# Current Practice

## Arterial Disease of the Gut

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Knowledge of ischaemic lesions of the alimentary tract has increased in recent years owing to the technical advances of radiology and vascular surgery. Lateral aortography and selective visceral angiography have facilitated the diagnosis of chronic ischaemic lesions, and vascular surgical techniques can offer restoration of normal blood flow.

The intestinal tract and accessory digestive organs are supplied with arterial blood through the three main arteries distributed to the fore, mid, and hind guts—that is, the coeliac axis and the superior mesenteric and inferior mesenteric arteries. The first two anastomose via the pancreatico-duodenal anastomosis and the two mesenteric arteries via the marginal artery of the colon. In sudden occlusion of healthy main arteries these anastomoses may be unable to maintain viability of the bowel, but in chronic arterial lesions where ischaemia develops slowly they can hypertrophy and supply sufficient blood to prevent necrosis. The adequacy of this collateral circulation will determine whether the patient remains symptom-free or whether he develops symptoms of intestinal ischaemia.

When arterial lesions lie distal to the main trunks the rich anastomosis of the arteries of the stomach, the numerous arcades in the arteries of the small intestine, and the marginal artery of the colon usually prevent ischaemia of bowel unless the lesions are widespread throughout the small arteries (e.g., thromboangiitis obliterans). At the present time radiological visualization of the small arteries is difficult even with selective catheterization techniques, and the part played by distal lesions in gut ischaemia is not yet delineated.

### Acute Intestinal Ischaemia

#### Superior Mesenteric Lesions

Acute occlusion of the superior mesenteric artery is due either to thrombosis or embolism.

The commonest cause of thrombosis is atherosclerosis, which usually narrows the proximal 2–3 cm. of the artery, but a minority of cases have intimal thickening and stenosis extending below the level of the mesocolon. Thrombosis is the final event in a long-standing degenerative process, and approximately 50% of cases experience premonitory symptoms of chronic ischaemia prior to developing acute infarction.[1]

Because of its size and the acute angle made with the abdominal aorta embolism of the superior mesenteric artery is commoner than that of the coeliac axis or the inferior mesenteric artery. The results depend mainly on the size of the embolus. If it is large extensive midgut necrosis will occur, but if it is small, if it breaks up into several fragments on impact, or if it consists of a shower of small emboli the arterial blockage may occur distally and the level and extent of ischaemic bowel will vary according to the degree of embolization and the development of a collateral blood flow.

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Other less common causes of acute ischaemia are superior mesenteric venous occlusion and "non-occlusive mesenteric ischaemia." Occlusion of the superior mesenteric vein may occur in cirrhosis of the liver, in portal or splenic vein thrombosis, or owing to compression by tumour. Non-occlusive mesenteric ischaemia[2] is a condition in which the bowel becomes necrotic in the absence of arterial or venous obstruction, and it is usually associated with or due to severe congestive heart failure, digitalis intoxication, or severe haemoconcentration.

Thrombosis and embolism of the superior mesenteric artery give similar pictures. In the early stages the bowel becomes pale and peristalsis may increase, but this is soon followed by congestion and thrombosis of the distal vessels in the absence of an adequate collateral flow. Within a few hours whole or part of the midgut becomes necrotic. Clinically, the probability of one or the other may be deduced from other evidence. In atherosclerosis there may be evidence of peripheral arterial occlusion, previous coronary artery thrombosis, or cerebrovascular accident, and in embolism it may be possible to recognize a possible source. Emboli are usually derived from the left atrium or auricular appendage in fibrillation, from the left ventricle when mural thrombus forms in coronary thrombosis, or from vegetations on diseased valves.

*Clinical Features.*—The initial presentation is usually that of an acute vascular catastrophe[3] with abdominal pain, general pallor, severe hypotension, and tachycardia, followed later by abdominal distension and peritonitis. Abdominal tenderness is present, but rigidity is frequently absent. Bowel sounds may initially be increased, but the abdomen soon becomes silent as peritonitis supervenes. The rate of deterioration of the patient's condition varies according to the amount of blood and fluid loss into the bowel, the extent of bowel necrosis, the patient's previous fitness, and the rate of bacterial proliferation. Some deteriorate dramatically, while others maintain a good general condition until laparotomy. The passage of blood in the stool is emphasized in many textbooks, but it occurs in less than half of the cases and is usually too late an event to rely on for diagnosis.

Some patients have a less dramatic presentation with insidious onset of ill-defined pain and gradual deterioration in their general condition. The diagnosis may not be suspected for some time and the initial few hours during which restoration of bowel viability is possible may be passed over by the patient who is reluctant to call his doctor or by the doctor or surgeon who is puzzled by the symptoms and signs.

Conservative treatment by transfusion, antibiotics, and anticoagulants is unlikely to yield any worth-while results, and early laparotomy is mandatory. The surgeon is then usually confronted by greenish-black necrotic bowel whose resection would leave inadequate bowel for long-term survival, and abdominal closure is followed by death within hours or a day or two.

A few patients can be saved. Successful embolectomy has been achieved on a number of occasions, intestinal necrosis may be limited in extent and successful resection achieved, or

bypass grafting from aorta to mesenteric artery may be possible. After an apparently successful operation further thrombosis may produce necrosis of the remaining bowel, and it is often advisable to reopen the abdomen a day or two later to anticipate this highly fatal complication. Survival depends on operative intervention within hours of onset or on the chance limitation of necrosis to a restricted segment, but the infrequent occurrence in any one clinical practice of these emergencies and the almost inevitable delay in diagnosis or operative intervention militate against survival.

The mortality rate approaches 100%, and the emphasis is now directed towards recognition of the prodromal symptoms of chronic intestinal ischaemia, at which stage operative intervention can revascularize the bowel and prevent thrombosis.

## Inferior Mesenteric Lesions

The effects produced by occlusion of the inferior mesenteric artery depend on the size of the artery and the integrity of the marginal artery of the colon. When the origin or the proximal centimetre or two of the artery become narrowed, as may occur in atherosclerosis or abdominal aneurysm, the colon is supplied by additional blood from the superior mesenteric artery via the marginal artery of the colon. In these circumstances occlusion or operative ligation of the inferior mesenteric artery may be symptomless or may produce only transient ischaemia.

When the inferior mesenteric artery is large, or when the marginal artery of the colon has been interrupted by previous bowel resection, the consequences of inferior mesenteric occlusion can be serious. The inferior mesenteric artery frequently becomes hypertrophied in response to superior mesenteric stenosis or occlusion and the midgut's vascular requirements are then supplied in part or whole via an enlarged marginal artery. In such circumstances ligation or occlusion of the inferior mesenteric artery gives rise to acute ischaemia of the hind gut and sometimes part of the midgut as well.

One additional important factor is the presence of virulent colonic bacteria, which may produce a fulminating infective colitis.

*Clinical Picture.*—Acute occlusion of the inferior mesenteric artery can therefore be symptomless or may present in different clinical forms as : (1) Transient ischaemia of part of the hind gut with congestion of bowel, abdominal pain, and bloody diarrhoea resolving spontaneously in a day or two. (2) A haemorrhagic proctocolitis resembling acute haemorrhagic ulcerative colitis with abdominal pain, diarrhoea, and granular rectal mucosa. (3) Incomplete recovery from either of the above can lead to atrophy of colon and simple stricture which may later give rise to intestinal obstruction. A similar stenosis is occasionally seen after closure of a colostomy, where interference with the blood supply causes atrophy and replacement fibrosis. (4) Acute infarction of the hind gut with necrosis, giving rise to acute abdominal pain, bloody diarrhoea, and tenderness and rigidity in the left side of the abdomen. Resection of the gangrenous bowel may be followed by recovery, but deterioration due to blood and fluid loss and toxaemia is usually rapid and the outcome often fatal. (5) Acute necrotizing colitis involving a segment or most of the hind gut. Laparotomy reveals necrotic colon whose ischaemic cause is unrecognized owing to the virulence of the coincident infection. These patients also suffer severely from toxaemia, and *Clostridium welchii* has been incriminated as the principal tissue invader in some cases.

"Ischaemic colitis" has been classified by Marston and his colleagues[4] into gangrenous, stricturing, and transient forms, and they suggest that it is a relatively common condition whose underlying vascular aetiology is not always evident.

Subsequent investigation may confirm the diagnosis. Aortography may demonstrate an occlusion of the inferior mesenteric artery, or barium enema may reveal the presence of sacculation, thumb-printing, or stricture of the colon.

## Chronic Intestinal Ischaemia

Chronic ischaemia of the gut is due to stenosis or occlusion of one or more of the three visceral arteries.

It is usually assumed that all three arteries are essential to normal function, but congenital stenosis or absence of either the coeliac axis or superior mesenteric artery may occur, and occasionally both of them can arise from a common trunk. Cases have been described in which all three arteries have been obliterated or devoid of a direct blood supply from the aorta[5] and no symptoms of ischaemia resulted.

Arterial abnormality or pathological occlusion can therefore exist without causing symptoms. Even when symptoms of chronic intestinal ischaemia do occur there is as yet no answer to the problem of which or how many cases inevitably proceed to acute ischaemia if untreated.

*Aetiology.*—The commonest cause of chronic intestinal ischaemia is atherosclerosis, in which it is usual for all three arteries to be diseased, although the lesion usually predominates in one vessel—most commonly the superior mesenteric artery. Sometimes the inferior mesenteric artery escapes and a hypertrophied artery supplies a large volume of blood through the marginal artery to the rest of the gut. Morris and his colleagues[6] state that symptoms usually do not arise until two of the three arteries are involved by disease, but in 1966 they suggested[7] that when symptoms are produced by occlusion of only one artery it is the coeliac axis which is affected.

It is now recognized that chronic intestinal ischaemia may be due to causes other than atherosclerosis. Stenosis of the coeliac axis alone may be due to constriction caused by the median arcuate ligament of the diaphragm.[8] On the aortogram this can be recognized by apparent notching of the upper surface of the outline of the coeliac axis, and at operation the coeliac axis is seen to take origin from the aorta behind the median arcuate ligament. Most of these patients are young or middle-aged, and, as the development of symptoms in adult life makes it unlikely that the lesion is congenital, it may be that some alteration in the relationship between the abdominal aorta and the median arcuate ligament has developed in adult life, possibly consequent upon elongation of the abdominal aorta.

Stenosis of the coeliac axis or superior mesenteric artery, stenosis of both arteries, or occlusion of either may be due to constriction of the main trunk of these vessels by fibrous tissue.[9] Fibrosis of tissues in and around the coeliac ganglia which encircle the two arteries is not uncommon, although the aetiology is obscure. Less commonly the perivascular fibrosis may be merely part of a generalized retroperitoneal fibrosis which in its common form presents by fibrotic constriction of the ureters.

Other less common conditions causing reduction or interruption of arterial flow are aneurysmal dilatation of aorta or visceral arteries, pancreatic or other tumours involving retroperitoneal tissues by direct spread, tumours originating retroperitoneally, and small artery lesions such as occur in thromboangiitis obliterans or rheumatic disease.

*Symptoms and Signs.*—Although the pattern of vascular involvement and the degree of collateral development are variable, the symptoms and signs of chronic intestinal ischaemia are caused by altered motility and inadequate absorption. The clinical picture is often indistinct and difficult to elucidate, and failure to make a correct diagnosis is usually due to failure of the clinician to suspect the existence of the condition.

The cardinal symptom is pain occurring after meals. In its characteristic form it occurs relentlessly after every meal and does not have the periodicity typical of ulcer pain. Its severity is increased by large meals and the patient tends to eat small

meals to avoid pain. In so far as it is an expression of inadequate blood flow in response to increased functional demands, it merits the classical description of "abdominal angina." In the majority of cases the pain is atypical in distribution and severity and is dull and poorly localized or colicky in type. Vomiting can occur and the bowel habit tends towards intermittent diarrhoea or, where fat absorption is grossly deficient, the voiding of pale bulky stools. Inadequate absorption of food, a reduction in the size of meals, and repeated vomiting all tend to produce loss of weight which may be severe and sometimes the most striking feature of the patient's condition.

Routine clinical examination is usually negative, apart from evidence of weight loss. A bruit may be audible in the presence of arterial stenosis, and the use of a stethoscope on the abdomen is a valuable aid to diagnosis. The bruit due to stenosis of the coeliac axis or superior mesenteric artery must be differentiated from that occurring in aortic bifurcation stenosis, in which there is associated reduction in the volume of the femoral pulses, from that of aortic aneurysm, which is usually but not always palpable, and from stenosis of the renal artery.

*Diagnosis.*—The patient may present with characteristic symptomatology, but more often the clinical picture is vague and indeterminate. Barium investigations are usually negative and failure to make the diagnosis is frequent.

Duodenal ulcer or other duodenal abnormality may be reported on barium meal and prove misleading, as duodenal ulcer and intestinal ischaemia can coexist.[10] The patient may be treated for some time by diet and antacids without relief of symptoms, and the true diagnosis becomes evident only when fatal intestinal infarction ensues.

Severe weight loss, which is a constant feature of chronic intestinal ischaemia, may suggest the presence of alimentary carcinoma, and suspicion may be increased by a positive test for faecal occult blood even when radiological demonstration of a tumour is lacking. If laparotomy is undertaken and no tumour found the surgeon may fail to appreciate that mesenteric arterial pulsation is reduced or absent.

In other patients inadequate absorption is the principal feature of the condition and loss of weight and steatorrhoea can be severe. Special investigations may show further evidence of impaired intestinal absorption.[10] Jejunal biopsy shows flattening and atrophy of the villi, and estimation of the enzyme content of the mucosa may show reduction in the sucrase, maltase, and lactase present. Glucose and lactose tolerance curves may be flattened and xylose absorption abnormal. Routine liver-function tests are usually normal, but bromsulphalein excretion can be abnormally high. Valuable time may be lost if such evidence of malabsorption is misinterpreted as adult coeliac disease or "malabsorption syndrome" or if the response to a gluten-free diet is awaited.

It must be emphasized that aortography should be undertaken when the possibility of chronic intestinal ischaemia is suspected and the patient is fit. There are undoubted complications of aortography, but these are not frequent or serious in skilled hands and the possibility of their occurrence does not contraindicate this investigation. Films taken in lateral projection will show the three visceral arteries in profile and abnormalities of the main trunks can be readily seen. The technique employed is usually the Seldinger method of percutaneous femoral catheterization, the catheter being passed upwards into the upper abdominal aorta before dye injection. Where aorto-iliac disease or tortuosity of vessels prevents the use of this technique translumbar aortography in the lateral position is undertaken.

*Surgical Treatment.*—When an arterial lesion associated with symptoms of chronic intestinal ischaemia has been demonstrated operation should be undertaken and revascularization of the intestinal tract performed by one of the several methods available. Full restoration of blood flow is not essential to recovery of the gut (unlike renal artery stenosis, where complete restoration of flow is obligatory), and revascularization of the superior mesenteric artery alone usually suffices, although Morris *et al.*[7] describe cases in which revascularization of more than one artery was undertaken.

At the present time preference is usually given to aorto-mesenteric bypass grafting using Dacron or saphenous vein, but effective restoration of blood flow can be achieved also by reimplantation of the superior mesenteric artery into the aorta, by lateral anastomosis of superior mesenteric artery and aorta, or, where other methods are not possible, by anastomosis of the ileo-colic artery to the right common iliac artery.

When the lesion is due to periarterial fibrosis or constriction by the median arcuate ligament, simple division of the constricting structures ensures adequate improvement in blood flow.

The prognosis after operation is good, especially in cases with simple constriction of the vessels. When atherosclerosis is the cause the prognosis depends largely on the natural history of this disease.

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# TODAY'S DRUGS

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

## The Macrolides and Lincomycin

The macrolides are a large group of antibiotics of which three are in clinical use. Others such as carbomycin have been abandoned because of some defect, or like tylosin have been used only in livestock.

### Erythromycin

This was the first macrolide discovered, and may well be considered still to be the best.

*Antibacterial Activity.*—The spectrum of erythromycin corresponds closely to that of penicillin: Table I compares them for a few representative species. Most bacteria may be

TABLE I.—*Minimum Inhibitory Concentrations (μg./ml.)*

| Species | Penicillin | Erythromycin |
|---|---|---|
| Gram-positive | | |
| *Str. pyogenes* | 0·01 | 0·03 |
| *" pneumoniae* | 0·02 | 0·03 |
| *Staph. aureus* | 0·02 | 0·12 |
| *Cl. welchii* | 0·1 | 0·8 |
| Gram-negative | | |
| *N. gonorrhoeae* | 0·005 | 0·4 |
| *H. influenzae* | 1·0 | 1·5 |
| *S. typhi* | 5–20 | 75 |
| *Proteus mirabilis* | 8 | 250 |
| *Esch. coli* | 25–100 | 100 |