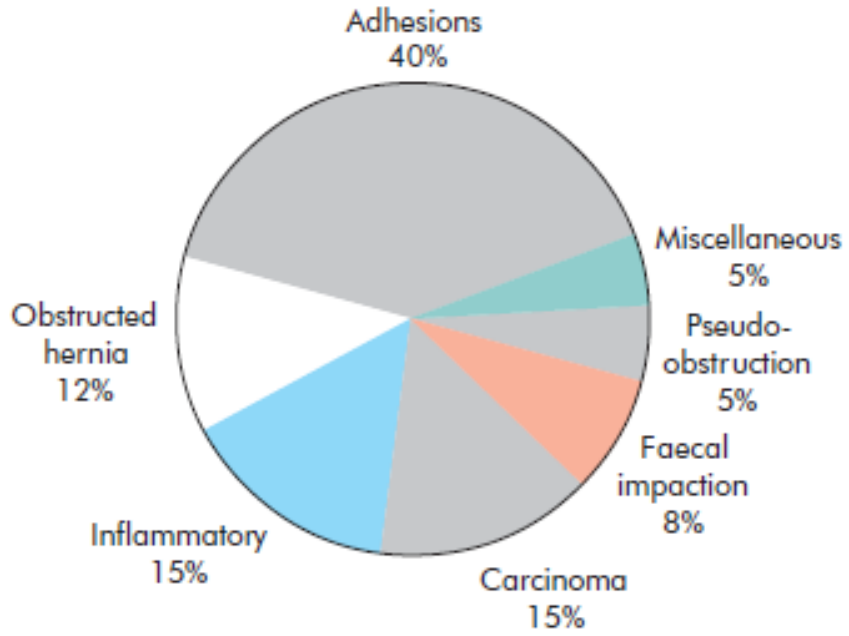
Intestinal obstruction

Intestinal obstruction may be classified into two types.

• **Dynamic** — where peristalsis is working against a mechanical obstruction. The obstructing lesion may be:
—***intraluminal***, for example impacted faeces, foreign bodies, bezoar, gallstones;
— ***intramural***, for example malignant or inflammatory strictures;
—***extramural***, for example intraperitoneal bands and adhesions, hernias, volvulus or intussusception.
• **Adynamic** — this may occur in two forms. Peristalsis may be absent (e.g. paralytic ileus) or it may be present in a non-propulsive form (e.g. mesenteric vascular occlusion or pseudo-obstruction). In both types a mechanical element is absent.



Pie chart showing the common causes of dynamic intestinal obstruction and their relative frequencies.

Dynamic obstruction
The diagnosis of intestinal obstruction is based on the classic quartet of pain, distension, vomiting and absolute constipation.
Obstruction may be classified clinically into two types:
• Small *bowel* obstruction — high or low;
• Large bowel obstruction.
In high small bowel obstruction vomiting occurs early and is profuse with rapid dehydration. Distension is minimal with little evidence of fluid levels on abdominal radiography.
In low small bowel obstruction pain is predominant with central distension. Vomiting is delayed. Multiple central fluid levels are seen on radiography.

In large-bowel obstruction distension is early and pronounced. Pain is mild and vomiting and dehydration are late. The proximal colon and caecum are distended on an abdominal radiograph.

The nature of presentation will also be influenced by whether the presentation is:

•Acute;
• Chronic;
• Acute on chronic;
• Subacute.

Acute obstruction usually occurs in small bowel obstruction with sudden onsets of severe colicky central abdominal pain, distension, with early vomiting and constipation.

Chronic obstruction is usually seen in large bowel obstruction with lower abdominal colic and absolute constipation, followed by distension.

In acute on chronic obstruction there is a short history of distention and vomiting against a background of pain and constipation.

Subacute obstruction implies an incomplete obstruction.

Presentation will be further influenced by whether the obstruction is:

• Simple — where the blood supply is intact;
•Strangulating/strangulated — where there is direct interference to blood flow, usually by hernial rings or intra-peritoneal adhesions/bands.

Pathophysiology

Irrespective of aetiology or acuteness of onset, the proximal bowel dilates and develops an altered motility. Below the obstruction, the bowel exhibits normal peristalsis and absorption until it becomes empty, when it contracts and becomes immobile. Initially, proximal peristalsis is increased to overcome the obstruction, with the length of time it remains vigorous being proportional to the distance of the obstruction. If the obstruction is not relieved the bowel begins to dilate causing a reduction in peristaltic strength, ultimately resulting in flaccidity and paralysis. This is a protective phenomenon to prevent vascular damage secondary to increased intraluminal pressure.

The distension proximal to an obstruction is produced by two factors:

• **Gas** — regardless of the level of obstruction, there is a significant overgrowth of both aerobic and anaerobic organisms resulting in considerable gas production. Following the reabsorption of oxygen and carbon dioxide, the majority is made up of nitrogen (90 per cent) and hydrogen sulphide.

• **Fluid** — this is made up of the various digestive juices. Following obstruction, fluid accumulates within the bowel wall and any excess is secreted into the lumen, whilst absorption from the gut is retarded.

Dehydration and electrolyte loss are therefore due to:

— Reduced oral intake;
— Defective intestinal absorption;
— Losses due to vomiting;
— Sequestration in the bowel lumen.

Strangulation

When strangulation occurs the viability of the bowel is threatened secondary to a compromised blood supply. This may be due to:

• ***External compression*** (hernial orifices/adhesions/bands);

• ***Interruption of mesenteric flow*** (volvulus, a twist of bowel loop on its mesenteric pedicle or intussusception where a segment of bowel invaginates into an adjacent segment);

• ***Rising intraluminal pressure*** (closed-loop obstruction);

• ***Primary obstruction of intestinal circulation*** (mesenteric infarction).

The venous return is compromised before the arterial supply unless primary obstruction in the mesenteric artery is present. The resultant increase in capillary pressure leads to local mural distension with loss of intravascular fluid and red blood cells intramurally and intra-and extraluminally. Once the arterial supply is impaired, haemorrhagic infarction occurs. As the viability of the bowel wall is compromised there is marked translocation and systemic exposure to aerobic and anaerobic organisms with their associated toxins. The associated danger is far greater for intraperitoneal strangulation than that of an external hernia where there is a smaller absorptive surface.

The morbidity and mortality associated with strangulation are dependent on age and extent

In strangulated external hernias the segment involved is short and the resultant blood and fluid loss is small. When bowel involvement is extensive the loss of blood and circulatory volume will cause peripheral circulatory failure.

Closed-loop obstruction

This occurs when the bowel is obstructed at both the proximal and distal point. It is present in many cases of intestinal strangulation. Unlike cases of nonstrangulating obstruction, there is no early distension of the proximal intestine. When gangrene of the strangulated segment is imminent, retrograde thrombosis of the mesenteric veins results in distension on both sides of the strangulated segment.

A classic form of closed-loop obstruction is seen in the presence of a tight carcinomatous stricture of the colon with a competent ileocaecal valve (present in up to a third of individuals). The inability of the distended colon to decompress itself into the small bowel results in an increase in luminal pressure, greatest at the caecum, with subsequent impairment of blood supply. Unrelieved, this result in necrosis and perforation.

Acute intestinal obstruction

Clinical features

There are four cardinal features:

• Pain;

• Vomiting;

• Distension;

• Constipation.

These features vary according to:
• Location of the obstruction;
• Age of the obstruction;
• Underlying pathology;
• Presence or absence of intestinal ischaemia.

Late manifestations which may be encountered include dehydration, oliguria, hypovolaemic shock, pyrexia, septicaemia, respiratory embarrassment and peritonism. In all cases of suspected intestinal obstruction, all hernial orifices must be examined.

Pain

Pain is the first symptom; it occurs suddenly and is usually severe. It is colicky in nature and is usually centred around the umbilicus (small bowel) or lower abdomen (large bowel). The pain coincides with increased peristaltic activity. With increasing distension, the colicky pain is replaced by a mild constant diffuse pain. The development of severe pain is indicative of the presence of strangulation. Pain may not be a significant feature in postoperative simple mechanical obstruction and does not occur in paralytic ileus.

Vomiting

The more distal the obstruction, the longer the interval between the onset of symptoms and the appearance of nausea and vomiting. As obstruction progresses the character of the vomitus alters from digested food to faeculent material due to the presence of enteric bacterial overgrowth.

Distension

In the small bowel the degree of distension is dependent on the site of the obstruction and is greater the more distal the lesion. Visible peristalsis may be present. ***It is delayed in colonic obstruction and may be minimal or absent in the presence of mesenteric vascular occlusion.***

Constipation

This may be classified as absolute (i.e. neither faeces nor flatus is passed) or relative (where flatus only is passed). Absolute constipation is a cardinal feature of complete intestinal obstruction. Some patients may pass flatus or faeces after the onset of obstruction owing to the evacuation of distal bowel contents.

The rule that constipation is present in intestinal obstruction does not apply in:
• Richter's hernia;
• Gallstone obturation;
• Mesenteric vascular occlusion;
• Obstruction associated with a pelvic abscess;
• Partial obstruction (faecal impaction/colonic neoplasm) where diarrhoea may often occur.

Other manifestations

Dehydration

This is seen most commonly in small bowel obstruction due to repeated vomiting and fluid sequestration. This results in dry skin and tongue, poor venous filling and sunken eyes with oliguria. The blood urea level and haematocrit rise giving a secondary polycythaemia.

Hypokalaemia
This is not a common feature in simple mechanical obstruction. An increase in serum potassium, amylase or lactate dehydrogenase may be associated with the presence of strangulation, as may leucocytosis or leucopenia.
Pyrexia in the presence of obstruction may indicate:
• The onset of ischaemia;
• Intestinal perforation;
• Inflammation associated with the obstructing disease.
Hypothermia indicates septicaemic shock.

Abdominal tenderness
Localized tenderness indicates pending or established ischaemia. The development of peritonism or peritonitis indicates overt infarction and/or perforation.

Clinical features of strangulation
It is vital to distinguish strangulating from nonstrangulating intestinal obstruction, as the former is a surgical emergency. The diagnosis is entirely clinical. In addition to the features outlined above, the following should be noted:
• The presence of shock indicates underlying ischaemia;
• In impending strangulation, pain is never completely absent;
• Symptoms usually commence suddenly and recur regularly;
• In nonstrangulated obstruction there may be an area of localized tenderness at the site of the obstruction; in strangulation there is always localized tenderness associated with rigidity/rebound tenderness.
• Generalized tenderness and the presence of rigidity are indicative of the need for early laparotomy.
• In cases of intestinal obstruction where pain persists despite conservative management, even in the absence of the above signs, strangulation should be diagnosed.
• When strangulation occurs in an external hernia the lump is tense, tender, irreducible, there is no expansile cough impulse and it has recently increased in size.

Radiological diagnosis
Both erect and supine abdominal films are required.
When distended with gas the jejunum, ileum, caecum and remaining colon have a characteristic appearance that allows them to be distinguished radiologically. The diameter of the distended viscus is not diagnostic.
• The obstructed small bowel is characterized by straight segments that are generally central and lie transversely. No gas is seen in the colon.
• **The jejunum** is characterized by its valvulae conniventes that completely pass across the width of the bowel and are regularly spaced giving a ‘concertina’ or ladder effect.
• Ileum — the distal ileum is described as being featureless.
• **Caecum** — a distended caecum is shown by a rounded gas shadow in the right iliac fossa.

• **Large bowel** — except for the caecum shows haustral folds which, unlike valvulae conniventes, are spaced irregularly and the indentations are not placed opposite one another.

Volvulus of the sigmoid colon has a characteristic radiological appearance with a grossly dilated loop of colon, with or without visible haustrae which arises from the pelvis and extends obliquely across the spine to the upper abdomen.

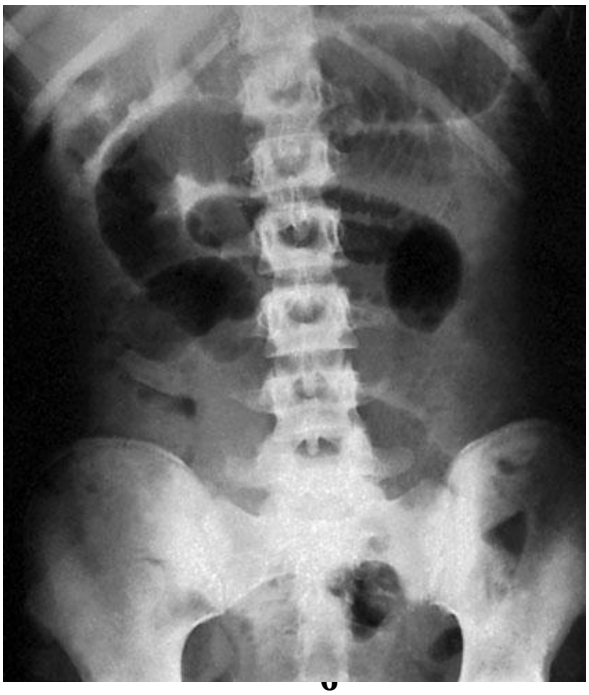
In intestinal obstruction **fluid levels** appear later than gas shadows as it takes time for gas and fluid to separate. In infants less than 2 years of age, a few fluid levels in the small bowel may be physiological. In adults, two inconstant fluid levels may be regarded as normal — one at the duodenal cap and the other in the terminal ileum.

During the obstructive process, fluid levels become more conspicuous and more numerous when paralysis has occurred. When fluid levels are pronounced the obstruction is advanced. In the small bowel, the number of fluid levels is directly proportional to the degree of obstruction and to its site; the number increasing the more distal the lesion.

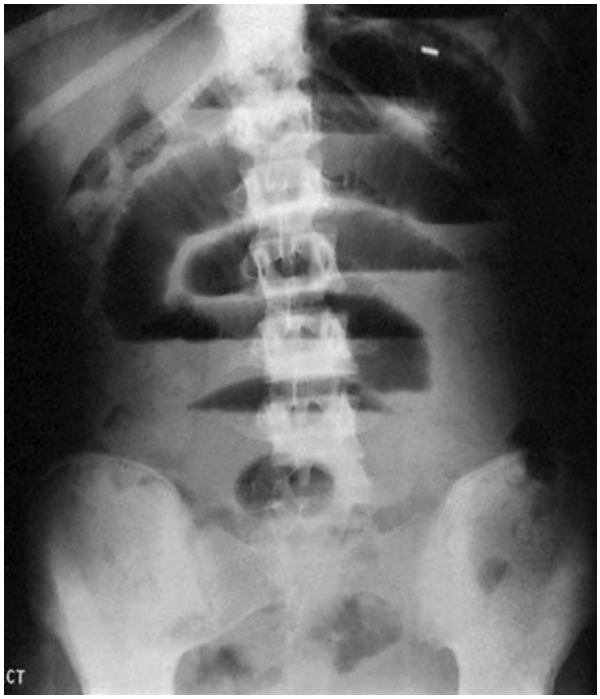
In contrast, low colonic obstruction does not commonly give rise to small bowel fluid levels unless advanced, whilst high colonic obstruction may do in the presence of an incompetent ileocaecal valve. Colonic obstruction is usually associated with a large amount of gas in the caecum. A limited water-soluble enema may be undertaken to differentiate large bowel obstruction from pseudo-obstruction. A barium follow-through is contraindicated in the presence of acute obstruction and may be life threatening.

Impacted foreign bodies may be seen on abdominal radiographs. **In gallstone ileus,** gas may be seen in the biliary tree with the stone visible, usually in the right iliac fossa, in 25 per cent of cases.

It is noteworthy that gas-filled loops and fluid levels in the small and large bowel can also be seen in established paralytic ileus and pseudo-obstruction. The former can, however, normally be distinguished on clinical grounds whilst the latter can be confirmed radiologically. Fluid levels may also be seen in non obstructing conditions such as inflammatory bowel disease, acute pancreatitis and intra-abdominal sepsis.



Gas-filled small bowel loop; patient supine



Fluid levels with gas above; 'stepladder pattern'. Ileal obstruction by adhesions; patient erect

Treatment of acute intestinal obstruction

There are three main measures:

• Gastrointestinal drainage;
• Fluid and electrolytic replacement;
• Relief of obstruction, usually surgical.

The first two steps are always necessary prior to the surgical relief of obstruction and are the mainstay of post-operative management. In a proportion of cases, particularly adhesive obstruction, they may be used exclusively.

Surgical treatment is necessary for most cases of intestinal obstruction, but should be delayed until resuscitation is complete, ***provided there is no:***

• Sign of strangulation;
• Evidence of closed-loop obstruction.

Supportive management

• Nasogastric decompression is achieved by the passage of a nasogastric tube. The tubes are nor-mally placed on free drainage, with 4-hourly aspiration, but may be placed on continuous or intermittent suction. As well as facilitating decompression proximal to the obstruction, they also reduce the risk of subsequent aspiration during induction of anaesthesia and postextubation.

• The basic biochemical abnormality is sodium and water loss, and therefore the appropriate replacement is Hartmann's solution or normal saline. The volume required varies and should be determined by clinical haematological and biochemical criteria.
• Antibiotics — whilst not mandatory, many clinicians initiate broad-spectrum antibiotic early in therapy because of bacterial overgrowth. Antibiotic therapy is mandatory for all patients undergoing small or large bowel resection.

Surgical treatment للاطلاع
The timing of surgical intervention is dependent on the clinical picture with the indications of early operation being:

• Obstructed or strangulated external herniae;
• Internal intestinal strangulation;
• Acute obstruction.
The classic clinical advice that 'the sun should not both rise and set' on a case of unrelieved intestinal obstruction is sound and should be followed unless there are positive reasons for delay. Such cases may include obstruction secondary to adhesions where there is no pain or tenderness, despite continued radiological evidence of obstruction. Under these circumstances, conservative management may be continued for up to 72 hours in the hope of spontaneous resolution.
If the site of obstruction is unknown, adequate exposure is best achieved by a midline incision. ***Operative assessment is directed to:***
• The site of obstruction;
• The nature of the obstruction;
• The viability of the gut.
Identification and assessment of the caecum is the best initial manoeuvre. If it is collapsed, the lesion is in the small bowel and may be identified by careful retrograde assessment. A dilated caecum indicates large bowel obstruction. To display the cause of obstruction, distended loops of small bowel should be displaced with care and covered with warm moist abdominal packs.
Operative decompression may be required if dilatation of bowel loops prevents exposure, the viability of the bowel wall is compromised or subsequent closure will be compromised. Its benefits should be balanced against potential risk of septic complications from spillage. Decompression may be performed using Savage's decompressor within a seromuscular purse-string suture. Alternatively, with a large bore nasogastric tube in place the small bowel contents may be gently milked in a retrograde manner to the stomach for aspiration. All volumes of fluid removed should be accurately measured and appropriately replaced.
The type of surgical procedure required will depend upon the nature of the cause — division of adhesions (enterolysis), excision, bypass or proximal decompression.

Following relief of obstruction, the viability of the involved bowel should be carefully assessed. Whilst frankly infarcted bowel is obvious, the viability status in many cases may be difficult to discern. If in doubt, the bowel should be wrapped in hot packs for 10 minutes with increased oxygenation and reassessed. The state of the mesenteric vessels and pulsation in adjacent

arcades should be sought. Nevertheless, nonocclusive vascular insufficiency may occur despite adequate pulsation. In doubtful cases, following resection, both ends of the bowel should be raised as stomas. This is not only safe but also allows regular assessment of the bowel. Where no resection has been undertaken or there are multiple ischaemic areas (mesenteric vascular occlusion) a second look laparotomy at 24—48 hours may be required.
Special attention should always be paid to the sites of constriction at each end of an obstructed segment. If of doubtful viability they should be infolded by the use of a seromuscular suture and covered with omentum.
The surgical management of massive infarction in the form of superior mesenteric artery occlusion is dependent on the patient's overall prognostic criteria. In the elderly, infarction of the small bowel from the duodenojejunal junction and involving the whole right colon may be considered incurable, whilst in the young, with potential for long-term intravenous alimentation and small bowel transplantation, a less conservative policy may be justified. Whenever small bowel is resected, the exact site of resection, the length of the resected segment and that of the residual bowel should be recorded.

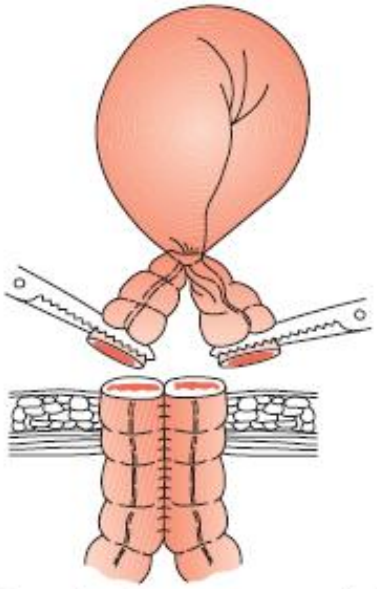
Differentiation between viable and non-viable intestine

	Viable	Non-viable
Circulation	Dark colour becomes lighter	Dark colour remains
	Mesentery bleeds if pricked	No bleeding if mesentery is pricked
Peritoneum	Shiny	Dull and lustreless
Intestinal musculature	Firm	Flabby, thin and friable
	Pressure rings may or may not disappear	Pressure rings persist
	Peristalsis may be observed	No peristalsis

Acute large bowel obstruction

This is usually due to underlying carcinoma or occasionally diverticular disease, and presents in an acute or chronic form. The condition of pseudo-obstruction should always be considered and excluded by a limited contrast study or air computerised tomography (CT) scan to confirm organic obstruction.
After full resuscitation the abdomen should be opened through a midline incision. Distension of the caecum will confirm large bowel involvement. Identification of a collapsed distal segment of the large bowel and its sequential proximal assessment will readily lead to identification of the cause. When a removable lesion is found in the caecum, ascending colon, hepatic flexure or proximal transverse colon an emergency right hemicolectomy should be performed. If the lesion is irremovable, a proximal stoma (colostomy or ileosotomy if the ileocaecal valve is incompetent) or ileotransverse bypass should be considered. Obstructing lesions at the

splenic flexure should be treated by an extended right hemicolectomy with ileodescending colonic anastomosis.
For obstructing lesions of the left colon or rectosigmoid junction, immediate resection should be considered unless there are clear contraindications such as:
• Inexperienced surgeon;
• Moribund patient;
• Advanced disease.
In rare instances, or where caecal perforation is imminent, time to improve the patient's clinical condition can be bought by performing an emergency caecostomy (or ileosotomy in the presence of an incompetent ileocaecal valve).
In the absence of senior clinical staff, it is safest to bring the proximal colon to the surface as a colostomy. Where possible the distal bowel should be brought out at the same time (Paul—Mikulicz procedure) to facilitate subsequent extraperitoneal closure.



The Paul–Mikulicz operation applied to volvulus of the pelvic colon.

In the majority of cases the distal bowel will not reach the skin and is closed and returned to the abdomen (Hartmann's procedure). A second-stage colorectal anastomosis can be planned when the patient is fit.
If an anastomosis is to be considered using proximal colon, in the presence of obstruction, it must be decompressed and cleaned by an on-table colonic lavage. Nevertheless, the subsequent anastomosis should still be protected with a covering stoma.

Obstruction from enteric strictures
Small bowel strictures usually occur secondary to tuberculosis or Crohn's disease. Malignant strictures associated with lymphoma are common, whilst carcinoma and sarcoma are rare. Presentation is usually subacute or chronic. Standard surgical management consists of resection and anastomosis. In

Crohn's disease strictureplasty may be considered in the presence of short multiple strictures without active sepsis.

Bolus obstruction

Bolus obstruction in the small bowel may be caused by food, gallstones, trichobezoar, phytobezoar, stercoliths and worms.

Gallstones

These tend to occur in the elderly secondary to erosion of a large gallstone through the gall bladder into the duodenum. Classically, there is impaction about 60 cm proximal to the ileocaecal valve. The patient may have recurrent attacks as the obstruction is frequently incomplete or relapsing due to a ball-valve effect. A radiograph will show evidence of small bowel obstruction with a diagnostic air—fluid level in the biliary tree. The stone may or may not be visible. At laparotomy it may be possible to crush the stone within the bowel lumen if it is soft, after milking it proximally. If not, the intestine is opened and the gallstone disimpacted, milked back and removed. If the gallstone is faceted a careful check for other enteric stones should be made. The region of the gall bladder should not be explored at this time but later on.

Food

Bolus obstruction may occur after partial or total gastrectomy when unchewed particles can pass directly into small bowel. Apple, coconut, brussels sprouts, dried fruit and orange pips are particularly liable to cause obstruction. The management is similar to a gallstone with intraluminal crushing usually being successful.

Trichobezoars and phytobezoar

These are firm masses of undigested hair balls and fruit/vegetable fibre, respectively. The former is due to persistent hair chewing and sucking, and may he associated with an underlying psychiatric abnormality. Phytobezoars are predisposed to by high fibre intake, inadequate chewing, previous gastric surgery, hypochlorhydria and loss of gastric pump mechanism.

Stercoliths

Usually found in the small bowel in association with a jejunal diverticulum or ileal stricture. Presentation and management are identical to gallstones.

Worms

Ascaris lumbricoides may cause low small bowel obstruction particularly in children, the institutionalized and those near the tropics. An attack frequently follows initiation of antihelminthic therapy. Debility is frequently out of proportion to that produced by the obstruction. If worms are not seen in stool or vomitus, the diagnosis may be indicated by eosinophilia or the sight of worms within gas-filled small bowel loops on a plain radiograph.

Treatment is surgical removal.