Current Practice

DISEASE OF THE DIGESTIVE SYSTEM

Intestinal Obstruction

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"Ay, but to die, and go we know not where;
T'o lie in cold obstruction, and to rot."
CLAUDIO in "Measure for Measure," Act II, Scene 2.

Matters of surgical technique are mainly of interest to surgeons. In the treatment of patients with intestinal obstruction diagnosis and management form more difficult and important problems confronting all doctors who engage with this condition. These are the two aspects on which attention is centred below. The diagnosis of intestinal obstruction is complex—not just a single opinion but a progressive series of assessments, starting with the decision that a patient is obstructing and proceeding to evaluate the mode, level, pattern, pathological cause, and effects of the obstruction. Management concerns the initial conservative treatment of the patient, and the question of whether and when to operate, and involves the general practitioner and junior hospital staff as well as the surgeon. The high mortality of this condition¹ is a challenge to all who treat it.

Mechanism

In order to appreciate fully the changes occurring in the obstructed bowel and their effect on the patient it is necessary to understand the mechanism of obstruction. Whatever the cause, blockage of the bowel at one point leads to distension of the immediately proximal loop; this distension in the first place is due to accumulation of gas. This has been confirmed by clinical studies in which patients have been given indwelling jejunostomy tubes after abdominal surgery and have shown a lower incidence of postoperative obstruction.² Wangenstein³ has shown that the greater proportion of this gas (68%) is swallowed air, which normally passes straight through the alimentary tract; smaller contributions to the volume of gas are made by the bacterial decomposition of protein, which is increased in the obstructed loop, and diffusion of gases, especially carbon dioxide, from the blood. The resulting increase of pressure in the lumen halts the absorption of water and cations, and at a certain critical level of tension fluid starts to pass in the opposite direction, from the blood stream into the lumen; the obstructed loop can secrete large amounts of water, sodium, and potassium.⁴ This mixture of fluid and air gives the typical appearance of a fluid level on a plain radiograph. This can be termed the *primary obstruction*. Other factors besides the distension arrest absorption and promote secretion, such as oedema and venous congestion of the bowel wall.

If the condition is unrelieved the distension is relayed proximally, and the above changes occur loop by loop in the direction of the mouth. Each proximal loop in turn becomes dilated, ceases to absorb, and starts to secrete fluid. Owing to the confines of the peritoneal cavity these loops of bowel become kinked at each end, and each forms a closed loop obstruction

as it becomes distended. At this stage relief of the *primary* obstruction alone will still leave the patient obstructed unless this *secondary* type of obstruction is attended to, as a vicious circle has been set up perpetuating the situation.

Intestinal Circulation

To evaluate the effects of this process of obstruction on the patient the concept of the intestinal circulation of fluid must be borne in mind. The gastrointestinal tract has two function-

Average Alimentary Secretions Over 24 Hours for a 70-kg. Adult

Saliva	..	..	1.0 l.
Gastric juice	..	..	3.0 "
Bile	..	..	1.0 "
Pancreatic juice	..	..	1.0 "
Intestinal	..	..	2.0 "
			8.0 l. total

ally different components; the proximal part (mouth, stomach and upper half of small intestine) is predominantly *secretory*, and large amounts of fluid normally pass from the *capillaries* in the wall of the tract and from the related exocrine *glands* into the lumen. If these amounts are tabulated it can be seen

FIG. 1.—Annular carcinoma of sigmoid causing closed-loop colonic obstruction.

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that a large proportion of the body water is secreted into the lumen of the proximal bowel each day in the healthy adult (see Table). As the average water content of a 70 kg. man is 42 litres this alimentary secretion represents almost 20% of total body water.

The distal part of the bowel (lower half of the small intestine and colon) has a mainly absorptive function and passes back into the blood stream from the lumen a volume of fluid equal to that secreted proximally. This circulation of one-fifth of the body fluid each day in the intestine, secreted proximally and absorbed distally, provides a vehicle for the digestion and assimilation of the products of nutrition. Obstruction of the intestines arrests this circulation, leads to rapid fluid depletion, and strikes at the basis of nutrition.

Diagnosis

The first stage in diagnosis is to establish that the patient is obstructing, and evidence of this is provided by careful history-taking and physical examination. Usually the patient can recognize the condition by a tetrad of classical symptoms—abdominal pain, vomiting, constipation, and abdominal distension.

The *pain* experienced may be of two types—central abdominal colic with spasms lasting a few minutes, indicating small intestinal efforts to overcome the blockage, or persisting pain, which may be in one area of the abdomen, indicating that a loop of bowel is persistently distended. Often pain starts as colic, then becomes persistent, and finally may reach a crescendo indicating perforation or impending perforation of the bowel. For example, an annular carcinoma of the sigmoid colon not infrequently leads to over-distension and rupture of the caecum, and the pain pattern—initially central and colicky and finally severe, persistent, and in the right iliac fossa—may suggest acute appendicitis.

Vomiting is obstructive in nature, and of large volume, showing a progression as the condition continues. Firstly, the gastric contents are returned and recent food is recognizable; this is followed by turbid biliary fluid indicating the contents of the upper part of the small intestine; finally the so-called “faeculent” vomit ensues—dark brown, evil-smelling fluid

FIG. 2.—Erect film showing high intestinal obstruction due to carcinoma of jejunum.

consisting of ileal content mixed with blood. This is not actually faeces, which are vomited only in gastric-colic fistula. This third stage of vomiting in the adult marks a period of severe danger for the patient, as it is always associated with considerable dehydration, toxæmia, and often anaemia. In children, where pathological change marches more quickly, the second stage of biliary vomiting is equally serious.

Progressive *distension* of the abdomen is usually noticed by the patient as well as the doctor, and is a reminder of the large amount of fluid that may be present in the lumen of the bowel and has been lost from the tissues. *Constipation* is a misleading feature, since the condition should be obvious some time before absolute constipation supervenes; the bowels may well be opened once or twice after obstruction has become complete, and an initial enema may give a positive result.

Examination

Examination should first be directed to the general condition of the patient. Skin ridging, sunken, dull eyes, a dry furred tongue, and fetor are the hallmarks of severe loss of fluid from the extravascular space; tachycardia, hypotension, and a cold, whitish-blue, clammy skin indicate an accompanying reduction of plasma volume leading to peripheral vascular failure.

The eyes, ears, and hands of the doctor should then be turned to the abdomen. Visible peristalsis can often be seen centrally associated with the ladder pattern of small intestinal distension, and before long this gives way to marked central distension. In the case of colonic obstruction the different pattern of peripheral distension in the flanks and upper abdomen can usually be recognized initially before the development of central small-intestinal distension. The stethoscope will reveal the presence of typically obstructive sounds reverberating throughout the abdomen, tinkling, high-pitched, and consonating. Disappearance of these marks the onset of peritonitis. In the early stages simple intestinal obstruction gives rise to a soft abdomen; as it progresses tenderness may appear owing to an undue degree of stretching of the intestinal wall; if muscle guarding is to be found this indicates some serious complication such as strangulation, impending perforation, or frank peritonitis. Digital examination of the rectum is essential to establish whether faeces are present or if there is the characteristic ballooning of the rectum that occurs below an obstruction, or a pelvic abscess, or malignant mass. An obstructed distended abdomen will yield a deep tympany on percussion; shifting dullness may also be elicited owing to large amounts of fluid and gas in the severely distended colon or ileum, but can be distinguished from ascites by the differing pattern of dullness and tympany. Finally, the groins must be carefully inspected for the presence of a strangulated hernia, or in the elderly for an absent or diminished femoral pulse that might indicate intestinal ischaemia or a leaking aneurysm.

Plain x-rays of the abdomen yield helpful information about the obstructed abdomen and can be as useful as contrast studies, which are usually contraindicated in the acute stage. The supine film shows the gas pattern of the bowel and will often indicate the site of obstruction. An erect film will reveal fluid levels in the colon or small intestine. The patient should be

FIG. 3.—In low intestinal obstruction fluid is secreted into the lumen of the obstructed loop, but then passes proximally and is absorbed by functioning intestine.

x-rayed after passing through the admission department on the way to the ward.

Modes of Obstruction

Consideration so far has been of simple occlusion of the bowel by a pathological lesion acting at one point, but other modes of obstruction include strangulation, closed loop obstruction, and functional obstruction.

Strangulation implies that the blood supply of the obstructed loop is impaired, and this may be brought about in two ways. The affected portion of bowel may be caught in a tight unexpandable space, and the entering and leaving loops may be constricted. This happens with internal or external herniae, volvulus, or

adhesions. The veins first become compressed, leading to venous engorgement, and when the tension in the bowel wall or mesentery reaches a certain intensity the arteries also become occluded. This results in two further severe complications in addition to the effects of the obstruction—blood loss into the lumen of the bowel, which may be considerable, and perforation of the bowel wall from the resulting ischaemia, leading to a mortal form of peritonitis. The presence of severe tenderness and guarding in a distended abdomen suggests this train of events and is an urgent indication for laparotomy. The second cause of strangulation is superior mesenteric arterial occlusion, due either to thrombotic degenerative arterial disease, when there may have been a previous history of mesenteric angina with upper abdominal pain after meals, diarrhoea, and weight loss, or to an embolism in a patient with atrial fibrillation. Laparotomy usually reveals long lengths of black small intestine, and despite resection mortality approaches 100%. Milder degrees sometimes respond to intravenous dextrans. A few successful attempts at embolectomy and bypass grafting have been reported.

Closed loop obstruction (Fig. 1) is most often exemplified by an annular carcinoma of the colon, most commonly the

FIG. 4

FIG. 5

FIGS. 4 and 5.—Low intestinal obstruction. Note the many small-intestinal levels, indicating about 3–4 litres of fluid in the lumen. This is due to appendix abscess as can be seen by the blind area in R.I.F. in Fig. 5.

FIG. 6.—Carcinoma of splenic flexure of colon, with closed-loop obstruction, and proximal small-intestinal obstruction.

sigmoid. In normal health the ileo-caecal valve is effective in preventing reflux of colonic contents into the small intestine. When an annular lesion reaches the stage of complete obstruction the colon between it and the ileo-caecal valve is converted into a closed loop, which proceeds to distend with fluid faeces and may reach a diameter of 5 or 6 inches. Unless relieved surgically in time the end point is perforation, often of the anterior wall of the caecum, and resulting faecal peritonitis—another condition that is highly lethal. Acute appendicitis is of course another example of closed loop obstruction.

Functional obstruction of the bowel results from inactivity of the bowel wall, usually associated with dilatation, in the absence of any mechanical blockage. This most commonly takes the form of paralytic ileus due to peritonitis from perforation or some type of suppurative lesion, when obstruction and its effects complicate the severe infection due to the underlying lesion. After an operation for generalized peritonitis paralytic obstruction is always a possibility and postoperative jejunostomy has

been employed to lower its incidence.⁵ The other causes of pseudo-obstruction are discussed below.

It is important to recognize which mode of obstruction presents, as this regulates the management of the patient and influences prognosis.

Levels

The effects of intestinal obstruction on the patient depend on the level of the obstruction. It is important to distinguish three levels—high and low small-intestinal, and colonic.

Obstruction high in the small intestine is the most serious. No fluids taken by mouth will reach absorptive bowel, and in addition the saliva, gastric juice, bile, and pancreatic juice will be vomited up—a total loss of 6 litres of fluid a day in the adult when the obstruction becomes complete—leading to severe and sudden dehydration. The diagnosis is often difficult and delayed, as no abdominal distension occurs; the large volume of vomit is the only clue. This is an uncommon occurrence, but it is found in duodenal atresia and annular pancreas in infants, and in carcinoma of the third and fourth parts of duodenum and upper jejunum (Fig. 2), duodenal ileus, and infiltrating carcinoma of the pancreas in the adult.

Obstruction in the lower ileum comes on more slowly and manifests all the typical features mentioned above. The loop of bowel proximal to the lesion dilates, loses its absorptive function, and secretes fluid into the lumen. However, much of this fluid may pass proximally into normal bowel initially

and become absorbed (Fig. 3). Ultimately the proximal loops may become affected in turn, but the lower the level of obstruction in the ileum the slower the effects will be to develop and the longer the proximal small intestine will continue to absorb (Figs. 4 and 5).

Colonic obstruction has already been discussed. Once the closed loop of the colon has started to distend it will act as an obstructing lesion and lead to a complete low small-intestinal type of obstruction, but with the extra risk of colonic perforation (Fig. 6).

(The clinical patterns of obstruction, pathological causes, and management will be discussed in next week's article.)

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TODAY'S DRUGS

With the help of expert contributors we print in this section notes on drugs in current use.

Oral Contraceptives—Choice of Product

Oral contraceptives administered for 20 or 21 days each cycle are now in general use throughout the world, and when properly taken are the most effective birth control method ever known, as well as being a most convenient form in which to prescribe hormonal treatment for various gynaecological troubles.

The medical practitioner, however, is confronted by a proliferation of products, all varying slightly in their spectrum of effects and in the overall oestrogen/progestogen balance. He may have difficulty, therefore, in choosing a suitable product for a particular patient and in deciding the best alternative product if undesirable side-effects occur.

Most products consist of one or other of nine orally active synthetic progestogens with one or other of the two synthetic oestrogens, ethinyl oestradiol (O.E.) or mestranol. The progestogens fall into two main groups. Norethynodrel, norethisterone and its acetate, lynoestrenol, ethynodiol diacetate, and norgestrel are all related to 19-nortestosterone. These compounds inhibit ovulation by blocking release of luteinizing-hormone and are to a varying extent metabolized to oestrogen, but nevertheless exert an anti-oestrogenic effect on organs such as the cervix and the endometrium. They also have mild anabolic activity, and have a strongly haemostatic effect on bleeding of endocrine origin. The second group, the 17 alpha-hydroxy progesterone compounds, do not inhibit ovulation, have no inherent oestrogenic effect, but indeed have a pro-

nounced anti-oestrogenic effect. Chlormadinone and megestrol acetate are the two available products in this group.

As far as the combined oestrogen plus progestogen products are concerned the overall balance, whether predominantly progestogenic or oestrogenic, depends on a number of factors. The *progestogenic* effect depends on the particular progestogen in the combination tablet and its spectrum of activity. It should be noted that the potency cannot be inferred from the milligram dosage, and progestogens cannot be classified as strongly or weakly progestogenic according to the dose. The progestational potency of a compound may be increased either by an increase in the dose of the progestogen or by a decrease in the amount of oestrogen or both; similarly, the oestrogenic potency of a compound can be increased by reducing the dose of progestogen or by increasing the dose of oestrogen or both. For all practical purposes the two oestrogens are similar in potency. The *oestrogenic* effect of a combined product, however, depends not only on the actual dose of oestrogen, but also on the particular progestogen with which it is combined and its dose: progestogens which are converted partly to oestrogens increase the overall oestrogenic effect. On the other hand, most progestogens oppose or decrease the effect of oestrogen on the tissues (that is the anti-oestrogenic effect), and the extent to which they do so has to be taken into account. Furthermore, the endogenous oestrogens are much reduced by oral contraceptive medication—again with considerable variation in the extent.

In the sequential formulations oestrogen only in dosage sufficient to inhibit ovulation is given in the early part of the cycle, and a progestogen is added for the last few days to regularize the withdrawal bleeding and prevent excessive blood