and the earlier is the onset of vomiting. The patient is usually earlier and more profoundly collapsed—the combined result of toxæmia and implication of the superior mesenteric plexus.

If the strangulation or volvulus of any part of the bowel is severe, the congestion and distension of the affected coils may be so great that the resultant persistent pain may almost overshadow the colicky pain which is caused by peristalsis of the bowel above the strangulation. In such cases localized bulging of the abdomen, accompanied by the sensation of a tumour mass on palpation (which used to be called von Wahl's symptom), may develop quite early, and before general distension occurs. This tumour is felt more easily during expiration and under anaesthesia.

The tumour mass, often sausage-shaped, which is frequently felt in cases of intussusception, varies in bulk and position according to the presence or absence of colic and spasm. During a spasm the mass becomes larger and progresses towards the rectum. In the intervals between spasms the mass may be followed with the fingers towards the iliac fossa, the tumour decreasing in size. Frequently it cannot be palpated, but absence of tumour should not interfere with diagnosis; presence of tumour merely confirms it. I have only once felt the apex of an intussus-

ception per rectum. Dance's sign is merely the appreciation of a vacuity in the right iliac fossa from which the caecum, etc., has taken flight.

Pressure with the flat of the hand, though stated to relieve the pain, does not do so, but often aggravates it, especially when a coil is strangulated. A succussion splash in the distended coils above the point of strangulation may be elicited in the intervals between spasms of colic, and may point to the site of the obstruction; this is also sometimes indicated by an area of great tenderness in the abdomen.

While general distension of the abdomen occurs fairly rapidly if the lower part of the small intestine is affected, it may not occur in strangulation of the upper part; the stomach alone may cause localized bulging. Distension is said to occur much later in obstruction of the lower part of the colon or in the rectum. It is suggested that when the small intestine is irritated and blocked, a considerable exudation of fluid occurs into the lumen of the bowel, which is not sufficiently rapidly absorbed, but that when the lower colon is blocked the upper part still goes on absorbing fluid fairly well. This explanation is not quite satisfying. In the large intestine gas-producing bacteria are ordinarily far more numerous than in the small bowel, and the former is, therefore, more tolerant of decomposition than is the small intestine, in which irritation, congestion, and distension are more likely to produce increased and abnormal secretion and inflammatory exudate, which, with the half-digested food material, promote decomposition and gas formation. Owing to congestion of the obstructed loops free fluid may accumulate in the peritoneal cavity, and may even be sanguinolent. In later stages its serous character may be lost owing to the onset of peritonitis.

Obstruction in the small intestine has been shown to render the bowel more permeable, so that imperfectly digested proteins, etc., gain entrance to the blood; this may explain the excessive amount of urea in the blood. This excess disappears at once when the obstruction is relieved and the bowel has resumed its function.

Constipation is a very prominent feature and should always be impressive, because when colic occurs without obstruction it is always accompanied, sooner or later, by the passage of gas or faeces. But here, after the bowel has become emptied of contents below the site of obstruction, constipation becomes absolute, usually neither flatus nor faeces being passed. Since ordinary colic is the result of indigestion, purgatives or enemata are often administered in an attempt to cure constipation when it is present. In cases of obstruction purgatives increase the colic and may bring on more violent vomiting. Because the bowel below the obstruction may have been more or less loaded with faeces, the first or even a second enema may have what is called a good result. But the giving of an enema will not relieve the pain and colic, unless in some rare cases of intussusception or kinking of the bowel where reduction takes place. Further enemata are unproductive. Thus there should not be great difficulty in diagnosis when the pains continue unabated or even exaggerated. The result of the first enema may be disregarded, but if subsequent enemata are followed by the passage of flatus or faeces the probability is against complete intestinal obstruction. If such obstruction is present, constipation is usually absolute after the first enema has evacuated the lower bowel; this fact is a most valuable aid in early decision about the case.

If the obstruction is not relieved the muscular coats of the bowel become exhausted, and on that account pain may be relieved to some extent. At this stage the nervous mechanism of the bowel is liable to become poisoned by toxins manufactured in the stagnating contents ("splanchnic intoxication"); this may result in paralysis or at least paresis of the bowel. Sooner or later organisms penetrate the walls of the bowel at or above the obstruction, add to the paresis, and cause peritonitis. A circumscribed patch of gangrene may occur owing to the pressure of the obstructing band on the outer wall of the bowel. Ulceration, commencing in the mucous coat, may result from the excessive stretching and consequent ischaemia, or one or more loops whose circulation has become obstructed, whether by band or

twist, may become necrotic *en masse*. Perforation may occur through the gangrenous area.

The picture presented by the patient at this time is a very miserable one. Some describe it as a condition of secondary shock. In large part the symptoms are due to more or less profound toxæmia and to loss of fluid, from persistent vomiting and sweating. The face is drawn and pale, often dusky, the eyes are sunken, the skin is cold with clammy sweat. The nose, ears, and fingers may be mottled and blue, with stagnating capillary circulation. The pulse is feeble, thready, rapid; respiration is rapid, often sighing, and the temperature is probably subnormal. There is persistent, insatiable thirst. There is only occasional colicky pain. Vomiting becomes of an overflow character. The patient may be unusually alert mentally; he often does not realize the seriousness of his condition, and the end may come rapidly.

Allowing for individual idiosyncrasies, there is no doubt that the amount of pain in intestinal obstruction varies considerably even while the pathological conditions are very serious. Stenosis and congestion may vary very widely, as may the clinical symptoms. Although usually the intensity of the symptoms is an indication of the severity of the pathological developments, yet sometimes there is no such correlation. Symptoms may be severe while the cause and pathological results are comparatively insignificant, and vice versa.

The chemical condition of the blood, the permeability of the blood vessels and of the mucous membrane, may be such that bleeding results very readily when the bowel is squeezed and becomes congested. One may find a strangulated loop of intestine full of clotted blood. I well remember a case of intussusception in which by far the most prominent symptom was severe haemorrhage from the bowel. The gripping pains were so slight that the medical attendant could hardly decide upon invoking surgical aid, yet a well developed ileo-colic intussusception was found. Although the haemorrhage had been profuse, the intussusception was reduced with extraordinary ease. Comparable conditions are seen when, from the straining and consequent congestion caused by vomiting or difficult defaecation, bleeding either from the nose or from the bowel occurs—sometimes quite profusely.

Lulls in the storm of obstructive symptoms occasionally occur and may be so decided that the patient and his friends may refuse to allow operation, however clear the diagnosis may be.

There is usually so great urgency in most of these cases that it is dangerous to wait for the results of special laboratory examinations, even though the urine and blood may give most valuable indications for both operative and post-operative treatment. The questions, whether blood urea is present in large excess, and whether chlorides have largely disappeared from the blood, ought not to influence the operative procedure. It is possible to decide, from the urgency of the vomiting and appearance of the vomitus, and, at operation, from the state of the bowel proximal to the obstruction, whether these constituents are likely to be found in abnormal quantities in the blood, and much more certainly it can be decided whether that part of the bowel is likely to recover its tone or not before the patient dies. Over-distended but still contracting bowel is likely to recover; over-distended but inert bowel, incapable of response to external stimulus such as pinching, will almost certainly not recover if left to itself. Before the latter condition has developed, gangrene of the strangulated part will probably have occurred. The only chance for the patient in such cases is either to excise the gangrenous part and flaccid bowel above, as Arthur Barker advised, or to make a smaller excision and then perform enterostomy, single or multiple, in the bowel higher up. The procedure I want to emphasize especially is enterostomy, through which opening the poisonous contents of the upper bowel are drained away.

TREATMENT.

Sir Berkeley Moynihan, in his book *Abdominal Surgery*, says that of any twenty consecutive cases of average intensity in fifteen operation has been too long deferred.

Again, "To operate early in a case of intestinal obstruction is an experience that few surgeons often enjoy." The mortality after operation in these cases is far higher than it might be. There are "few surgeons who, in a series of twenty or more cases, can show a lower mortality than 50 per cent. Anything over a 10 per cent. mortality is the mortality of delay." Serious consideration of the symptoms which I have detailed ought to establish the diagnosis within a few hours.

The use of morphine or other sedative is frequently a bar to early diagnosis, because it is often used without good judgement, though it is often necessary to give morphine when the patient is seen for the first time, on account of the excruciating pain and impending shock. But, as Moynihan says, avoid needless repetition of morphine; this does the harm. If a small dose of morphine fails to relieve the recurrent colicky pains, apprehension should arise that early operation may be required. There is no absolute necessity to give morphine, and still less is there justification to repeat the doses until diagnosis is established. When that is done, then, as Moynihan remarks, "morphine is a remedy and not merely a refuge."

There are certain necessary procedures which are accessory to operation and which become increasingly important as the condition develops in intensity.

First, it is necessary, if the patient has been vomiting at all, to wash out the stomach thoroughly, especially before giving a general anaesthetic, otherwise he runs the risk of being drowned by his own vomit or of inhaling vomited material and bringing on septic pneumonia. Spinal or even local anaesthesia does not entirely obviate these risks.

Many years ago I was called to a remote village, to find the patient, a large, heavy man, in a very pitiable and distressed state owing to neglected chronic obstruction which had suddenly become acute. I thought that while he would not stand a general anaesthetic, he would be able to control his vomiting, so that it would not choke him, if we gave him a spinal anaesthetic. I wanted to avoid for him the further distress of having his stomach washed out, since he was very nervous and had a bad heart. I neglected to tell him that his legs might become paralysed temporarily. When the spinal anaesthetic took effect, he suddenly realized that he could not move his legs. He took fright, became greatly excited, fainted, vomited profusely, and was really drowned by his own vomit, although his enfeebled heart failed also.

In some cases, when vomiting persisted and was tending to become overflow in character, I have left the stomach tube *in situ* during the operation, or if the stomach and upper intestine were found distended when the abdomen was opened, I have passed it again.

The next indication is to restore as far as possible the depleted body fluids which have been extracted from the tissues everywhere. The increasing diminution of the chlorides of the blood has apparently a great deal to do with the recuperative powers of the patient; the loss of these chlorides must be made up, but no excess be given. Therefore give, according to the size of the patient, the state of his heart, and the effect on his pulse and general condition, intravenous, subcutaneous, and rectal infusions of saline solution. His tissues will tend to take it up rapidly, so encourage absorption by making the solution isotonic. The saline may have to be given before or during the operation, as well as later on. In the most severe cases it may be necessary to give hypertonic saline, as Ross has recommended.

I think it is better not to give active aperients after operation until the bowel has had time to recover its tone. Such a stimulus may, I believe, have the opposite effect to what is desired and cause the debilitated bowel to cease to function. I prefer to give enemata, to empty the bowel from below so that less strain is required to push on what may come along from above, and at the same time I try to coax back vitality and vigour and co-ordinate movements in the injured part by such simple remedies as belladonna by the mouth, or, if sickness threatens, atropine or eserine hypodermically in small doses. The first enema is usually given on the day succeeding that of operation. When evidence of activity becomes manifest in spontaneous passage of flatus or faeces or even by desire to go to stool, then I give, for example, small repeated doses of cascara and belladonna, or even a dose of castor oil and

laudanum. As soon as the patient feels desire for it, I give him ordinary food so as to stimulate normal peristalsis and prevent noxious adhesions.

The part of the bowel which has been strangled tends to form adhesions. On more than one occasion I have had to open the abdomen again on account of fresh obstruction from this cause. Sometimes, if the bowel has been severely congested and dilated, although it may recover its colour quickly at operation, it may require several weeks before it works properly; in these cases the pulse remains abnormally rapid. Both feeding and the administration of medicinal intestinal and cardiac stimulants may have to be carefully regulated.

In discussing actual operative measures I wish first to lay stress upon the situation and manner of the incision. I almost invariably make a long, at least six inches, para-central incision on the right side, a good inch from the mid-line, and displace the rectus from the mid-line outwards. I like to infiltrate the area of incision with 1/2 per cent. novocain solution (*plus* adrenaline) so that I may, if necessary, carry the making of the incision under local anaesthesia alone at least as far as the peritoneum and at the end of the operation be able to close the abdomen without more general anaesthetic than may have been required for intra-abdominal manipulation. This reduces the length and strain of general anaesthesia very greatly, and it is a much more simple business than making, for example, paravertebral injections. A short gas and oxygen anaesthesia will not harm the patient unless his condition is very desperate. I think that the long incision, by permitting easier examination and handling, more than makes up for the extra time expended on it. When sutured in layers post-operative hernia is avoided more by this than by any other method of making a long incision.

Having opened the abdomen, the hand is inserted into the lowest part of the pelvis and pulls up any collapsed coils there. The collapsed bowel is then followed upwards until the point of obstruction is reached. If no collapsed bowel is found in the pelvis the caecum is examined. If the caecum is collapsed the obstruction must be above it; if distended, then the obstruction is below it, so the sigmoid is next inspected; if this is collapsed the obstruction lies between the caecum and sigmoid. When there is much difficulty in arriving at a diagnosis of the site of obstruction I allow the distended coils of bowel to come out of the abdomen, but I cover them carefully with warm dry towels and gauze strips to prevent undue cooling. There is really no necessity for more elaborate apparatus. I prefer dry material to moist material; the latter, by its evaporation, fosters cooling of the bowel. If warm lotion is continuously poured over it in order to obviate cooling there results a sloppy mess besides the introduction of extra risk. I do not think that evil results follow the use of dry material so much as when moistened packs are used.

After finding the obstruction further proceedings range from the mere snipping of a band to the difficult and tedious separation of adhesions, short-circuiting, or the actual excision of a gangrenous loop followed by entero-anastomosis. In the last case I prefer to make end-to-end anastomosis whenever possible. If the patient is very bad it may be compulsory, after dealing with the cause of obstruction, simply to bring the gangrenous loop well out of the abdomen, fix the viable proximal and distal parts of the bowel to the edges of the wound, and open the loop. One or more enterostomies should be made above the affected loop through separate small wounds. Enterostomies, reserved for the worst cases, procure the advantage of draining different parts of the bowel and at the same time permit the introduction of saline solution. Every surgeon of experience knows how inert the bowel may become, and though drainage effectively empties one loop, adjacent loops may remain distended. The bowel kinks very readily in such circumstances. The saline solution must be introduced at very low pressure—over-distension prevents recovery of tone, besides tending to cause kinking. I believe that the performance of enterostomy gives better results than emptying the proximal bowel during operation. The latter procedure demands too much handling of the bowel and tends to increase shock; the same remark applies, of course, to wide excision of the bowel. In the

most desperate cases it is permissible only to make enterostomy, under local anaesthesia. In such cases I think that one opening should be made in the bowel by incision above the umbilicus and another well below it. The upper opening will combat the proteose toxæmia, the lower will combat the septic toxæmia.

It is sometimes difficult to decide when to perform enterostomy. I believe that if strong colicky pains have persisted right up to the time of operation and if, at the actual operation, the bowel responds to local stimulation, such as pinching, it will likely recover without enterostomy. If, however, the colicky pains have become feebler, if general distension of the abdomen is present, and if, at operation, the coils are found to be in any degree flaccid and floppy and do not respond definitely to local stimulus, enterostomy is compulsory. In cases of doubt, rather make an enterostomy than not. Vomiting will not get rid of the toxin, nor will a parietic bowel pass it downwards. As Wilkie of Edinburgh pointed out, paresis of the bowel is an important contributory factor to toxic absorption. The bowel will not recover unless the toxic contents are removed. If the obstruction has been in the large intestine I like to make a caecostomy as well as an enterostomy. In cases of this degree of severity I should also administer 20 per cent. hypertonic saline intravenously.

Sir William Taylor of Dublin has given indications as to the likelihood of having to perform enterostomy in cases of obstruction. Roughly speaking, according to him, the necessity corresponds to the stage of the illness, and he defines three stages. In the early stage it is only necessary to treat the cause. In the second stage, when distension of the bowel has occurred, the obstruction should be relieved and jejunostomy performed. Drainage may be required for only twenty-four to forty-eight hours. In the third stage the condition of the patient is so bad that it will not permit exploration, therefore jejunostomy only should be done, and later on, if the patient recovers, the obstruction may be dealt with. In my opinion that "if" is a big one; I prefer to ascertain what the pathological condition inside is. There is practically no hope for a patient who has to deal with, for example, a gangrenous loop of bowel in his interior. Enterostomy alone will not save him. Before giving up all thought of exploration, I like to see the effect of stimulation by drugs, intravenous infusions of saline solution, and blood transfusion.

In making an enterostomy, I think a Pezzier's catheter is a suitable tube to use, and of a medium size, but an ordinary large catheter serves the purpose equally well and is easier to remove. The latter has to be fixed by suture. Only exceptionally are there solid particles in the small intestine, so that the contents drain through comparatively small tubes.

I prefer to make the enterostomy, when the condition of the patient permits, after drawing the selected loop of bowel through a hole made in the omentum. When practicable, the tube is inserted in the bowel in a manner similar to the Witzel method employed in gastro-enterostomy, but in many cases one has to dispense with this refinement and simply insert the tube through a straight puncture, surrounding it by a purse-string suture. One of the fine chromic catgut sutures is left long and is used to moor the bowel to the abdominal wall. The loop of bowel is pushed back through the hole in the omentum, which is then made to lie snugly around the tube. If the enterostomy has been made from the exploratory incision, I draw the tube, before inserting it into the bowel, through a new small incision in the abdominal wall; this is usually most suitably made somewhere to the left of and slightly above the umbilicus. By this precaution contamination of the main wound is avoided. For suturing such easily torn bowel as is met with in this condition the so-called "atraumatic needle" advertised by the London Hospital appeals to me. The traumatizing "catch" of the thread at the eye of the needle experienced when ordinary needles are used may tear and cause a leak.

After multiple enterostomy I like to remove one tube at a time, the upper before the lower. The upper part of the small intestine seems to recover tone more quickly than the lower; the character of the toxin producing the paresis of the bowel may account for this as well as the fact that

the paresis has usually been present in the lower bowel for a longer time. I have an impression that the higher the enterostomy the longer it takes to heal. The tubes may be removed and the stomata allowed to close as soon as the patient is established in convalescence—that is, really, when vomiting has stopped and the bowels have "moved" satisfactorily. Evacuation of toxic contents is one important factor, but restoration of peristalsis is also essential to recovery.

A simple application for preventing excoriation of the skin round an enterostomy is ethereal rubber cement solution (1 in 4) applied several times a day. The ether evaporates and leaves a thin film of rubber over the skin.

It is sometimes almost impossible to tell, from the appearance of the bowel, whether the colour and the swelling are due to intense congestion and subperitoneal or interstitial hæmorrhage alone or whether gangrene has actually occurred. The presence of the hæmorrhagic effusion constitutes the difficulty. The purple colour of mere congestion soon gives way to a more normal appearance when strangulation is relieved, but effused dark blood does not dissipate and there may be no change of colour or only in patches. Of course, the discoloured, flaccid, inert, and often brittle state of the really gangrenous bowel is easy to distinguish—but there are all gradations. It must be remembered also that interstitial extravasation of blood has probably taken place before gangrene has occurred. I have never found the following test to fail, though I have used it for many years. I incise, with a sharp knife, the peritoneum and very superficial fibres of the muscular coat of the suspected part of the intestine. If the circulation is intact, definite bleeding, although possibly in small amount, will occur. It is found, then, that each time a clean piece of gauze is pressed lightly on the incised part it will be stained with fresh blood. Viability of the bowel is still more certain if the blood can be seen oozing steadily from the cut. If gangrene has occurred, the first or second piece of gauze may be definitely stained, but not more. There is practically never any doubt in actual experience. If the gut is viable the small incision is closed by a mattress suture of fine linen or catgut.

REFERENCES.

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PYELITIS FROM INFECTION WITH MORGAN'S NO. I BACILLUS.

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As I have been unable to find any reference to urinary infection with Morgan's No. I bacillus, I think that the following two cases are worth recording. The association of this bacillus with infantile diarrhoea, and as a concomitant organism in bacillary dysentery due to the bacillus of Shiga and Flexner, is generally recognized. Recently I have found this organism in the faeces of many patients suffering with symptoms of bacillary dysentery, when repeated attempts have failed to isolate any other dysenteric organism. While believing it to be pathogenic, I had no positive proof of this until I isolated it from two cases of very definite pyelitis.

CASE I.

Private L., aged 24, a British soldier at Khartoum, stated that while in India (Agra) he had the first symptoms of his present condition. In February, 1923, he had a sudden sharp pain in the left side, which remained localized, was worse when he was in bed, was not propagated, and had no relation to micturition. For several days he had frequency of micturition and noticed that his urine was thick. The acute symptoms passed off in a fortnight on the exhibition of urotropine. Two months later (in April, 1923) he had a similar attack; pus was found in the urine, but no blood or casts were seen.

No further symptoms occurred until November, 1925, when he had to be relieved on guard duty because of a sudden onset of acute pain in the left lumbar region. The symptoms again passed off in ten days, but a further attack occurred only one month after. A radiogram then taken showed no detectable pathological condition.

The present attack, when the patient first came under my notice, was on March 15th, 1926. While on police duty he was