POST-GRADUATE MEDICAL JOURNAL

DIAGNOSIS OF ACUTE INTESTINAL OBSTRUCTION

by JOHN. T. MORRISON, F.R.C.S.

(Honorary Surgeon, Royal Liverpool United Hospital (Royal Southern Hospital Branch), etc.;
Lecturer in Surgery, University of Liverpool)

The lay public like to think that there are many occasions when delay of even a few minutes in the doctor's arrival will make all the difference between life and death. We know that this dramatisation has no basis in fact apart from the real emergencies of haemorrhage, suffocation, and perhaps open wounds of the thorax. There are, however, a number of conditions in which delay in diagnosis and treatment, for more than a very few hours, may convert a relatively safe and straightforward operation into one fraught with deadly peril, to which the patient succumbs or recovers only on the brink of disaster. Of these, acute intestinal obstruction is a striking example.

It should not be necessary at this time of day to give warning that the symptoms and signs described in the older textbooks, faecal vomiting, abdominal distension, and cardio-vascular collapse, are not symptoms of intestinal obstruction, but as has been well said, symptoms of impending dissolution. Diagnosis at that stage is easy, but is not likely to lead to treatment of any value. Diagnosis at the inception of the condition is often difficult, but so important that I make no apology for dwelling on it. First of all, however, I think it would be wise for us to reflect on the causation of the signs and symptoms for which we look. It will be helpful if we recognise from the start that it is relatively seldom that we see a case of pure intestinal obstruction. Nearly always there are interwoven with the symptoms and signs of intestinal obstruction the symptoms and signs of intestinal strangulation. In our minds we should keep this clear, remembering, too, that in the later stages peritoneal infection is added still further to complicate the picture.

When we are faced with a patient in whom these problems arise, inquiry into the previous history will often give valuable pointers. Of course patients frequently misinterpret symptoms, and mild obstructive symptoms which often precede—it may be for years—the actual onset of acute obstruction are described as “indigestion” or merely “wind.” There may be clearly defined attacks subsiding spontaneously, which correspond to periods when acute obstruction has been avoided only by the thinnest margin. This is particularly true of many cases of external hernia, but is also found in cases of internal hernia and volvulus. It

is rare in acute intussusception, while in gall-stone ileus previous sub-acute attacks are specially characteristic, because the stone acts as a ball valve, releasing itself when a certain degree of distension is reached. What is curiously absent in these latter cases is a history of gall-stone disease. There may be nothing to suggest it. In cases of tuberculous stricture, too, a history of intractable enteritis might be expected. It is often completely absent. Where obstruction is due to adhesions, it is fairly common to trace an acute abdominal illness which was probably the original peritonitis, but it should be recalled that some of these adhesions are due to old tuberculous disease which may well have developed, run its course and been overcome, without ever causing symptoms sufficient to justify a diagnosis. Even when an external hernia is an obvious cause, patients sometimes fail to mention it unless specific inquiry is made.

In thinking of symptoms let us take first an example of pure obstruction such as we see in gall-stone ileus, tuberculous stricture, or non-strangulating adhesions. The symptoms fall naturally into two groups, those due directly to the physical block, and those due to associated phenomena. The barrier to the onward flow of the bowel contents leads to violent peristalsis in an effort to overcome it, and this, as Poulton by careful experiment has shown, gives rise to pain, not in the area of spasm, but in the overstretched wall of the distended loop immediately beyond it. It is a characteristic griping pain which may be of such intensity as to give rise to syncope, or at least to a feeling of faintness, and it is often followed by vomiting. As with all pain of small intestine origin—and I am referring throughout to small intestine only—it is felt in the mid-abdominal region. It dies away between the attacks, but not infrequently there remains a vague sense of discomfort, which may be attributed to a minor degree of stretching in the loop or loops involved.

This, then, is the first cardinal symptom of acute intestinal obstruction—*persistent griping pain*. It is not of course diagnostic, for the most violent and fairly persistent griping pain may occur in simple constipation. In this, however, an enema will speedily remove all doubts, not because an action of the bowel results, for in obstruction the distal bowel may empty spontaneously or in response to an enema, but because the hitherto persistent griping pain ceases.

The second cardinal symptom is *persistent vomiting*. I have already mentioned how the first attacks of violent pain may be followed by faintness, nausea, or vomiting. This is not the specific vomiting of intestinal obstruction, but the vomiting which may follow any severe pain. “I felt quite

sick with the pain," we often hear. It is presumably of central nervous origin. In acute intestinal obstruction the characteristic vomiting soon makes its appearance, and, unlike the vomiting associated with early inflammatory disease in the abdomen, tends to increase as time goes on. We all know how the character of the vomitus changes from gastric to duodenal and then to jejunal and finally ileal contents, alkaline in reaction. But why is vomiting so typical of obstruction? It goes on long after stomach and small bowel ought to have emptied themselves, proving that large quantities of fluid are excreted into these parts of the alimentary canal. At operation we can see that the intestine is full of fluid as well as gas, and the high rate of chloride loss from the blood indicates that the same is going on in the stomach. As long therefore as the muscle of the intestine remains active, this fluid in default of passing on by the normal channel is forced to flow back towards the stomach. It is not necessary to assume a definite reverse peristalsis, though that may occur. From the stomach it is evacuated because of its very bulk, not because of any specially irritating qualities, for the passage of a tube will always reveal that the stomach is full.

Thirdly, and again this is of great importance, the *pulse rate rises steadily*, not dramatically, but progressively. When the first shock of a sudden obstruction passes the pulse remains quiet and slow for a time, but within a matter of hours it begins to climb 4, 8, 10, 15 beats a minute. Perhaps the severe persistent colic is exhausting. Possibly there is absorption of toxins from the distended loop. Bacterial toxins some say, and, indeed, many acute clinical observers attribute the deaths that sometimes result in spite of the relief of obstruction, to absorption of these poisons from the distal gut rapidly flooded with them after operation. Others declare that the poison is a proteose formed in the mucosa above the obstructed point. Experimental evidence, however, is not very conclusive for either of these theories.

We are probably on surer ground when we take into account nervous influences on the pulse rate in these early stages. Much experimental work has demonstrated that distension, not blockage of bowel causes the phenomena of acute obstruction, and that these effects are delayed in their onset and life prolonged by denervation of the distended loop. From the clinical angle we note that the typical picture of acute obstruction may be seen in cases of Richter's hernia when in fact no complete obstruction is present. The nervous stimuli arising in the nipped and distended knuckle are enough apparently to set the whole train in motion, including a reflex inhibition of peristalsis distal to the lesion.

Finally, in this connection we must just glance

at certain chemical factors. We have to admit that death may result without gross changes in the blood chemistry, but we cannot rule out the possibility that even in the early stages such changes are beginning and may exercise some effect on the circulation. If vomiting is excessive there is not only loss of fluid, and consequent physical difficulty in maintaining an adequate circulation, but there is also loss of chlorides, and probably before very long of sodium ions also. These electrolytes are apparently essential for the retention of water in the tissues, and their loss maintains and aggravates dehydration. Perhaps we should also consider the possibility that loss of sodium may leave the blood calcium in relative imbalance, and so increase the irritability of the heart muscle. In all probability, however, we should not blame any one but rather a combination of several factors.

Last amongst classical symptoms comes *absolute constipation*. Apart from the emptying of the lower bowel already referred to, there is passage of neither faeces nor flatus. A second enema in cases of doubt is justifiable and indeed commendable, but other methods to stimulate further evacuation waste valuable time and may be dangerous.

I have dealt in considerable detail with the symptoms to be found in cases of acute obstruction. I now pass to physical examination. The general appearance and the state of the tongue will of course be noted, and the temperature respiration and pulse rate recorded. The abdomen will next be examined.

In cases of pure obstruction there may be no visible or palpable evidence of the type of lesion present, though from the nature of the underlying pathology, acute is quite often superimposed on pre-existing chronic obstruction, and therefore it is not unlikely that from the start there may be some distension and possibly visible peristalsis. There may be slight tenderness, and perhaps some protective muscle guarding, but often enough there is no definite change in muscle tone. A vague mass will suggest that at least one loop is already distending, but I would not like to have it thought that all or any of these signs need be present. In speaking of the examination of the abdomen too much stress cannot be laid upon the importance of specifically examining the hernial orifices. If a hernia is present it is not necessarily the cause of the symptoms, though it must be regarded as such until the contrary is proved. Note must be taken of irreducibility, loss of expansile impulse on coughing, tenseness and tenderness of the swelling. In assessing irreducibility taxis must not be unduly vigorous. One has been impressed many times, when the neck of a strangulated hernia has been exposed at operation with the utter impossibility of effecting anything

by taxis. Once or twice I have seen spontaneous reduction of a genuine strangulated hernia during transference by ambulance to hospital; but in the great majority of cases, after a brief period of firm but careful manipulation, the attempt at reduction should be abandoned for fear of further damage to an already damaged bowel. In small children hope of reduction should never be given up until the patient has been allowed to settle down and stop crying, the foot of the cot having been raised so high that the child is inclined head down at an angle of 25 to 30 degrees. This is often successful. Complete irreducibility of a hernia previously reducible should be regarded as evidence of strangulation.

In cases of intra-abdominal strangulation palpation may reveal a somewhat indefinite mass, rounded and a little tender. This may be either the strangulated loops of a volvulus or internal hernia or the distended loops immediately proximal to an obstruction. The classical example of a palpable mass in acute intestinal obstruction is of course the "tumour" felt in cases of acute intussusception. The clinical picture in this disease is now so well known that it would seem unnecessary to devote further time to it. It may, however, be worth while to mention that blood is not always present in the stools, though usually it is, nor is the "tumour" always palpable. It is best felt when the child is asleep between attacks of pain, and if the examiner is patient and his hand is warm, useful information can be obtained in this way. Rectal examination, coupled with a hand on the abdomen, is often invaluable. Sometimes an anaesthetic is necessary, particularly where the "tumour" is high up under the liver.

I do not want to dwell on what is, meanwhile, happening with the obstructed loop. Its bearing on early diagnosis should not be great. It is only in neglected cases that it becomes important to remember that these changes will lead to intense congestion, and then infection of the intestinal wall, and this, aggravated by ischaemia from distension or thrombosis, may well end in gangrene and perforation. Even before this stage, however, infection may reach the peritoneal cavity. In either case, of course, the signs and symptoms of peritonitis will be superimposed on those of obstruction.

If strangulation is present as well as obstruction—and that is in the great majority of cases*—a great

stimulus is given to these infective changes. The damage to the arterial supply may be so great that definite gangrene may result quite early, and hence the peritoneal inflammatory signs in the shape of local pain, tenderness, and well-defined muscular resistance are found at a much earlier date. We do not, of course, get rigidity in the proper sense of that much-abused word, unless actual perforation of a gangrenous loop takes place—a rare happening and always late. If it occurs in an external hernial sac the general condition does not deteriorate to anything like the same extent as when the general peritoneal cavity is involved. Indeed, I have seen such a patient survive as long as ten days, before effective surgical treatment was instituted. The pain and tenderness referred to is much more acute and more localised than the vague sensations complained of in pure obstructions. They are due, as of course the pain in peritoneal infections always is due, to stimulation of the parietal layer of the membrane by inflammatory products. If the general peritoneal cavity is involved the condition ultimately is indistinguishable from a general peritonitis from any other cause. And here, perhaps, I might point out that one of the basic factors responsible for the symptomatology in general peritonitis is in turn an intestinal obstruction, brought about by the paralysis that ensues when coils of intestine are immersed in toxic fluid. It is interesting to note that this paralysis is inhibited by spinal anaesthesia. It would appear therefore that this is not a local toxic action, but a reflex paralysis resulting from afferent stimuli, carried by the lumbar sympathetic rami. We have here a mechanism which reinforces and exaggerates the symptoms and signs of obstruction, as well as giving rise to others specifically indicative of toxæmia.

Another though less dangerous feature of strangulation is that, as always, it is the venous circulation which is first affected. Hence an early result is congestion, swelling and turgidity of the affected part, and then an effusion into the peritoneal cavity. Sometimes it is clear, but often it is blood-stained, and it may be quite dark red in colour. At a later stage this fluid may become turbid and even offensive through infection. The clinical demonstration of the fluid is not easy, and so far as diagnosis is concerned is not a matter of any great importance.

Auscultation is not of great value either at the stage of obstruction when it is most needed and diagnosis is difficult, i.e. at the beginning, but in late cases and in the obstruction due to paralysis of bowel in acute peritonitis it is a useful adjunct to clinical investigation, revealing the absence of all sounds of intestinal movement.

X-rays do not play much part in the diagnosis of acute obstruction, but a "straight" X-ray may

* In the last ten years at the Royal Southern Hospital and in my private practice the proportions have been:—

External herniae—50 per cent.

Volvulus—8.4 per cent.

Bands and adhesions—16.6 per cent.

Other causes—25 per cent.

This latter figure is swollen by the fact that it includes intussusception, of which at one time a good deal came my way.

be helpful in demonstrating a distended loop or loops of bowel, with perhaps fluid levels in them. An X-ray may also show up a large gall-stone, and if it fails to do so this may be itself an important piece of negative evidence.

In conclusion a few words may be said about differential diagnosis. I do not mean differential diagnosis of the exact nature of the obstructing lesion. That may well be impossible. But differential diagnosis between obstructive and non-obstructive conditions is sometimes difficult. Mistakes are made, and it may be that at operation nothing abnormal is found. Occasionally one finds areas of intestinal spasm for which no adequate cause can be found. At other times some condition is present which gives rise to reflex symptoms suggesting an atypical obstruction. Such, for instance, is torsion of a piece of omentum or of the pedicle of a small ovarian cyst or fibroid. I think the surgeon is right to consider obstruction in such cases, and if in real doubt he must explore.

Strangulated omental herniae may give rise to much anxiety, and there may be definite reflex interference with bowel function. It is often impossible to be sure that there is no bowel present in the sac, and all such cases ought to be operated on. In the same way strangulation of an ovary in a hernia—the ovary in little girls is not a very unusual content of an inguinal hernial sac—may be very confusing. If its function is to be conserved it should in any case call for early operation even if intestinal obstruction can be excluded. Strangulation of a testis impacted in the inguinal canal may also simulate intestinal obstruction.

I have been called to a case of hydrocele of the cord believed to be a strangulated hernia. It is true that these hydroceles do sometimes swell up very quickly, but the excellent general condition of the young patient, and the absence of abdominal symptoms should save us from all real anxiety. There is perhaps a little more to be said for those who mistake an inflamed lymph gland over the external ring for a strangulated hernia. Though not recorded in the textbooks of anatomy a gland undoubtedly occurs in this situation at least in a proportion of people. Adenitis is rather an acute process, the tenderness considerable, the temperature will probably be raised, and there may be some vomiting, but the local signs and the toxæmic symptoms are quite out of proportion to obstructive intestinal ones, and the gland can be felt distinct from the opening of the inguinal canal, so that hernia can be excluded.

The condition that gives most difficulty is perhaps an incarcerated umbilical hernia. It is irreducible of course, and constipation may be very complete. It is as a matter of fact a real case of obstruction, but as there is no strangulation and

the part of the bowel affected is usually low down in the alimentary canal, the symptoms are correspondingly free from urgency. The danger here is that a false sense of security is engendered and, operation delayed until considerable deterioration of the patient's condition has taken place.

Lastly, I would mention that in elderly people there is a tendency for symptoms suggesting obstruction to overshadow the inflammatory signs in cases of intra-peritoneal infections, particularly appendicitis. In these, and indeed in every case where we are faced with an abdominal emergency, the possibility of obstruction has always to be borne in mind. The clinical picture will often tell us clearly that obstruction is present, though it may not justify us in hazarding more than a guess at the exact pathology. That is enough. Where real doubts exist operation in experienced hands represents a very much less risk than any policy of *laissez faire*.

REFERENCES

- BARCROFT, *Lancet*, 1944, **i**, 489.
 POULTON, E. P. *Lancet* 1928, **ii**, 1223.
 HERRIN and MEEK, *Arch. of Int. Med.*, 1933, **i**, 152.
 WHIPPLE ET AL. *Bull. Johns Hopkins Hosp.*, 1912, **xxii**, 159.
 WHIPPLE ET AL., *J. Exp. Med.*, 1913, **xvii**, 286.
 BEST and TAYLOR, *Physiological Basis of Med. Practice*, 3rd Ed., 854.
 DRAGSTEDT and ELLIS, *Amer. J. of Physiol.*, 1930, **xcviii**, 407.
 HALLIBURTON and MCDOWALL, *Handbook of Physiol.*, 34th Ed.
 WILLIAMS, B. W., *B.J.S.*, **xiv**, 295.
 MOLESWORTH, H. W. L., *B.J.S.*, **xvii**, 346.
 SULLIVAN, *Arch. of Surg.*, **xxi**, 1930, p. 531.
 PROF. NEWTON, Dept. of Phys., Univ. of Liverpool, personal communications.

CAMPBELL and MARCOWITZ, *Amer. J. of Physiol.*, 1927, **lxxxv**, 101.
 I have pleasure in acknowledging the assistance I have received from Miss Doreen Watt and Mr. J. T. Marsden, Surgical Registrars at the Royal Southern Hospital.

THE DIAGNOSIS AND SURGERY OF CHRONIC INTESTINAL OBSTRUCTION

INCLUDING ACUTE ON CHRONIC OBSTRUCTION

By Professor W. GEMMILL, M.A., M.B.,
Ch.M., F.R.C.S.

(Hon. Surgeon, Queen Elizabeth Hospital, Birmingham; Professor of Surgery, Birmingham University.)

An obstruction to the passage of the contents of any tubular organ such as the intestine may arise (a) from pressure on the tube from without, (b) from a condition arising in the wall of the tube itself, or (c) from the nature of the contents.

The commoner causes in the case of the intestine are as follows:—

(a) Pressure from without by a cord-like adhesion between two coils of intestine, or between the