of cases. The leading symptoms are obstructive jaundice and rapid loss of weight; in 50 per cent. of cases the jaundice is painless. As in other varieties of obstructive jaundice, pruritus is a distressing feature. Frequently anorexia is present. Usually the liver is enlarged, and the gall-bladder is palpable in 75 per cent. of cases, but sometimes, as a result of recanalisation consequent upon ulceration of the growth, the distended gall-bladder, initially palpable, becomes impalpable. As a rule the disease is rapidly fatal, death occurring on an average fourteen weeks after the onset of symptoms. Hepatic failure with coma is often a terminal event; sometimes this is preceded by cholangitis, with a high temperature and rigors.

Treatment.—Early laparotomy affords the patient a slender chance of prolonged survival. In about 15 per cent. of cases the growth is found to be resectable. In a number of the remainder a palliative procedure such as choledochoduodenostomy is possible, and symptomatic relief, particularly of the jaundice, is afforded thereby.

Carcinoma of the Ampulla of Vater (see p. 475).

CHAPTER XXIII

THE PANCREAS

HAMILTON BAILEY

Embryology.—The pancreas is developed from two buds of that part of the foregut which becomes the duodenum, one bud passing into the layers of the ventral and the other into the dorsal mesentery. The ventral bud arises from the base of the diverticulum that forms the liver (fig. 616, A), and forms the head and the uncinate

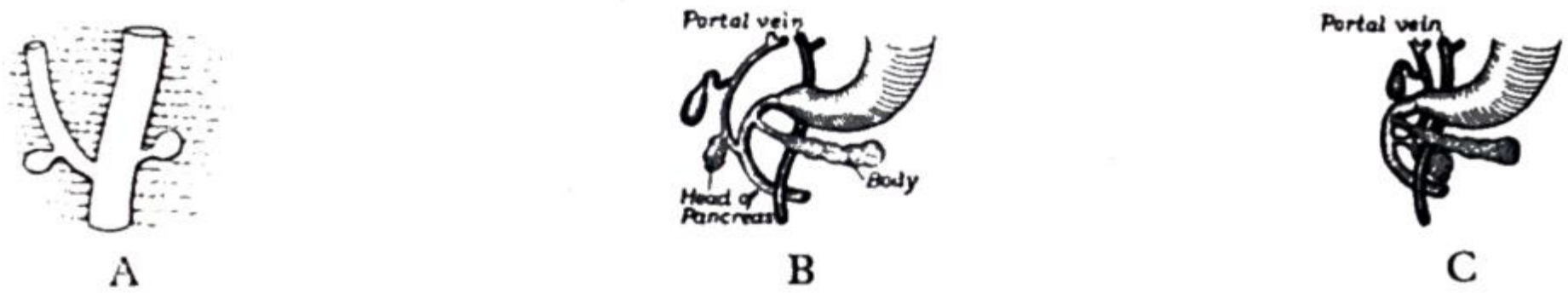

FIG. 616.—A, Primary pancreatic buds from the foregut ; B, Later stage before rotation ; C, After rotation, but before fusion : embyro fifth week.

process of the pancreas ; its duct becomes the duct of Wirsung. The remainder of the pancreas is formed from the dorsal bud, which arises nearer the pylorus than its fellow, its duct being the future duct of Santorini (fig. 616, B). With rotation of the stomach, the two pancreatic anlages close on the portal vein (or superior mesenteric vessels) like a book on a bookmark (fig. 616, C). Having come into contact, the two anlages fuse, and in so doing the duct of Wirsung becomes the main duct of both embryological components.

Surgical Anatomy.—Every student is familiar with the head, neck, body, and tail of the pancreas. It is necessary to recall that from the lower and left part of the head projects the uncinate process (fig. 617), upon which rest the superior mesenteric vessels. The posterior surface of the neck is grooved deeply by these vessels, and more superiorly by the portal vein. In spite of the intimate contact of the portal vein with the pancreas, unless it is bound down by inflammatory adhesions, in neoplastic infiltration it can be separated from the pancreas comparatively easily. In so far as its blood supply is concerned, the two embryological divisions of the pancreas retain their pristine independent nutrient vessels. Thus the head is supplied by the pancreatico-duodenal arteries while the body and the tail are nourished mainly by branches of the splenic artery. The distal part of the common bile duct lies in a deep sulcus between the second part of the duodenum and the head of the pancreas. To examine the pancreas properly by direct palpation, the organ must be mobilised by dividing the peritoneum (*a*) along the right margin of the duodenum and (*b*) along the lower border of the pancreas, the inferior pancreatico-duodenal artery being divided between ligatures (A. O. Whipple).

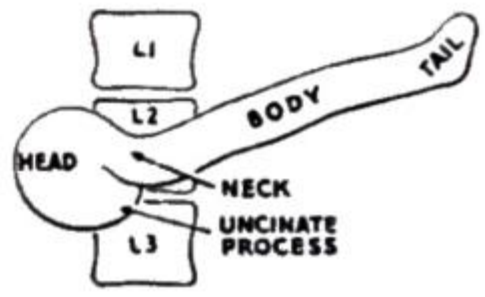

FIG. 617. — The various parts of the pancreas and its relationship to the lumbar vertebræ. (*After Rodney Smith.*)

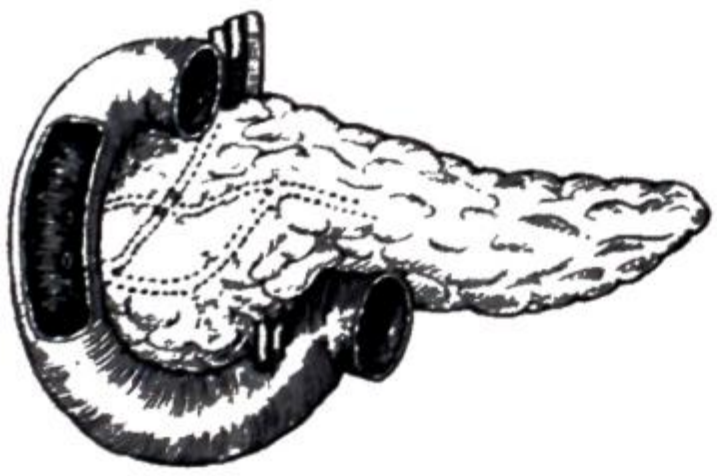

FIG. 618.—The most constant arrangement of the pancreatic ducts. (*After Rienhoff and Pickrell.*)

The ducts of the pancreas are subject to anatomical variations, but the most usual arrangement is shown in fig. 618 ; the duct of Santorini has a separate orifice about

Johann Wirsung, Prosector at Padua, was murdered when entering his house at night in 1643.
Giovanni Domenico Santorini, 1681–1737. Professor of Anatomy, Venice.
Allen Oldfather Whipple, Contemporary. Emeritus Professor of Surgery, Columbia University, New York City.

½ inch (1·25 cm.) above the duodenal papilla. This papilla is surrounded by Oddi's sphincter, spasm of which is believed to play a considerable rôle in the production of pancreatitis and post-cholecystectomy symptoms. It is well to know that in 10 per cent. of cases the duct of Santorini is the main secretory duct of the pancreas, in which event the bulk of the secretion of the gland is ejected into the duodenum through the superior papilla.

LABORATORY TESTS FOR INSUFFICIENCY OF PANCREATIC ENZYMES

The external secretion of the pancreas is regulated by both nervous and chemical stimuli. The vagus nerve induces a secretion of a viscid fluid, rich in enzymes. Secretin is formed by the action of HCl on the duodenal and jejunal mucous membrane. This hormone, having been liberated, is absorbed into the circulation and on reaching the pancreas provokes a thin, watery alkaline secretion, poor in enzymes but containing much inorganic material. In a normal adult 2¾ to 3½ pints (1,500 to 2,000 ml.) of pancreatic secretion are discharged into the duodenum every twenty-four hours.

The stools of a patient with pancreatic insufficiency are very bulky, pale, and offensive, and their weight, when dry, is much greater than normal.

Azotorrhœa.[1]—The patient should be given at least 100 G. (3½ oz.) of minced beef or ham for three days. The presence of undigested meat fibres in the stools is indicative of deficiency of trypsin. When positive, this is a reliable test.

Steatorrhœa.—The normal fat content of the fæces is about 20 per cent., but in chronic pancreatitis it may rise to between 50 and 90 per cent. Steatorrhœa is present only in advanced cases of pancreatic disease.

Increase of Pancreatic Enzymes in the Blood and Urine.—Obstruction to the pancreatic ducts is an important factor by which increase in concentration of serum amylase and serum lipase is brought about. In the absence of obstruction, or when the pancreas is largely destroyed by disease, these tests may be completely negative. They are :

Serum Amylase Concentration.—The determination of the serum amylase can be available within an hour after the necessary blood for the test has been taken. This test is considered in greater detail in connection with acute pancreatitis (see p. 464).

Serum lipase concentration cannot be used for emergency purposes, since the results cannot become available for twenty-four hours after withdrawing the blood.

The diastatic index of the urine may be increased (see p. 465).

CONGENITAL ABNORMALITIES

Annular Lobe of the Pancreas.—In this condition the second part of the duodenum is surrounded by a collar of pancreatic tissue of varying thickness containing its own duct that joins the duct of Wirsung. This annular lobe is probably caused by adherence of the right (ventral) pancreatic anlage to the duodenum during rotation. In most instances the accessory lobe is symptomless, and is found at necropsy. On rare occasions it gives rise to a varying degree of duodenal obstruction, in some cases amounting to almost complete stenosis with dilatation of the first part of the duodenum (fig. 619). Most of the patients with these symptoms are adults, but one-third are infants under one year of age. The diagnosis is confirmed by X-ray, when the characteristic duodenal 'double bubble' is present.

FIG. 619.—Annular lobe of the pancreas causing duodenal obstruction.

Treatment.—Sometimes excision of an annular pancreatic lobe has been followed by a persistent pancreatic fistula. Therefore, unless this lobe is comparatively thin and separates easily, the best treatment is to bring the first coil of jejunum through a hole made in the right-hand part of the mesocolon, and anastomose it to the dilated first part of the duodenum—duodenojejunostomy.

An ectopic pancreas is found in the submucosa of some part of the duodenum or small intestine (including Meckel's diverticulum), the gall-bladder, adjoining the pancreas (e.g. in the hilum of the spleen) or, most exceptionally, within the liver in

[1] Azotorrhœa = excessive quantity of nitrogen in the fæces.

Ruggiero Oddi, 1845–1906. Surgeon and Anatomist, Rome.
Johann Meckel, 1781–1833. Professor of Anatomy, Obstetrics and Botany, Berlin.

2 per cent. of carefully conducted necropsies. It is improbable that it would give rise to any symptoms were it not for the fact that it is liable to be the starting-point of an intussusception. Occasionally an ectopic pancreas is present in the wall of the stomach, and sometimes undergoes cystic degeneration. It gives rise to epigastric discomfort, and is usually diagnosed on radiographic findings as a benign neoplasm.

Congenital cystic disease of the pancreas sometimes accompanies congenital disease of the kidneys and liver.

Fibrocystic disease of the pancreas is but one manifestation of a congenital abnormality of mucous secretion that renders mucus exceedingly viscid. This hereditary abnormality has been named mucoviscidosis. Mucoviscidosis affects mucus-secreting glands, in particular those of the pancreas and bronchioles. Viscid mucus obstructs the pancreatic ducts, and retention of pancreatic secretions results ; some of the alveoli rupture, pancreatic enzymes escape and become activated by tissue juice (M. Bodian). Thus, from birth or before it, the infant suffers from pancreatitis with subsequent fibrosis. The secretion of abnormally viscid mucus into the bronchial tree is the cause of respiratory infection, which takes the form of bronchiolitis, and frequently progresses to bronchiectasis. In 25 per cent. of cases the liver shows focal biliary cirrhosis. The sweat glands produce sweat containing two or four times more NaCl than normal. Thus this remarkable disease affects not only mucus-secreting glands, but all exocrine glands.

Clinical Features :

The life of one afflicted with fibrocystic disease of the pancreas is fraught with dangers.

At Birth.—In 10 per cent. of cases the infant is born with meconium intestinal obstruction (see Chapter 26), or more rarely with meconium peritonitis (see p. 491).

During Infancy.—In most cases a few weeks or months after birth bronchiolitis develops. There is dyspnœa, with inspiratory indrawing of the lower chest and the suprasternal notch, and a distressing spasmodic cough. In a number of instances the chest becomes barrel-shaped, and a radiograph shows bronchiectasis. In 90 per cent. of cases steatorrhœa is present, and usually the stools are unduly frequent, greasy, and their smell obnoxious. In the absence of severe respiratory distress the appetite is voracious, in spite of which the infant is marasmic, except for a distended abdomen.

In Older Children.—Steatorrhœa with wasting (fig. 620) continues, and is distinguished from cœliac disease by the early onset, the excellent appetite, the almost constant accompaniment of attacks of respiratory infection, and, possibly by a history of death in infancy from intestinal obstruction or pneumonia of siblings.[1] In hot weather excessive loss of electrolytes in sweat sometimes results in severe dehydration and even death. Cirrhosis of the liver and hypertension frequently develop ; indeed one-third of all cases of portal hypertension at this time of life occur in subjects with this disease.

If the patient reaches late childhood without gross pulmonary or hepatic damage, improvement is remarkable and often sustained.

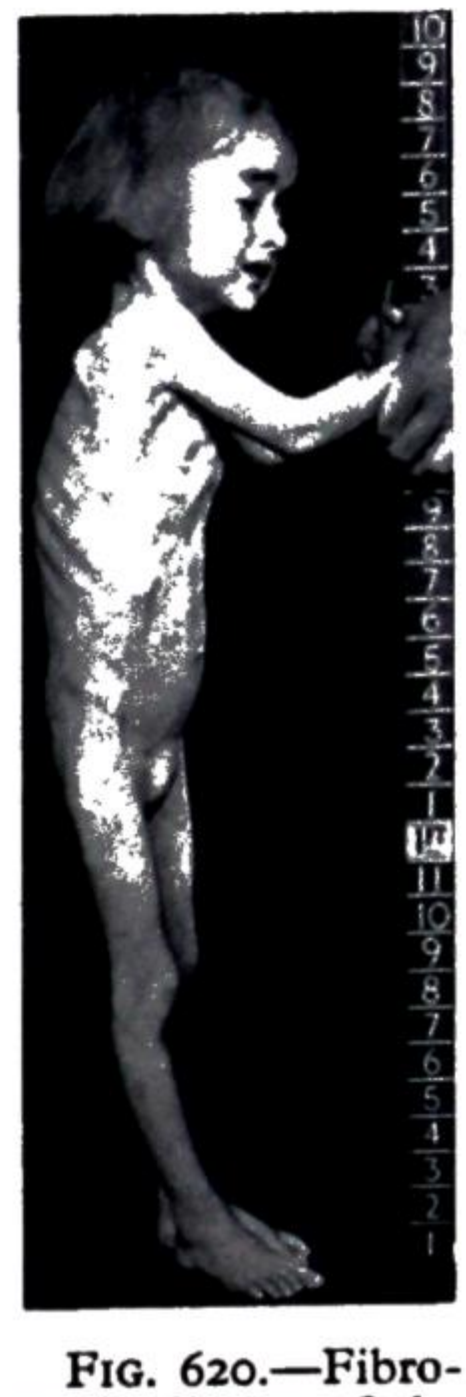

FIG. 620.—Fibrocystic disease of the pancreas in a child of two years of age. (*Dr. A. P. Norman, London.*)

Confirmatory Tests :

1. The amount of trypsin in the duodenal content is very low or absent.

2. *Sweat Tests:* (*a*) *The Full Sweat Test.*—If sweat is collected by encasing the patient in a polythene bag, no less than 99 per cent. of patients with fibrocystic disease of the pancreas show the abnormal sweat content described above.

(*b*) *The Finger-Print Sweat Test.*—A finger-print is taken on an agar plate. The agar is impregnated with silver nitrate and potassium chromate. In fibrocystic disease of the pancreas the excessive NaCl in the sweat bleaches the suspended chromate in under twenty minutes.

Treatment should aim at controlling respiratory infection, which is nearly always

[1] Siblings = offspring of the same parents.

Martin Bodian, Contemporary. Morbid Anatomist, Hospital for Sick Children, London.

staphylococcal. The antibiotic selected will depend on the laboratory tests of sensitivity of the infecting organism. A major problem is development of resistance by the organisms to one antibiotic after another.

Much can be done for fibrocystic disease of the pancreas by symptomatic treatment. Extra proteins should be given. The sugar intake should be high, but that of starch must be moderate. Fat is curtailed drastically, whereby the frequency, bulk, and foulness of the stools are diminished. Provided this diet is adhered to and enteric-coated capsules of pancreatin, 5 to 10 G. are taken before feeds and 200 units of vitamin D and 10,000 units of vitamin A are given daily, there is nearly always a substantial gain in weight.

Lastly, there should be liberal salting of food. In hot weather extra salt, 2 G. per day, should be supplied. The dietetic régime must be followed throughout life.

INJURIES

On account of its deeply placed, protected position, injuries to the pancreas are uncommon. Blows on the abdomen and traffic accidents are the usual cause; these injuries can also occur from under-water blast. In many instances when the pancreas is damaged, other organs, particularly the spleen and the duodenum, are involved simultaneously. That the pancreas has been severely damaged can be suspected pre-operatively only by a high serum amylase estimation.

Type 1.—The patient, who has had a severe injury to the upper abdomen, presents signs of a serious lesion of some intra-abdominal organ, and it is thought wise to explore. When the pancreas is the injured organ, there is often blood-stained fluid in the lesser sac, and fat necroses are sometimes present.

Type 2 (*Milder Injuries*).—The first intimation that the pancreas has been lacerated is often the development of a pseudo-pancreatic cyst (see p. 471).

Treatment (*Type* 1 *Injury*).—If the tail of the pancreas is completely or nearly severed, it should be removed, the duct closed by two very fine absorbable sutures, and the raw surface covered by a free omental graft. When the head or body is deeply lacerated it is best to approximate the two fragments with a single stitch, which is not tied tightly (multiple stitches cause necrosis of the pancreas). The lesser sac is drained, and the abdominal wall closed with non-absorbable sutures. Propantheline bromide (Probanthine, G. D. Searle and Co.) is given by mouth in doses commencing at 15 mg. daily, working up to 60 mg. daily by the fifth day, and stopping the drug on the tenth day. Unless the total fluid intake is limited to about 1,500 ml. daily, propantheline bromide cannot be expected to diminish the flow of pancreatic juice, which is so important in expediting healing of the pancreas.

The commonest complication of a non-fatal pancreatic injury is the subsequent development of a pancreatic fistula.

Injury during subtotal gastrectomy for duodenal ulcer is not uncommon. During the dissection of the first part of the duodenum from the head of the pancreas, the latter is bruised and possibly the duct of Santorini is severed inadvertently.

INFLAMMATIONS

Pancreatitis
- Acute
  - Ultra acute = hæmorrhagic (*syn.* acute necrosis of the pancreas).
  - Œdematous.
- Subacute.
- Chronic
  - Relapsing.
  - Unremitting.

ACUTE PANCREATITIS

Pathology.—The following outstanding features are seen at necropsy. The great omentum and subperitoneal fat are studded with opaque areas termed fat necroses (fig. 621). If these are dabbed with a solution of cupric

acetate, they will be seen to stain an intense blue (Benda's test).

Fat necroses are not always limited to the abdominal cavity. They can sometimes be demonstrated beneath the pleura and pericardium, and even in the subsynovial fat of the knee joint.

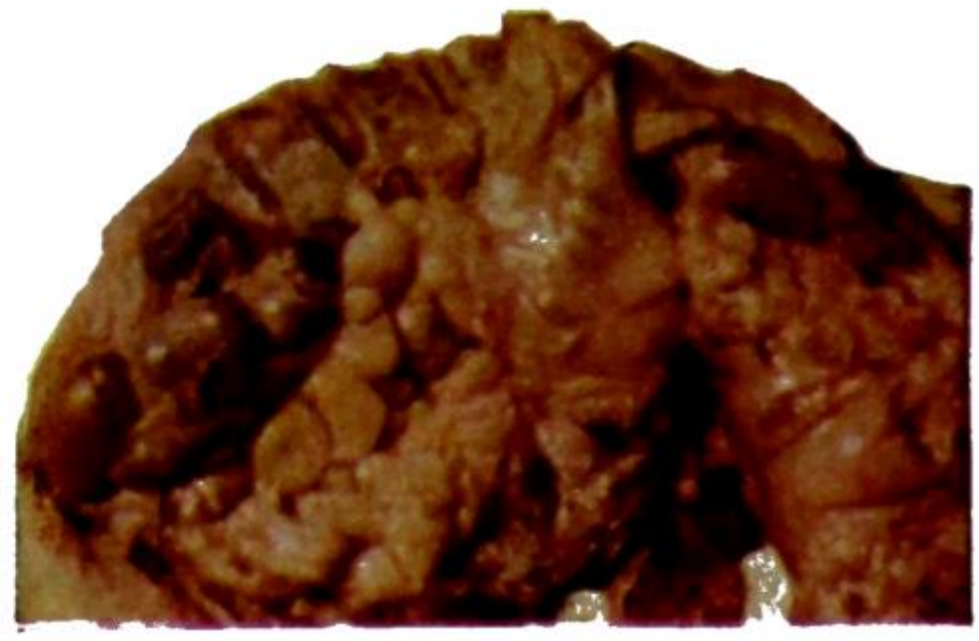

FIG. 621.—Necropsy on an obese subject. Death from acute hæmorrhagic pancreatitis. The colour photograph shows the hæmorrhage into the lesser sac in the upper part of the picture, and fat necroses on the great omentum. These can be seen particularly in the centre of the lower part of the picture.

The peritoneal cavity, especially the lesser sac, contains a blood-stained exudate which, in very acute cases, appears to be almost pure blood. The retroperitoneal tissues in the vicinity of the pancreas are infiltrated with blood-stained fluid, or, more rarely, with pure blood, giving the appearance of a retroperitoneal hæmatoma. The pancreas is swollen, and in some cases all or part of it is necrotic. Not infrequently the adrenals are found to be surrounded by inflamed, semi-necrotic tissue. Sometimes the gall-bladder is thin-walled and normal in appearance ; more often it is thickened and fibrotic from previous chronic cholecystitis. In over 50 per cent. of cases the gall-bladder contains stones. The gall-stones are nearly always small, and occasionally one or more stones are found in the common bile duct. On culture the bile may be found to be infected, most often with *Esch. coli* or *streptococci*.

When the lesser sac has been drained and the patient survives, sometimes portions of necrotic pancreatic and peri-pancreatic tissue are discharged through the wound as putty-like material. On analysis this material has been shown to be composed largely of calcium stearate.

Ætiology.—The cause of acute pancreatitis has not been elucidated fully. On rare occasions the ampulla of Vater has been found blocked by a gall-stone or, more rarely still, by a roundworm. Somewhat less infrequently a strictured orifice of the duct of Wirsung has been demonstrated. In a high percentage of cases metaplasia of the epithelial lining of the main duct is present—sufficient to obstruct its lumen at one or more points (Rich and Duff). If the outflow of pancreatic juice is obstructed during the height of secretory activity, the pressure of pent-up secretion may be sufficient to rupture some of the acini. Thus, it is postulated, trypsinogen escapes into the pancreatic tissues, becomes activated by tissue juices and causes autodigestion of the pancreas. The liberated pancreatic enzymes include steapsin (lipase) which splits fats into glycerol and fatty acids. Free fatty acids combine with calcium to form soaps = fat necrosis.

Accessory factors:

Alcoholism is fairly often a precursor, usually in male patients.

Previous Cholecystectomy.—Probably some degree of pancreatitis was present before the removal of the gall-bladder. Should there be spasm or stenosis of the ampulla of Vater and a common orifice of the bile and pancreatic ducts is present, the absence of the gall-bladder invites reflux of bile, possibly infected, along the duct of Wirsung.

Trauma of the pancreas during partial gastrectomy, or other operations entailing considerable handling of the pancreas, is an infrequent but an established cause of acute pancreatitis (see p. 345).

Associated with Mumps.—It is probable that a few cases of acute pancreatitis are

Carl Benda, 1857–1933. Professor of Anatomy, Berlin.
Arnold Rice Rich, Contemporary. Professor of Pathology, Johns Hopkins University, Baltimore, U.S.A.
George Lyman Duff, Contemporary. Professor of Pathology, McGill University, Montreal.

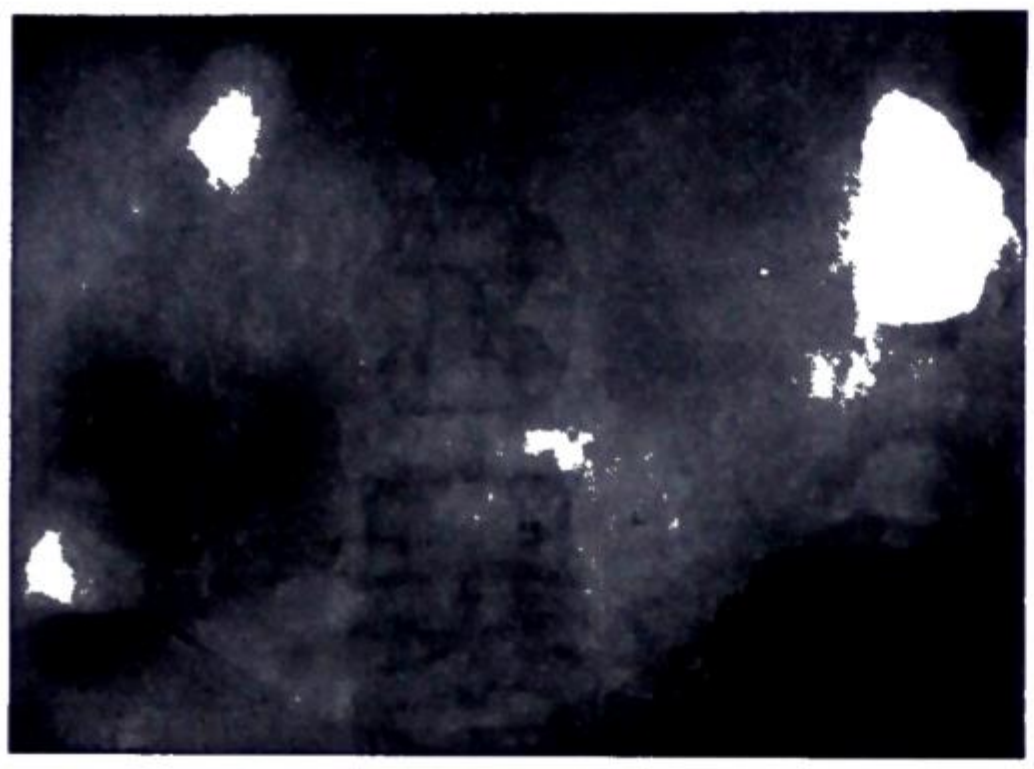

FIG. 622.—Acute pancreatitis. Radiograph two hours after onset, showing gas in the duodenum and a little in the stomach. (*S. Olivier, 'Radio-Diagnostic des Occlusions Intestinales Aiguës,' Paris.*)

Diagnostic Aspiration.—In cases where there is shifting dullness and it is imperative to know the nature of the fluid, diagnostic aspiration, employing a fine needle attached to a syringe, is permissible. In acute pancreatitis the fluid is typically of a prune-juice shade and its serum amylase content is high.

Delayed Treatment.[1]—When the diagnosis of acute pancreatitis can be made with reasonable assurance, the highest aim of the surgeon should be to assist probable resolution by non-operative measures.

Analgesics.—It should be remembered that morphine often increases the pain of acute pancreatitis; the agonising pain can usually be relieved by pethidine, 2 ml. (100 mg.) intravenously, which can be repeated in one hour if necessary.

When the pain is very severe, it is best relieved by giving propantheline bromide (pro-banthine), 30 to 45 mg. six-hourly, via the indwelling gastric tube, or 15 mg. intramuscularly. If required, this drug, which reduces pancreatic secretion and lowers the concentration of enzymes in the juice, can be continued for several days. Much the same effect, though less selectively, can be obtained by atropine.

Transnasal gastric aspiration is of great service; in addition to preventing vomiting and retching, aspiration of gastric juice, as it is secreted, inhibits the normal hormonal stimulation of the pancreas. Thus physiological rest is afforded to the pancreas as well as to the stomach.

Fluid Therapy.—A continuous intravenous drip of isotonic dextrose-saline is administered. The keeping of an accurate fluid balance-sheet is essential. A daily urinary chloride estimation is imperative to guard against hypochloræmia. In no intra-abdominal catastrophe is the replacement of lost water and electrolytes more necessary than in acute pancreatitis.

Correction of Hypotension.—In severe cases the associated peripheral circulatory hypotension can often be rectified by infusion of plasma which, in addition to combating shock, is alleged to contain sufficient anti-tryptic activity to neutralise some of the released trypsin. When, in spite of the administration of plasma followed by blood transfusion, hypotension persists, the intravenous administration of noradrenaline has proved valuable.

Antibiotic Therapy.—As the leading lethal factors are secondary infection of the acutely inflamed pancreas, fat necroses, and the peritoneal exudate, antibiotic treatment is indicated. Aureomycin (or terramycin) is the anti-

[1] With delayed treatment the mortality of acute pancreatitis has decreased from 60 per cent. in the 1920s, when immediate operation was the standard treatment, to 10 per cent. or under.

biotic of choice because it is concentrated in the bile, and on account of the accompanying ileus it must be administered parenterally.

Correction of Hypocalcæmia.—When fat necrosis is extensive there is concomitant hypocalcæmia that calls for calcium gluconate intravenously; 10 ml. of a 10 per cent. solution is given six-hourly until the blood calcium reaches a normal level.

Hydrocortisone in Desperate Cases.—In patients with ultra-acute hæmorrhagic pancreatitis who seem likely to die, hydrocortisone, 20 to 50 mg. intramuscularly, in some cases has brought about a remarkable change for the better, in which case it should be followed by prednisone, 25 mg. daily, in divided doses by mouth for five days, after which the dose is diminished slowly. It may well be that the beneficial results are due to the drug's supportive action during a period of temporary adrenal failure (see Pathology, p. 463). This form of treatment must only be employed in desperate cases.

Early exploratory operation should be avoided unless it is impossible to rule out some other condition requiring urgent laparotomy. When, for this reason, it is imperative to open the abdomen, after preliminary medication with pethidine, a short midline incision is made under local infiltration anæsthesia. If the case is one of acute pancreatitis, blood-stained peritoneal fluid will be observed and tell-tale areas of fat necrosis will be seen, in which event, after aspirating as much peritoneal exudate as possible, the incision is closed with through-and-through non-absorbable sutures. Should some other intra-abdominal catastrophe be revealed, the patient must be anæsthetised more fully, and the incision extended as necessary.

Intermediate operation is sometimes required about the fourth to the sixth day in order to drain a collection of fluid in the lesser sac; again local anæsthesia is advised, and nothing more than opening and draining the lesser sac (fig. 623) should be attempted at this stage. Sump drainage is an excellent prophylactic against digestion of the skin by escaping pancreatic ferments. Abdominal corsets should be applied to guard against wound disruption. Another reason for an intermediate operation is the development of a localised abscess. Œdema and discoloration of the lumbar region are the most usual presenting signs. The abscess should be drained posteriorly (fig. 623).

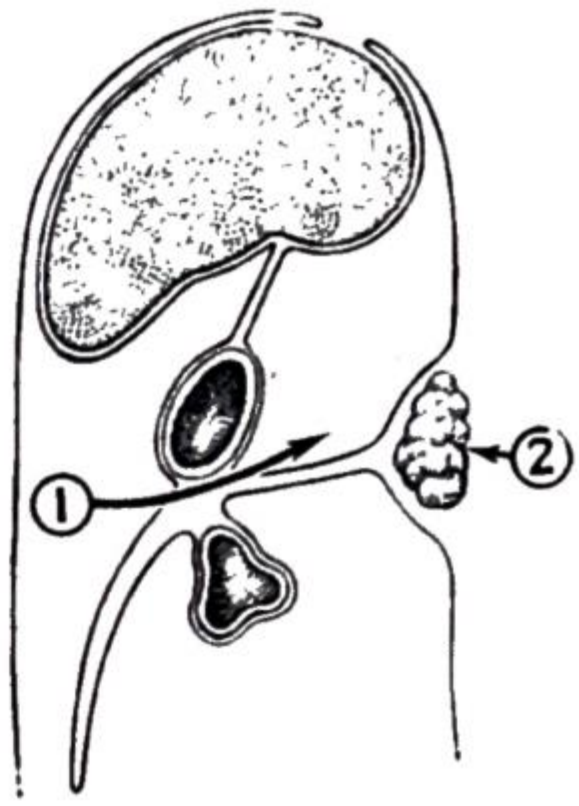

FIG. 623.—1. The approach to the pancreas between the stomach and the transverse colon. 2. Retroperitoneal route for draining a pancreatic abscess.

Laparotomy after Acute Pancreatitis has Subsided.—Acute pancreatitis is notoriously recurrent. Often attack follows attack in a crescendo of severity. With the object of preventing future attacks, operation is undertaken in a quiescent period before the patient leaves hospital.

On opening the abdomen it is likely that some evidence of fat necrosis will still be present, and the pancreas will feel enlarged. Attention is directed to the gall-bladder. The course to be followed will vary according to whether

the gall-bladder is thin-walled and comparatively normal, or thickened and fibrotic. Undoubtedly the operation of choice for an obese, poor-risk patient is to proceed at once to perform cholecystjejunostomy, with the objective of by-passing the bile. In other circumstances cholangiography is helpful in determining whether obstruction to the ampulla of Vater is present. In any case, when the gall-bladder is small, fibrotic, and contains calculi, cholecystectomy is indicated. Nevertheless, unless the patient is fit enough to undergo sphincterotomy in addition, it is unlikely that cholecystectomy *per se* will prevent further attacks of acute pancreatitis.

Sphincterotomy.—Division of the sphincter of Oddi (fig. 624) is a well-tried method of treating pancreatitis, both acute and chronic ; in the former instance it is undertaken only in the quiescent stage. The operation is not easy because rugæ tend to hide the papilla and the mucous membrane in the vicinity of the duodenal papilla does not hold traction sutures well : should these sutures cut out the field becomes obscured by blood. A sphincterotome is sometimes employed, but blind sphincterotomy has possible dangers and disadvantages. A satisfactory method is to open the supra-duodenal portion of the common bile duct, insert a Moynihan's flexible probe towards the ampulla, and then to make a small incision into the second part of the duodenum. The sphincter can now be divided for 8 to 10 mm. under vision, using the malleable probe as a guide. The object of sphincterotomy is to provide free drainage to the duct of Wirsung and the common bile duct. After sphincterotomy the duodenum is closed in two layers (the outer being of unabsorbable suture material) care being taken not to narrow the lumen of the duodenum unduly. The choledochostomy opening is closed around a suitably sized T-tube. Sphincterotomy destroys the ability of the gall-bladder to fill normally. The gall-bladder, whether diseased or not, must therefore be removed. A theoretical objection to forfeiting the sphincter is ascending cholangitis which, fortunately, seldom occurs.

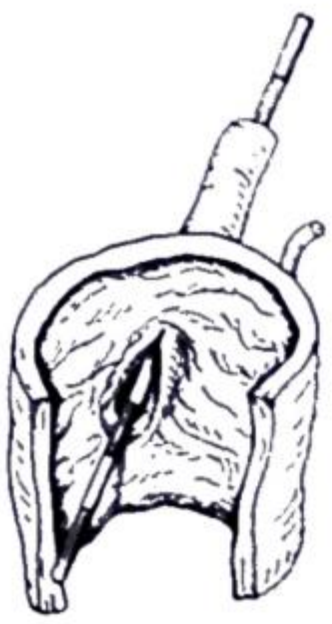

FIG. 624.—Division of the sphincter of Oddi (sphincterotomy). (*After P. F. Partington.*)

Subacute pancreatitis gives rise to symptoms similar to those of acute cholecystitis ; indeed, in many instances the diagnosis is not made until laparotomy has been performed after the attack has subsided. However, a serum amylase test, repeated if negative, in every case of acute cholecystitis reduces the number of cases that escape earlier detection.

CHRONIC RELAPSING PANCREATITIS

Pathology.—Early in the disease the gland is somewhat enlarged, pale, and firm. Later the organ, notably its head, becomes more enlarged, more indurated, and adherent to surrounding structures, particularly the stomach. Frequently small areas of fat necrosis can be observed upon the surface of the pancreas. The duct of Wirsung is often dilated, and may contain calculi. In advanced cases there is replacement of the pancreatic acini and, to a much lesser extent, of the islets of Langerhans, by fibro-fatty tissue that sometimes becomes calcified. The cut surface of the gland does not bleed as readily as normal pancreatic tissue. Pressure on the superior mesenteric and the portal vein sometimes results in portal hypertension.

Clinical Features.—The disease tends to become progressively worse and is characterised by :

Lord Moynihan, 1865–1936. Professor of Surgery, University of Leeds.
Paul Langerhans, 1847–1888. Professor of Pathology, Freiburg.

Pain coming on in attacks that last three or more days (cf. biliary colic, the attacks of which last from minutes up to half an hour). The pain is epigastric, central, and passes to the back—a radiation which accounts for it being so frequently confused with biliary colic.

Vomiting is usually oft repeated, and commences some time after the pain ; the contents of the duodenum, as well as of the stomach, are ejected.

Stools.—More than one-quarter of the patients with chronic relapsing pancreatitis develop steatorrhœa, but this is a late manifestation of the disease. In suspected cases of chronic pancreatitis stools should be examined macroscopically for fat. A severe degree of pancreatic insufficiency causes diarrhœa, characterised by malodorous and voluminous stools. The existence of pancreatic insufficiency is practically certain if the patient passes quantities of fat or oil that separates from the non-fatty portion of the fæcal matter.

Remissions of several months are usual.

Weight.—Loss of weight is sometimes alarming, but nearly as often an obese patient remains overweight.

Jaundice is somewhat exceptional ; in several large series of cases it has been present in under 15 per cent. of cases. Observing that gall-stones, if present, are nearly always confined to the gall-bladder, it would seem that mild jaundice is hepatogenous in origin (R. B. Cattell), although undoubtedly in the more obvious cases it is the result of pressure upon the terminal part of the common bile duct.

Arteriosclerosis and chronic relapsing pancreatitis often go hand in hand (E. G. Saint).

Alcoholism or drug addiction are so often present (40 to 50 per cent., that either is to be regarded as an important diagnostic datum. The habit is acquired in the endeavour to seek relief from the intolerable pain.

Previous abdominal operation is very common. Appendicectomy may have been performed for the condition and it does no harm, but the same cannot be said for cholecystectomy, for in many cases the loss of the gall-bladder seems to increase the pain.

There are no pathognomonic physical signs of this disease.

Radiography.—On no account should a barium meal be commenced without a preliminary film, otherwise pancreatic calculi or calcareous plaques in the pancreas, which are not very unusual, are likely to be missed. Very occasionally the enlargement of the head of the pancreas is sufficient to increase the breadth of the U of the duodenum.

Tests for Pancreatic Insufficiency (see p. 460).—At least one of these tests is nearly always positive, at any rate during the attack.

Treatment is difficult. Medical treatment is usually unavailing, and surgical treatment often unsatisfactory ; consequently there is a large choice of dissimilar procedures, any of which afford relief in some cases, and fail in others. That is why more than one operation is often required. Each of the following measures has a place in the treatment of established chronic pancreatitis. By pure chance the first operation may be the one to give relief, and possibly arrest the disease.

Richard Bartley Cattell, Contemporary. Surgeon, Lahey Clinic, Boston, Mass.
Eric Galton Saint, Contemporary. Professor of Medicine, University of West Australia, Perth.

FIG. 625. — Cholecystjejunostomy-en-Y (Roux).

Cholecystjejunostomy-en-Y (Roux) (fig. 625) is the oldest, and for a patient in poor condition is inferior to none, provided the gall-bladder is even moderately healthy.

Cholecystectomy is necessary when the gall-bladder is grossly diseased, but it should be combined with sphincterotomy (see p. 468), otherwise the symptoms often return.

Retrograde pancreatojejunostomy is indicated especially in the presence of pancreatic calculi. The tail of the pancreas is amputated and the stones are removed through the open duct. If the duct is dilated, and an injection of diodone down the duct, followed by radiography, reveals obstruction to the egress of dye into the duodenum, much can be expected from the performance of retrograde pancreatojejunostomy (see fig. 627). Should no obstruction be present the cut surface can be converted into a V, and closed.

Splanchnicectomy (fig. 626).—The right great splanchnic nerve supplies the bile ducts and the left sends branches to the pancreas. Resecting the latter nerve effects a vasomotor change that breaks the vicious reflex circle responsible for the attacks (P. Mallet-Guy); a posterior subdiaphragmatic approach can be used, the twelfth rib being resected if more room is needed. About 3 cm. of the great splanchnic nerve is removed between the splanchnic ganglion and the diaphragm.

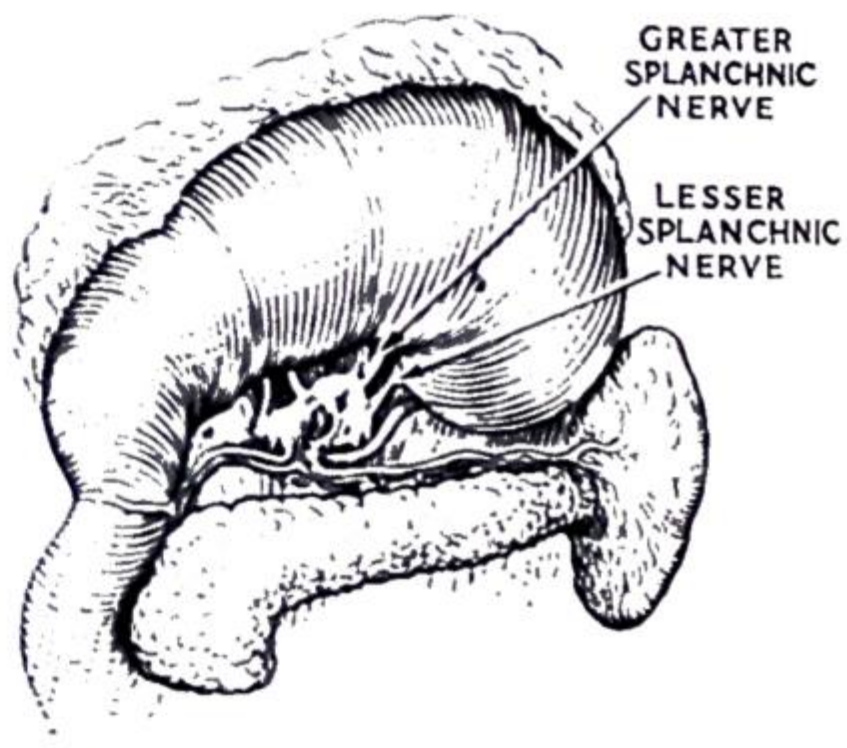

FIG. 626. — The splanchnic nerves and the splanchnic ganglia. (*After C. P. Puestow.*)

Subtotal gastrectomy and vagotomy is sometimes undertaken for pancreatitis by those who believe that if the secretive mechanism is interrupted the pancreas, no longer stimulated, is afforded rest.

When the Lesion is situated in the Head of the Pancreas.—If biopsy and frozen section is considered advisable, it should be performed in the manner described on p. 478. When no decision can be arrived at as to whether an indurated lesion in the head of the pancreas is a patch of chronic pancreatitis or a carcinoma, pancreatoduodenectomy may be considered to be the best course. On the other hand, most surgeons of experience have encountered occasional cases where a patient, often in poor condition and deeply jaundiced, is considered to have a carcinoma of the head of the pancreas. Accordingly, palliative cholecystjejunostomy was performed, and to everyone's astonishment the patient has not only survived for many years, but has remained in good health. Obviously the patient must have been suffering from chronic pancreatitis.

Unremitting chronic pancreatitis is less common than the foregoing. The pain lasts for weeks, months, or even years. It demoralises the patient by its continuity rather than its severity. Probably the incessant pain is due to pressure of interacinous fibrosis on the terminal nerve filaments. Splanchnicectomy combined with cholecystjejunostomy are the measures most likely to give relief. When these measures fail, and the pain becomes unbearable, the advisability of performing chordotomy will have to be considered.

PANCREATIC CALCULI

Pancreatic calculi, which must be distinguished from calcareous plaques in the pancreas, occur infrequently. The symptoms are those of chronic pancreatitis, unless such a stone becomes lodged in the ampulla of Vater, when the symptoms are identical with a gall-stone in that situation (see p. 448). Pancreatic calculi are composed mainly of calcium phosphate and carbonate, and consequently can be demonstrated clearly by radiography.

César Roux, 1857–1934. Professor of Surgery, University of Lausanne, Switzerland.
Pierre Mallet-Guy, Contemporary. Professor of Surgery, Faculté de Médicine, Lyons.

From the point of view of treatment, pancreatic calculi can be divided into two varieties :

1. A stone, or stones, is located in the duct of Wirsung.

2. The gland is peppered with small calculi situated in the alveoli as well as the ducts. Such stones should be looked upon as a consequence, rather than the cause, of chronic pancreatitis.

Treatment.—Type 1 can usually be located by palpation of the mobilised pancreas, and removed by an incision into the duct through the substance of the pancreas, or by slitting up Oddi's sphincter transduodenally, according to circumstances. Drainage to the site of operation must never be omitted.

Type 2 would prove a Sisyphean[1] task by the operation just described, and the only method of dealing with them is to resect the body and tail of the pancreas and to remove as many stones as possible from the head of the organ through the transected duct. To allow exit for any stones that remain, or form subsequently, the operation is concluded by performing pancreatojejunostomy (fig. 627).

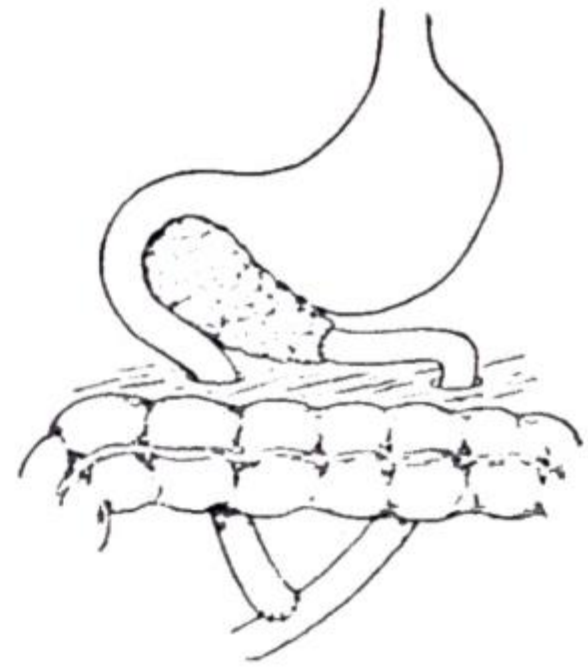

FIG. 627.—Retrograde pancreatojejunostomy. (*After M. K. Du Val Jnr.*)

PANCREATIC CYSTS { Pseudo-cyst. 60%. / True. 40%.

Pseudo-cyst of the Pancreas.—The collection of fluid in the lesser sac (fig. 628) is the result of an injury of the pancreas, or it follows acute pancreatitis ; very occasionally it is due to a minute perforation of a peptic ulcer situated on the posterior wall of the stomach. The condition commences as peritonitis limited to the lesser sac, the foramen of Winslow becoming occluded in the process, thus shutting off the general peritoneal cavity.

Clinical Features.—In traumatic cases abdominal pain, frequent vomiting, and epigastric tenderness are in evidence after the accident. In other cases the swelling develops during the conservative treatment of acute pancreatitis. Exceptionally the patient is admitted with a pseudo-cyst of the pancreas, and gives a history of an attack

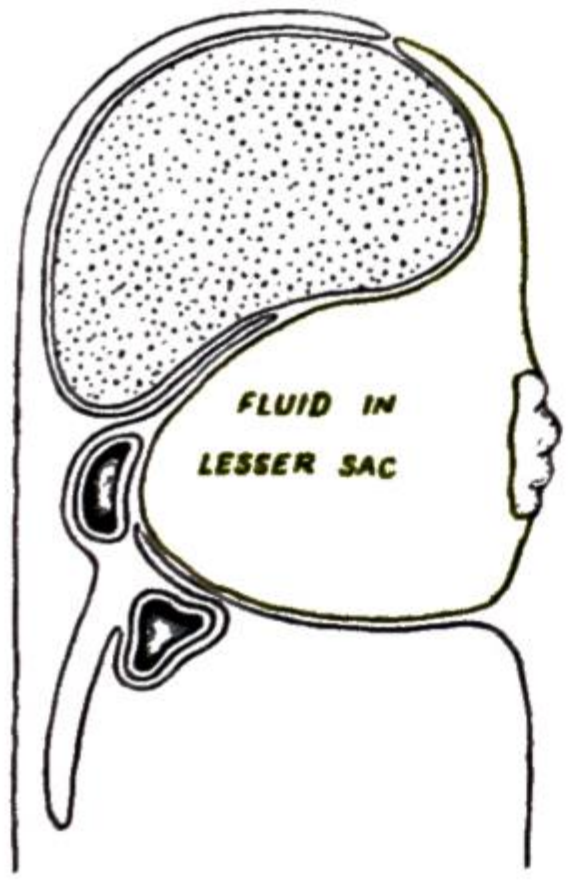

FIG. 628.—Pseudo-pancreatic cyst.

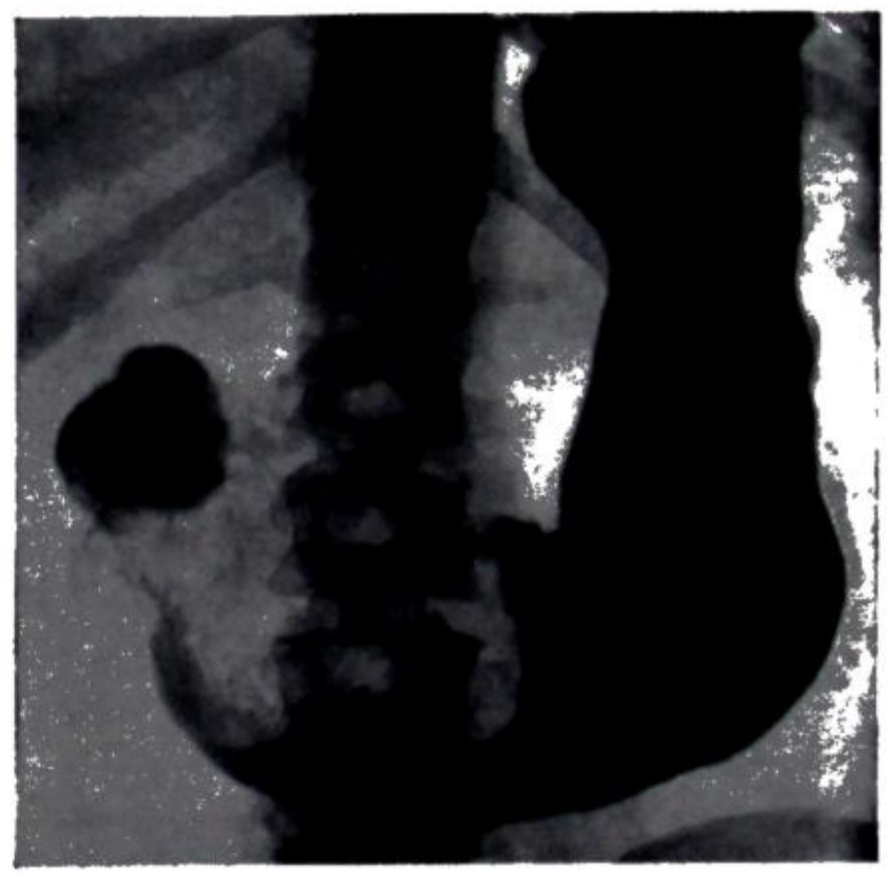

FIG. 629.—Pseudo-pancreatic cyst causing gastric deformity.

of severe abdominal pain occurring a week or more previously. On examination there is a swelling, sometimes a very large swelling, placed centrally above the umbilicus : the swelling is fixed, and in many instances it is so tense that fluctuation cannot be elicited. As a rule, transmitted pulsation from the abdominal aorta is very noticeable, but the pulsation becomes less pronounced or ceases when the patient is

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[1] Sisyphus, a King of Corinth who was condemned to roll to the top of a hill a huge stone that constantly rolled down again, making his task incessant.

Jacob Winslow, 1669–1760. A Dane who migrated to Paris, and there established a School of Anatomy.

examined in the knee-elbow position. In this way the possibility of the swelling being an aneurism of the abdominal aorta is eliminated. Its fixity distinguishes it from a mesenteric cyst, and the fact that it rarely, if ever, invades the pelvis usually rules out an ovarian cyst. A barium meal shows displacement of hollow viscera (fig. 629).

FIG. 630.—Transgastric anastomosis between the stomach and the cyst. (*After B. Szabo.*)

Treatment.—As the disadvantages of external drainage are (*a*) enzymic excoriation of the abdominal wall, (*b*) the pancreatic fistula continues to discharge, or (*c*) healing is followed by another pseudocyst, which has to be reopened; modern practice is veering towards internal drainage. A large opening into the cyst is made through the posterior wall of the stomach by the transgastric route (Jurasz's operation). The contents of the cyst then drain into the stomach. Convalescence is greatly shortened thereby, and the complications referred to are obviated.

True Pancreatic Cyst (fig. 631).—In 40 per cent. of cases a cyst of the pancreas is a true cyst, and falls into one of the following categories :

(*a*) *Retention cysts*, due to impaction of pancreatic calculi or fibrosis around the duct of Wirsung.

(*b*) *Cyst-adenomata.*—Most true cysts belong to this group. Occasionally the cyst wall undergoes a carcinomatous change.

(*c*) *Fibrocystic disease of the pancreas* (see p. 461).

(*d*) *Congenital Cystic Disease.*—Very rare indeed.

(*e*) *Hydatid Cyst.*—The ubiquitous hydatid completes the list.

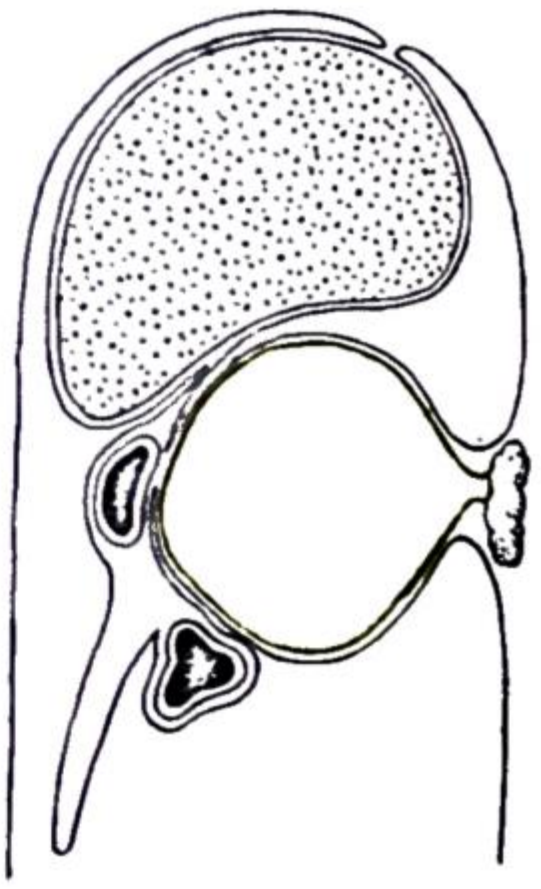

FIG. 631.—True pancreatic cyst.

The cyst commences in the body or, less frequently, in the tail of the pancreas ; only occasionally is the head the seat of origin. The cyst, which is filled with watery brown fluid or mucoid material, is generally unilocular. It is lined by columnar or cubical epithelium, which tends to degenerate, consequently the wall consists mainly, if not entirely, of fibrous tissue. Most often the swelling comes to the surface between the stomach and the colon. Sometimes it protrudes between the stomach and the liver.

Clinical Features.—Epigastric discomfort and the presence of a swelling are the usual reasons that cause the patient to seek advice. When the swelling arises in the tail of the pancreas it may be impossible to differentiate it from a hydronephrosis until pyelography has been performed. In those exceptional cases where the cyst arises in the head of the pancreas, the symptoms are identical with those of a choledochus cyst (see p. 434).

Treatment.—In exceptional circumstances a comparatively small cyst can be removed by dissection. The Jurasz's operation (*vide supra*) is often applicable to a cyst in contact with the posterior wall of the stomach. The cut edges of the cyst wall and the stomach can be united readily. In other circumstances a large cyst can often be anastomosed to the greater curvature of the stomach. When the cyst (or cysts) is situated towards the tail of the pancreas (this is the site of election for cystadenoma) the affected portion of the pancreas can be amputated.

PANCREATIC FISTULA

The quantity and composition of the discharge varies very considerably. As to quantity, in many cases the amount is only a few ounces daily; in others it is as much as 2 litres in the twenty-four hours. Such a loss of fluid and electrolytes, unless counterbalanced, is a menace to life. As a rule the fluid is charged with active ferments, and full precautions must be taken to prevent auto-digestion of the abdominal wall.

Treatment.—When the leakage is watery and profuse, the juice should be removed by continuous suction through a catheter attached to an electric pump, and

Antoni Aleksander Tomasz Jurasz, Contemporary. Professor and Director of the Surgical Clinic, Poznan, Poland.

methods to protect the skin of the abdominal wall by applying aluminium skin paint or a silicone barrier cream, are instituted. Returning the fluid aspirated from the fistula to the patient via a transnasal intragastric tube should be tried. If this method causes nausea and vomiting, fluid balance must be maintained parenterally. Plasma infusion or blood transfusion may be necessary. In most cases there is a gradual lessening of the amount of leakage. Among the methods recommended for hastening closure is complete alimentary rest, nutrition being maintained parenterally with intravenous dextrose-saline and protein hydrolysate for as long as seventeen days. The administration of pro-banthine helps to reduce the amount of pancreatic secretion.

Although it is sometimes protracted (several months), conservative treatment is successful in 75 per cent. of cases. When conservative treatment fails, transduodenal sphincterotomy, by draining more thoroughly the duct of Wirsung, frequently results in closure of a fistula without disturbing the fistulous track.

In a few instances a long-standing fistula with a fibrous wall has been cured by dissecting it, together with a margin of skin from the abdominal wall past the level of the peritoneum, and implanting the mouth of the fistula into a loop of jejunum.

ISLET-CELL TUMOURS OF THE PANCREAS

- Insulin producing = Insulinoma.
- Non-insulin producing
  - Innocuous, unless its very size causes symptoms.
  - Engendering persistent peptic ulceration.

Because the islets of Langerhans are aggregated mainly in the tail and body of the pancreas, when it occurs, an islet-cell tumour is located in the caudal part much more often than in the head of the gland. Regardless as to whether the tumour secretes hormones in sufficient quantities to cause symptoms, or is functionless, its histological structure closely resembles that of the islets themselves.

Insulinoma :

Pathology.—The severity of the symptoms is unrelated to the degree of hyperinsulinism or the size of the tumour. As a rule the tumour is 1 to 2 cm. in diameter ; rarely does it attain the size of a barcelona nut. It is purple in colour, and in most instances it is a solitary, well-encapsulated, benign adenoma. A small proportion of these tumours show an absence of (or an invasion of) the capsule and cellular anaplasia ; they must, therefore, be classified as malignant adenomata. Nevertheless, if removed, it neither recurs nor does it give rise to metastases. A very small proportion of insulinomas are adenocarcinomata and unless such a tumour is treated by early partial pancreatectomy, it gives rise to insulin-producing metastases.

Occasionally, adenomatosis of the islets of Langerhans is the cause of hypoglycæmic attacks.

Clinical features are not stereotyped, and unless the attending clinician is alive to the bizarre symptoms to which it gives rise, islet-cell tumour of the pancreas, still somewhat of an enigma, will often continue to be displayed only at that final Court of Appeal—the post-mortem room, for so often the patient is diagnosed as psychoneurotic, epileptic, a malingerer, or a sufferer from organic nervous disease.

The attacks, due to hypoglycæmia, come on at irregular intervals and often with progressively increasing frequency and severity. Four stages are recognised, and in the beginning the attacks do not necessarily progress beyond the first or second stages.

Stage 1.—Often the symptoms simulate duodenal ulcer, awakening the patient in the early hours of the morning with vague abdominal discomfort, relieved by carbohydrates. At other times nervous symptoms predominate. There is a vague feeling of being unwell, a disinclination for exertion, and possibly some disorientation.

Stage 2.—Trembling, sweating, dizziness, blurring of vision, and great hunger.

Stage 3.—Sluggish mind, inarticulate speech, inco-ordinated movements, diplopia, sometimes hallucinations.

Stage 4.—Fits indistinguishable from epilepsy[1] passing into semi-consciousness or coma, with dilated pupils and extreme spasticity. In course of time intellectual deterioration is liable to follow.

Usually the patient is an adult under forty years of age, but a few cases have

[1] The brain cannot store dextrose, which it requires in large amounts. Deprivation of this essential brings about a burst of nervous energy ; the higher centres are affected first.

occurred in children. Because eating relieves the symptoms, over-weight not infrequently results. Pain is not a feature of benign insulinoma, but is present in most cases where a malignant change has occurred.

In a few instances the tumour contains sufficient calcium to show in a radiograph.

Diagnosis.—The three criteria (Whipple's triad), which, if present, establish the diagnosis, are :

1. An attack can be induced by complete starvation (food, not water) for twelve to twenty-four hours.

2. During the height of the attack there is hypoglycæmia, often below 50 mg. per 100 ml.

3. The symptoms usually pass off rapidly after ingestion or intravenous administration of dextrose solution.

Sugar tolerance tests show that usually, while fasting, a patient with a functioning islet-cell tumour has hypoglycæmia. Negative findings are equivocal. A normal blood-sugar should not be allowed to exclude the diagnosis of a suspected case in coma, and after 2 pints (1·14 l.) of isotonic dextrose solution has been given intravenously without response, 5 to 10 units of insulin should be tried. The successful termination of prolonged coma can sometimes be brought about in this manner, the dose of insulin acting as a stimulus to the cerebral mechanism. As a first-aid measure rectal administration of dextrose can be tried, but the absorption is slow and incomplete.

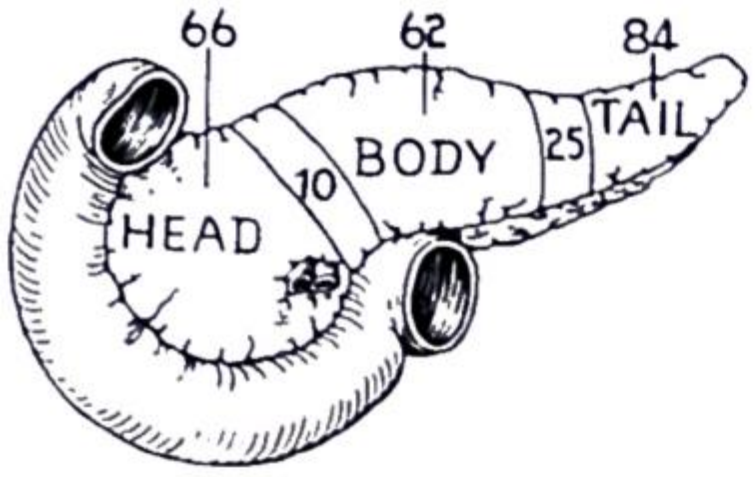

FIG. 632.—Site of the insulin-secreting adenoma in 254 cases. Seven were situated in an ectopic pancreas. Twenty were overlooked at operation. (*J. M. Howard et al's statistics.*)

Treatment.—The only curative treatment is extirpation of the tumour or tumours. With an intravenous drip infusion of isotonic dextrose solution running, laparotomy is performed and the pancreas is displayed. When the tumour (fig. 632) is not located at once, the pancreas should be mobilised (see p. 459) to enhance thorough palpation. In 12 per cent. of cases more than one tumour is present. Once found by palpation, an incision is made in the pancreas over the tumour. As a rule complete enucleation is possible. Drainage of the area is essential. Undue hardness of the tumour calls for partial pancreatectomy, but this measure is required most often because of failure to locate the tumour, in which event more than half the specimens removed reveal no abnormality. Consequently, the result in these instances is not always what is hoped for. In cases where the tumour is removed, 87 per cent. of patients gain relief of all symptoms ; in 4 per cent. there is slight or no relief. The mortality is about 8 per cent.

Non-insulin Producing Islet-cell Adenoma.—Structurally similar, symptomless islet-cell adenoma is very much more common than its symptom-producing sisters. If looked for especially, a small tumour (usually it is single) answering this description is found within the pancreas in 0·5 to 1 per cent. of necropsies. It is inaccurate to state that these tumours never cause symptoms ; at least four non-insulin producing tumours have progressed to form an abdominal mass, and have been removed by partial pancreatectomy.

Non-insulin Producing Islet-cell Adenoma engendering Peptic Ulcer.—Recently a rare new clinical entity consisting of fulminating and frequently fatal peptic ulceration, associated with a non-insulin producing islet-cell tumour or tumours of the pancreas has been described. The syndrome is characterised by excessive gastric secretion and a rapidly progressive, often atypically located, peptic ulcer that recurs despite adequate medical or surgical treatment (E. H. Ellison). Following enucleation of the tumour, or partial pancreatectomy, gastric hypersecretion and recurrent peptic ulceration cease, suggesting that an as yet unidentified hormonal factor is elaborated by the pancreatic tumour. Obviously, therefore, in cases of recurrent peptic ulceration a careful search should be made of the pancreas for such a tumour. Finally, because of the difficulties encountered in palpating small pancreatic adenomata it is suggested that rather than subject the patient to total gastrectomy as a final heroic method of controlling recurrent peptic ulceration, it may be advisable to amputate the body and tail of the pancreas.

Edward Homer Ellison, Contemporary. Associate Professor of Surgery, Ohio State University College of Medicine, Columbia, Ohio.

CARCINOMA OF THE PANCREAS

- **of the Head 70%**
  - The head proper (more common).
  - The periampullary region (rare).
- **of the Body and Tail 30%.**

Primary carcinoma of the pancreas accounted for 3,458 deaths in England and Wales in 1955. Most of the victims of the disease are past the meridian of life, the average being fifty-seven years of age ; it is, however, not very unusual to encounter this condition in patients who are in their thirties. Men are attacked twice as often as women.

Pathology.—Usually the lesion is scirrhous, almost gritty to the knife edge, and fibrous in appearance. Occasionally it is medullary ; exceptionally it is a cystadenocarcinoma.

Carcinoma of the head proper never attains the size of a fist ; rarely is it more than one-quarter of this size. The reason for this is that while still comparatively small it compresses the common bile duct (fig. 633), and unless the obstruction is relieved early, death from cholæmia results.

FIG. 633.—Carcinoma of the head of the pancreas causing obstruction to the common bile duct and the duct of Wirsung.

Periampullary Carcinoma.—In less than one-third of cases of carcinoma of the head of the pancreas the neoplasm arises on the duodenal papilla (fig. 634), in the ampulla of Vater, or in the duodenal mucosa adjacent to the papilla. When such an operation or necropsy specimen is examined, usually it is impossible to decide which of the three was the seat of origin—hence they are grouped together under the comprehensive term ' periampullary.'

FIG. 634.—Periampullary carcinoma. While still small, it obstructs the flow of bile and pancreatic juice, rendering comparatively early diagnosis possible.

Carcinoma of the body and tail tends to attain a greater size than the foregoing varieties ; it also metastasises more widely.

Spread : *Local Spread.*—Carcinomata belonging to the first and third categories tend to adhere to, and invade, adjacent structures, particularly the duodenum, occasionally the pyloric antrum, and rarely the colon or the kidney.

Lymphatic spread from the head is first to the subpyloric lymph nodes ; that from the body and tail is to the gastric, cœliac, mesenteric, and para-aortic lymph nodes. The supraclavicular lymph nodes are involved less commonly than in cases of carcinoma of the stomach.

Spread by the blood-stream occurs somewhat later. Metastases are carried especially to the liver ; usually the hepatic metastases are small and numerous.

Peritoneal implantation resulting in ascites occurs in 10 per cent. of cases.

Clinical Features :

Painless jaundice is commonly regarded as the hall-mark of carcinoma of the pancreas. Undoubtedly the great emphasis placed on this phenomenon has led many to believe that carcinoma of the pancreas necessarily presents in this guise, which is highly erroneous and has militated against early diagnosis of this fell disease not presenting in this form. Actually painless jaundice is associated only with periampullary carcinoma, and not more than 16 per cent. of carcinomata of the head of the pancreas belong to this category. So it comes about that in carcinoma of the pancreas more patients have pain first than jaundice first. In point of fact, in 80 per cent. of cases pain precedes the jaundice.

Pain which is unrelated to food takes two forms. In growths of the head of the pancreas there is a dull ache in the epigastrium. In growths in the body or tail the pancreatic pain radiates to the back or left side. The latter pain is worse when the patient lies down, and therefore is most in evidence at night. Perhaps the most distinctive feature of the pain of carcinoma of the pancreas is its persistence—week in, week out ; this symptom alone should call for laparotomy.

Loss of weight occurs in 90 per cent. of cases, and although it may not bring the patient to seek advice, it frequently averages 5 lb. (2·3 kg.) per month.

Jaundice.—It is fortunate that in the great majority of cases of carcinoma of the head of the pancreas, obstruction to the common bile duct occurs at a relatively early stage. One characteristic feature should be noted especially—jaundice is steadily progressive. The icteric tinge becomes deeper and deeper until the skin and conjunctivæ assume an almost mahogany hue.

An exception to this rule sometimes occurs in cases of periampullary carcinoma. After about a month of progressive (usually painless) jaundice, necrosis of a portion of the growth occurs, and pent-up bile escapes into the duodenum. Variations in the depth of the jaundice and pyrexia, from infection ascending the bile passages from the necrotic growths, heightens the similarity of the symptoms caused by this neoplasm to those of obstruction of the common bile duct by a gall-stone.

In carcinoma of the body or tail of the pancreas when jaundice arises (which it does in 10 per cent. of cases) it is due to secondary deposits.

Itch.—In most instances itching coincides with the onset of jaundice, and persists as the jaundice deepens. In a few instances, despite deepening jaundice, the itch lessens or disappears. In periampullary tumour, itching, which is due to bile-salts in the blood, precedes jaundice by several days.

Vomiting is not rare, and strangely it occurs more frequently in cases of periampullary carcinoma.

Bowels.—Constipation is most frequent, but sometimes diarrhœa occurs in cases of carcinoma of the head, due to steatorrhœa. In cases with obstructive jaundice the stools, of course, are putty-coloured.

Distended Gall-bladder.—In cases of carcinoma of the head of the pancreas the most important physical sign is enlargement of the gall-bladder, which is

palpable in about 60 per cent. of cases. In many of the remainder the gall-bladder, although enlarged, cannot be felt because of the intervention of an enlarged liver.

Enlargement of the Liver.—The liver is palpably enlarged in 65 per cent. of cases of carcinoma of the head of the pancreas. As a rule this enlargement is due to biliary obstruction, in which case the liver is tender, or to fatty infiltration consequent upon deficiency of lipotropic substances which are dammed back within the obstructed pancreas. On the other hand, in cases of carcinoma of the body or tail, enlargement of the liver is due to hepatic metastases which, of course, can be the cause of super-added enlargement in late cases of carcinoma of the head of the organ.

An epigastric mass (not the liver) is present in 1 in 3 cases of carcinoma of the body of the pancreas.

Anæmia is not so much in evidence as in carcinoma of the stomach.

Thrombophlebitis migrans of the lower extremities is often the first striking symptom of carcinoma of the tail of the pancreas. In a number of instances this manifestation alone has led to the performance of laparotomy for ' ? carcinoma of the tail of the pancreas,' and the diagnosis has proved to be correct. The explanation of this phenomenon, which was first described by Trousseau, is still obscure.

Acute cholecystitis occasionally is the first indication of carcinoma of the head of the pancreas, and in patients of the older age group this possibility should come to mind.

Diabetes and Carcinoma of the Pancreas.—(*a*) Diabetes is occasionally the first sign of carcinoma of the pancreas. Typical pain may be a pointer or completely absent. The condition should be suspected when an elderly person develops diabetes and, in spite of adequate diabetic treatment, continues to lose weight. The sedimentation rate is helpful (R. D. Lawrence). In these patients often the disease runs a galloping course.

(*b*) The pancreas is the most common site for malignant disease in the diabetic patient, and it is 6 to 16 times as frequent in these patients as in the population at large.

Investigations.—Those who rely mainly on radiography and laboratory procedures to provide the diagnosis will, in early cases of carcinoma of the pancreas, go unrewarded.

Radiography.—As will be appreciated readily, radiography has nothing to offer in cases of carcinoma of the body or tail of the pancreas. In the case of carcinoma of the head of the organ the U of the duodenum filled with barium is sometimes broadened—the pad sign (fig. 635), and periampullary carcinoma occasionally provides a filling defect. Visualisation of the common duct by intravenous cholangiography in non-jaundiced patients might conceivably demonstrate displacement of the common bile duct. Too often by the time these signs are positive, the growth is far advanced.

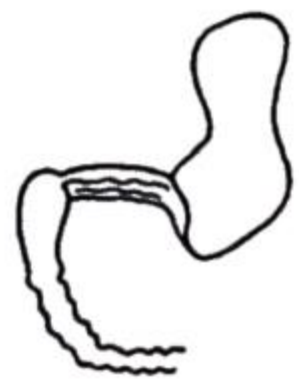

FIG. 635.—The pad sign.

Laboratory Tests :

The stercobilinogen content of the fæces is often lower than that found in chronic pancreatitis—less than 10 mg. per day.

Armand Trousseau, 1801–1867. Physician, Hôtel Dieu, Paris.
Robert Daniel Lawrence, Contemporary. Physician in charge of the Diabetic Department, King's College Hospital, London.

Occult blood in the stools is a valuable finding. It is especially frequent in cases of periampullary carcinoma.

Hypoproteinæmia occurs earlier, and more frequently, than it does in carcinoma of the stomach.

When jaundice is present the problem of distinguishing obstructive jaundice from infective hepatitis is a weighty one, and liver function tests (see p. 394) and liver biopsy are of cardinal importance.

Finally, patients presenting with upper abdominal pain, loss of weight, and negative X-ray and laboratory findings deserve abdominal exploration if the diagnosis of carcinoma of the pancreas is to be made at a curable stage.

TREATMENT OF CARCINOMA OF THE HEAD OF THE PANCREAS

When the patient is deeply jaundiced and the liver function is poor, preliminary cholecystostomy is performed through a small incision, with a view to improving the patient's general condition. In other circumstances, after suitable pre-operative preparation, including a high intake of dextrose and vitamin K_1 injections, laparotomy is performed, a blood transfusion being given and continued throughout the operation.

FIG. 636.—Cholecystjejunostomy with jejunojejunostomy (to minimise ascending cholangitis).

When the growth is inoperable, as shown by secondary deposits, cholecystjejunostomy is performed. In this instance the type of anastomosis usually carried out is depicted in fig. 636. This measure, by short-circuiting the obstructed common bile duct, relieves the jaundice and prolongs life by several months. Perhaps the major blessing it confers is relief from the intolerable itching.

Confirmation of the Diagnosis of an Early Case at Operation.—A tumour of the papilla can be inspected directly by opening the duodenum. A growth at the lower end of the bile duct closely resembles a stone when palpated through surrounding tissues. When the common bile duct is opened a probe is passed to the site of the obstruction. The escape of biliary mud and a metallic ring is indicative of obstruction by a calculus. When a tumour is present in the head of the pancreas proper, greater difficulty is liable to be encountered in differentiating it from chronic pancreatitis. Biopsy through the duodenum, using an ophthalmic trephine, is safer than biopsy through the anterior surface of the gland, but the interpretation of the resulting frozen section is not infrequently difficult and unsatisfactory.

FIG. 637.—Approximating the cut edges of the common bile duct with those of the duodenal mucous membrane. Encroachment on the pancreatic duct must be avoided. (*After Rodney Smith.*)

Local excision of the neoplasm is to be undertaken only when the patient is in poor condition and a periampullary growth is found to be manifestly circumscribed. Through the open duodenum the growth is grasped in suitable forceps and excised with a margin of healthy tissue, using a diathermy needle. During the procedure the obstructed common bile and pancreatic

ducts are entered, and their pent-up secretions gush forth. A field dry of these secretions having been obtained, bleeding-points are coagulated. Mattress sutures are inserted in such a way as to approximate the common bile duct to the duodenal mucous membrane, but avoiding the duct of Wirsung (fig. 637). The anterior duodenal wall is closed.

Pancreato-duodenectomy.—The first step is to mobilise the pancreas (see p. 459). Only after this has been accomplished is it possible to determine by palpation whether resection is feasible. If the index finger can be passed from above *under* the neck of the pancreas, between it and the portal vein, so as to emerge below anterior to the superior mesenteric vessels, no special difficulty should be experienced in the resection.

The stomach is transected between crushing clamps proximal to the pylorus. The neck of the pancreas is elevated on the finger and a ligature on a needle is passed through the superior, and another through the inferior border of the nearby body of the pancreas. These ligatures are tied, thus cutting off the main blood supply to the head of the pancreas. The ligatures are left long to act as retractors (see fig. 638). The body of the pancreas is divided, bleeding on the side nearer the head of the organ being controlled by hæmostats. Before completing the transection the duct of Wirsung is so dissected as to project from the left side of the cut body. A small rubber catheter is placed into the duct to aid in its identification. With the exception of the duct, the cut surface of the body of the pancreas is closed with mattress sutures (fig. 638). The common bile duct is divided. Should the cystic duct join it low down, the gall-bladder must be removed.

FIG. 638. — The stump of the body of the pancreas prepared for anastomosis with the jejunum or the stomach.

The ligament of Treitz is divided, freeing the commencement of the jejunum, which is divided and its proximal end ligated, the ligatures being left long. By the aid of the long ends of these ligatures the proximal jejunum and the fourth part of the duodenum are drawn beneath the superior mesenteric vessels. The head of the pancreas with its uncinate process is elevated and branches feeding them from the superior mesenteric vessels are ligated; this is the most difficult part of the operation. Oozing can be controlled by absorbable gauze. The excision is now complete.

The continuity of the alimentary canal, the biliary and pancreatic tracts are restored, as shown in fig. 639. Alternatively, the body of the pancreas can be anastomosed to the stomach, as some prefer, this organ holding sutures more firmly. The abdomen is closed with wire sutures and drainage is provided. Transfusion of blood should be continued after the operation, as necessary, and vitamin K_1 injections continued for several days.

FIG. 639.—Reconstruction after pancreato-duodenectomy. The duct of Wirsung is being anastomosed to the jejunum. The common bile duct will be anastomosed to the apex of the jejunal loop and an end-to-end anastomosis performed between the stomach and the distal end of the jejunum. (*After R. B. Cattell.*)

The general mortality of this extensive procedure is about 10 per cent. The complications are principally those of leakage from the pancreas or common bile duct anastomoses. Acute dilatation of the stomach is prone to occur, and must be guarded against by gastric aspiration.

Owing to the development of metastases the life of those who survive this procedure is often only a matter of eighteen months. However, an increasing number of five-year survivals are being recorded. Naturally the most favourable cases are those where the carcinoma was of the periampullary variety.

Wenzel Treitz, 1819–1872. Professor of Pathology, Prague.

TREATMENT OF CARCINOMA OF THE BODY AND TAIL OF THE PANCREAS

When laparotomy is performed before a growth situated towards the tail of the organ has metastasised, it can be resected with comparative ease, and some lasting cures have been reported. The outlook in cases of papillary cystadenocarcinoma is particularly favourable.

Total pancreatectomy has been performed in some cases involving the neck of the organ. The survivors have lived only a comparatively short time, up to two years (because of metastases). They require surprisingly little insulin—30 to 70 units daily. Pancreatin, 10 to 12 G. daily, is given to improve fat absorption.

CHAPTER XXIV

THE PERITONEUM, OMENTUM, MESENTERY, AND RETROPERITONEAL SPACE

HAMILTON BAILEY and MCNEILL LOVE

Surgical Physiology.—The peritoneal cavity is the largest cavity in the body, the surface area of its lining membrane being nearly equal to that of the skin. This veil-like serous membrane is composed of flattened polyhedral cells, one layer thick, resting upon a thin layer of fibro-elastic tissue, the two layers constituting the peritoneum. Beneath the peritoneum, supported by a small amount of areolar tissue, lie a network of lymphatic vessels and rich plexuses of capillary blood-vessels from which all absorption and exudation must occur. In health only sufficient peritoneal fluid, which is a pale yellow, somewhat viscid fluid containing lymphocytes and leucocytes, is secreted to ensure that the more mobile viscera glide easily. Under the influence of decreased intra-abdominal pressure, occasioned by the upward movement of the diaphragm during expiration aided by capillary attraction, this secretion travels continuously in an upward direction to the subdiaphragmatic spaces, where experimental evidence shows that fine coloured particulate matter (and bacteria) are absorbed rapidly into the subperitoneal lymphatic network. In a matter of minutes these coloured particles reach the lymph nodes *above* the diaphragm.

When there is an outpouring of many ounces of peritoneal fluid, and, perhaps in addition, the contents of the stomach or duodenum escape into the peritoneal cavity through a perforation, it is extremely doubtful if the forces just described are powerful enough to counteract the law of gravity. In all probability excess of free fluid within a peritoneal cavity unobstructed by adhesions, given the opportunity, runs downwards.

After the abdomen has been opened, air enters, and if the patient is nursed in a sitting position, which is usual, the entrapped air obeys the laws of gases and comes to lie under the diaphragm, where it can be demonstrated radiologically, until it is absorbed. The same phenomenon is seen in many cases of perforated peptic ulcer, air having entered through the perforation. So long as an air-lock remains beneath the diaphragm, the forces referred to first are not operational. In these circumstances it is probable that the laws of gravity are obeyed in their entirety.

ACUTE PERITONITIS

Nearly all varieties of peritonitis are due to an invasion of the peritoneal cavity by bacteria. To such an extent is this true that when the term 'peritonitis' is used without qualification, bacterial peritonitis is implied.

Paths of Bacterial Invasion.—

Direct infection	Via perforation of some part of the gastro-intestinal canal.
	Through a penetrating wound of the abdominal wall.
	Post-operative.
Local extension	From an inflamed intraperitoneal organ, e.g. appendicitis, cholecystitis.
	Transmigration through damaged gut wall, e.g. unrelieved intestinal obstruction.
	From or *via* the Fallopian tubes.
Blood-stream	Part of general septicæmia.

Gabriello Fallopio, 1523–1562. *Anatomist and Professor of Surgery, University of Pisa.*

Even in non-bacterial peritonitis (e.g. intraperitoneal rupture of the bladder or a hæmoperitoneum) the peritoneum soon becomes infected by transmigration of organisms from the bowel, and it is not long (often a matter of hours) before chemical peritonitis becomes a peritonitis in the usual meaning of the term.

Bacteriology is very variable and accounts to a large extent for the mildness or virulence of the peritonitis.

Bacteria from the Alimentary Canal.—Nearly always bacteria causing peritonitis are derived from the alimentary canal. Usually the infection is caused by two or more strains. The commonest invaders are *Escherichia coli*, äerobic and anäerobic streptococci, and the bacteroides. Less frequently the *Clostridium welchii* is found ; still less frequently staphylococci or *Klebsiella pneumoniæ* (Friedländer's bacillus), and so on. Many of the strains of *Esch. coli*, bacteroides, and *Cl. welchii* produce toxins, and in this fact lies their ability to cause severe illness and death when they invade a large absorptive area.

The Bacteroides[1] are the predominant organisms in the lower intestine, where they greatly outnumber *Escherichia coli*. Only recently has the frequency and importance of the presence of bacteroides in the causation of peritonitis been realised. These Gram-negative, non-sporing organisms often escape detection because they are strictly anäerobic, and slow to grow on culture media unless there is an adequate carbon-dioxide tension in the anäerobic apparatus (W. A. Gillespie), and in many laboratories, if there is no growth in forty-eight hours, the culture is discarded. The most important members of this group are :

1. *B. Fundiliformis.*—In early infections this organism is cocco-bacillary in shape. Later it becomes filamentous. It is the most pathogenic variety of the bacteroides. In animals it is the cause of calf diphtheria and foot-rot of sheep.
2. *B. Fragilis* is a small bacillus of constant form.
3. *Fusobacterium plauti-vincenti* is the identical organism that is so well-known as an associate of *Borrelia vincenti* (see p. 152).

These organisms, which are resistant to penicillin and streptomycin but sensitive to tetracycline and chloramphenicol, are often the cause of peritonitis (in conjunction with other organisms) and suppuration of the abdominal wall following laparotomy.

Bacteria NOT *from the Alimentary Canal.*—There are forms of peritonitis that are due to organisms that do not originate from the intestinal tract. Examples are peritonitis due to the gonococcus, beta hæmolytic streptococcus, pneumococcus, and the *M. tuberculosis*. In time past hæmolytic streptococcal peritonitis was associated with a high mortality, but since the advent of antibiotics it has lost much of its dreaded lethal properties.

Factors which favour localisation of peritonitis are anatomical and pathological.

Anatomical.—Excluding the subphrenic spaces, which will be considered later, the greater sac of the peritoneum is divided into (*a*) the pelvis and (*b*) the peritoneal cavity proper. The latter is re-divided into a supracolic and an infracolic compartment by the transverse colon and transverse mesocolon, which deters the spread of infection from one to the other. The lower abdomen is divided into a right and a left compartment by the mesentery and the bodies of the vertebræ, both of which hinder the passage of infection across the middle line, but each compartment communicates freely with the pelvis. When the supracolic compartment overflows, as is often the case

[1] Bacteroides = bacteria with rounded ends.

William Henry Welch, 1850–1934. Professor of Pathology at the Johns Hopkins University, Baltimore.
Carl Friedländer, 1847–1887. Prosector, Berlin-Friedrichshain Hospital.
William Alexander Gillespie, Contemporary. Pathologist, Royal Infirmary, Bristol.
Jean-Hyacinth Vincent 1862–1950. Professor of Epidemiology, Val de Grâce Military Hospital, Paris.

when a peptic ulcer perforates, it does so over the colon into the right infracolic compartment, and so, by way of the right paracolic gutter, to the right iliac fossa, and thence to the pelvis.

Pathological.—Inflamed peritoneum loses its glistening appearance and becomes reddened and velvety. Flakes of fibrin appear and cause coils of intestine to become adherent to one another and to the parietes. There is an outpouring of serous fluid rich in leucocytes and antibodies, that soon becomes turbid ; should the infection not be overcome quickly, and especially if localisation occurs, the turbid fluid becomes frank pus. Peristalsis is retarded in affected coils, and this helps in preventing distribution of the infection to other coils. The greater omentum, by enveloping and becoming adherent to inflamed structures, often forms a substantial barrier to the spread of infection. In short, the clinical course is largely governed by the manner in which adhesions form. If, for instance, a carcinoma of the sigmoid colon ulcerates slowly until a state is reached where bacteria can penetrate the intestinal wall without a breach of continuity thereof, adhesions between the serosa and neighbouring structures are likely to prove a formidable barrier to the spread of peritonitis should perforation occur.

Factors which tend to cause diffusion of peritonitis are as follows :

(*a*) The prime factor in peritonitis is whether it develops rapidly or slowly. If an inflamed appendix or other hollow viscus perforates before the natural defences set out above have had time to come into action, there is a gush of intestinal contents (fig. 640) into the peritoneal cavity that spreads over a large area almost instantaneously.

(*b*) The ingestion of food, or even water, by stimulating peristaltic action, hinders localisation. Violent peristalsis occasioned by the administration of a purgative or an enema causes a widespread distribution of an infection that would otherwise have remained localised.

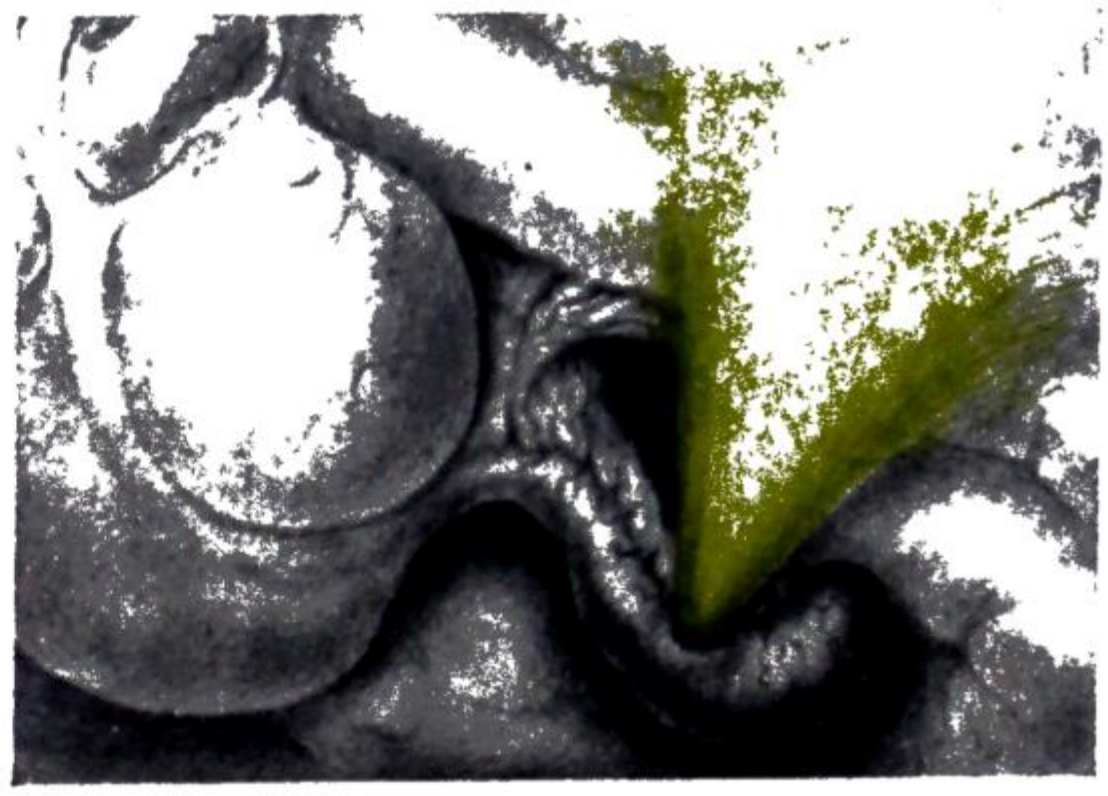

FIG. 640.—Sudden perforation, especially if engendered by purgation, often results in an immediate widespread bacterial peritonitis.

(*c*) When the virulence of the infecting organism is so great as to render the localisation of the infection difficult or impossible.

CLINICAL FEATURES

Localised peritonitis is bound up intimately with the causative lesion, and the initial symptoms and signs are those of the lesion. When the peritoneum becomes inflamed the temperature, and especially the pulse-rate, rise. The pain increases and usually there is repeated vomiting. The most important sign is rigidity of the abdominal wall over the area of the abdomen

which is involved and as, even in favourable circumstances, the process of walling-off by adhesions cannot be expected to occur for at least twenty-four hours, in the beginning the area of rigidity is likely to be relatively extensive. In cases of pelvic peritonitis arising from an inflamed appendix in the pelvic position or from salpingitis, the abdominal signs are often slight, deep tenderness of one or both lower quadrants alone being present, but a rectal or vaginal examination reveals tenderness, often exquisite tenderness, of the pelvic peritoneum. With appropriate treatment localised peritonitis usually resolves. In about 20 per cent. of cases an abscess follows. Infrequently, localised peritonitis becomes diffuse. Conversely, in favourable circumstances diffuse peritonitis can become localised, most frequently in the pelvis.

Diffuse (*syn.* **Generalised**) **Peritonitis.**—The most typical example is that caused by perforation of a peptic ulcer. In this instance the onset of symptoms is followed in a matter of minutes by widespread board-like abdominal rigidity. The commonest cause of diffuse peritonitis is gangrene or perforation of an inflamed appendix occurring within twenty-four hours of the attack, that is before there has been time for the infection to become walled-off from the general peritoneal cavity. Diffuse peritonitis from any cause may be divided into three stages :

1. *Early.*—This is the stage of diffusing or spreading peritonitis. Pain, which commenced in one position of the abdomen, becomes more widespread ; the exception to this rule is post-operative peritonitis, in which pain is almost absent. Vomiting becomes very frequent, bile-stained, and often effortless. The patient lies supine, with his knees flexed. The temperature is usually raised, but in fulminating cases it may be subnormal. The pulse-rate is elevated. A rising pulse-rate, as shown by a two-hourly or, in very acute cases, a one-hourly pulse-chart, is an indication that peritonitis is advancing. On examination the tongue is moist and the face somewhat flushed. Inspection of the abdomen shows that there is little or no respiratory movement of the abdominal wall. Palpation reveals widespread rigidity. On auscultation of the abdomen no sound of peristalsis can be heard.

2. *Intermediate.*—The third day is the critical one in diffuse peritonitis. In cases where a favourable outcome can be confidently expected, the pulse-rate ceases to rise, or commences to fall. The temperature, which may have been normal or subnormal, rises and less fluid is withdrawn on gastric aspiration. If the inflammation is not subsiding, the pulse-rate continues to rise. The rigidity to some extent passes off, and gives place to distension. The whole abdomen is acutely tender. The amount of fluid removed by gastric aspiration increases and becomes fæculent. Even after an enema, little or no flatus is passed.

3. *Later Stage.*—If by the fourth or fifth day no semblance of localisation has occurred the patient's condition becomes extremely grave. The whole abdomen is grossly distended (meteorism). The pulse becomes thready and rapid. The eyes are sunken but bright, the nose is pinched, there are sordes on the lips, the tongue is dry and shrivelled, the forehead and the hands are

cold and clammy, and the facies drawn and anxious (Hippocratic facies). Finally the patient lapses into semi-consciousness (fig. 641) and the end is not far distant.

FIG. 641.—The facies in terminal diffuse peritonitis.

TREATMENT

Early Operative Treatment.—After adequate preparation by measures detailed under conservative treatment (see below), early operation is indicated in cases of perforation of any hollow viscus and in early localised as well as in diffuse peritonitis due to acute appendicitis. If it is decided that operation is necessary, an injection of omnopon, possibly with scopolamine, is administered while the pre-operative measures are carried out. In this group of cases early operation, by preventing further extravasation of infected material or by extirpating the focus of infection, usually results in rapid resolution of the inflammation. Pre- and post-operative treatment includes antibiotic therapy and many of the measures included under conservative treatment.

Drainage is not required in many cases of early infection of the peritoneum. After dealing with the primary focus, purulent exudate is removed by suction or by gentle mopping, and the abdomen is closed. Apart from drainage of residual abscesses, which will be considered later, drainage of the peritoneal cavity is recommended in the following circumstances :

Suprapubic drainage by a tube passed into the pelvis through a stab incision in the middle line above the pubis is advised :

1. After suture of a perforated peptic ulcer when there is considerable extravasation of fluid and more than six hours has elapsed from the time of perforation.

2. After appendicectomy in cases of peritonitis due to perforated obstructive appendicitis.

3. In the uncommon cases of diffuse peritonitis due to perforation of the small or large intestine, to perforation of an infected gall-bladder or bile duct, and in cases of primary streptococcal or pneumococcal peritonitis submitted to operation.

Drainage through the flank is called for when retroperitoneal tissues are infected, as is often the case in gangrenous retrocæcal appendicitis.

A suprapubic drainage tube is usually rotated on the second day, and removed on the third day. A drainage tube in the flank, if a considerable amount of pus is being discharged, should be shortened but left in place for upwards of five days.

Conservative treatment is indicated :

1. In any case of diffuse peritonitis where the general condition is poor.

2. Usually in localised peritonitis due to cholecystitis.

Hippocrates, by common consent the Father of Medicine, was born in the Island of Cos about 460 B.C. He lived to be 109 years of age in an era when the average expectation of life was 32 years.

Albert John Ochsner, 1858–1925, Surgeon, Augustana Hospital, Chicago, formulated the conservative treatment of late cases of diffuse peritonitis.

3. In most cases of localised peritonitis due to appendicitis of over forty-eight hours' duration.

4. In peritonitis due to salpingitis.

5. In pneumococcal peritonitis when the diagnosis is certain.

In cases where conservative measures are employed, there is usually a decided improvement within six hours, which is maintained. Patients undergoing this form of treatment require careful watching and repeated examination. Operation is required when the response to the treatment is not wholly satisfactory, or in cases of non-resolution of a local abscess.

Antibiotic Therapy.—A combination of penicillin, 500,000 units *bis die*, and streptomycin, 0·5 G. *bis die*, given intramuscularly and continued until the temperature and pulse-rate have been normal for several days, is justly popular. Nevertheless, the surgeon should be prepared to substitute one of the tetracyclines, preferably tetracycline 0·5 to 1 G. in a litre of dextrose-saline given slowly into a vein by the drip method during each period of twelve hours, should the former drugs not prove effective. Tetracycline is chosen because it is associated with the lowest incidence of fulminating staphylococcal gastro-enterocolitis (see p. 534).

Posture.—Fowler's position, by which the aid of gravity is invoked to direct purulent fluid within the peritoneal cavity towards the pelvis, is not considered so essential as formerly. Nevertheless, during the first forty-eight hours of treatment of peritonitis, most surgeons consider it advisable to prop the patient up with a back-rest and pillows. The high position, whereby the head of the bed is raised 18 inches (45 cm.) on blocks has, to a large extent, been abandoned in favour of the low position, where 6-inch (15-cm.) blocks are employed. All are agreed on one fundamental point—that in no circumstance should a pillow be placed under the thighs or knees, or a mechanical device (e.g. a 'donkey' or cranking frame) be employed under the mattress to maintain the position—because these devices predispose to phlebothrombosis. Frequent changes of position are highly desirable, by which is meant that the patient is urged to lie at one time on the right side and at another on the left. These expedients discourage the development of phlebothrombosis decubiti, and they also facilitate deep respiration.

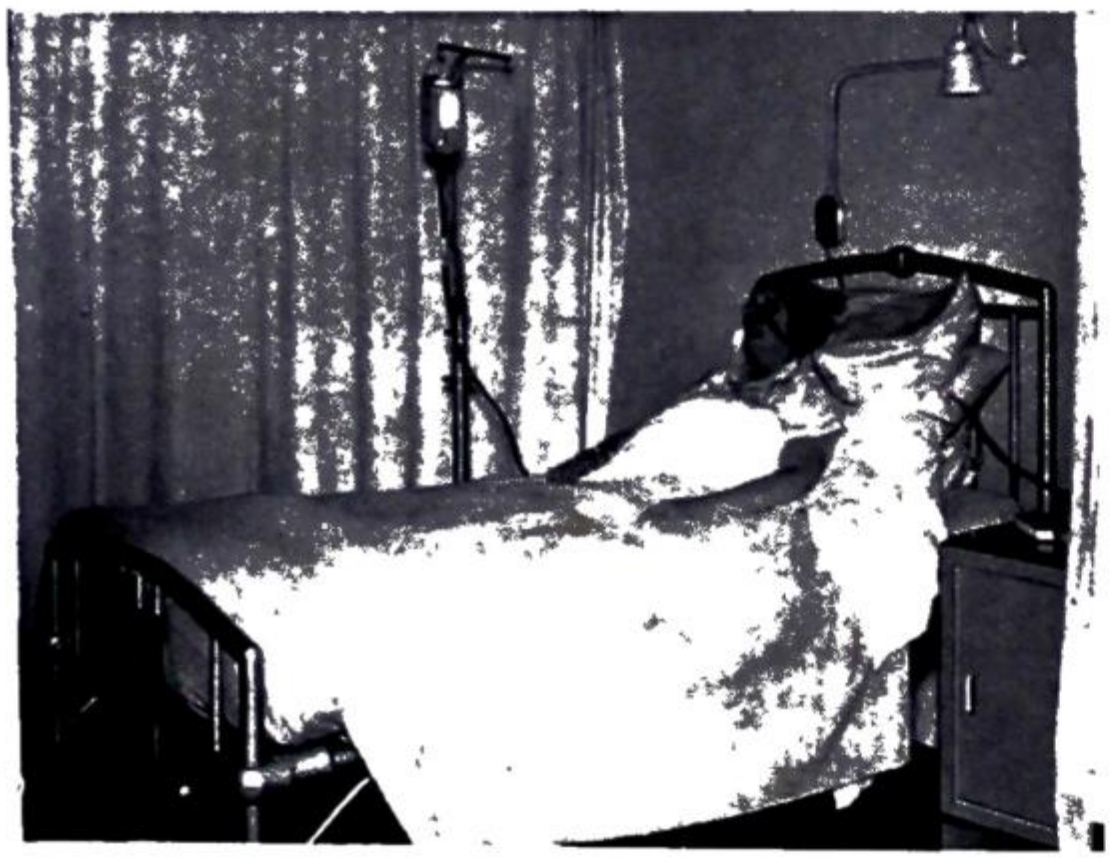

FIG. 642.—The bulwarks of the conservative and post-operative treatment of peritonitis: (1) Modified Fowler's position. (2) Transnasal gastric aspiration. (3) Continuous intravenous dextrose-saline. (4) *Charts*. A two-hourly pulse and temperature chart and a fluid balance chart.

George Ryerson Fowler, 1848–1906. Surgeon, Brooklyn Hospital, New York.

Rest to the Alimentary Canal.—No food or fluid is allowed to be ingested until the inflammation has subsided, as indicated by a fall of the pulse-rate to the eighties and a corresponding fall in the temperature. The bowels are left confined, unless they are opened naturally; enemata are not given until the acute stage is over, but a flatus tube is passed. There should have been no vomiting or potential vomiting (i.e. material withdrawn via the indwelling gastric tube) for forty-eight hours, a return of bowel sounds before fluids are allowed by mouth, and the bowels should have acted before even milky foods are allowed. Usually these criteria are reached by the fourth day, but in severe cases it may be the fifth or sixth day.

Gastric aspiration by a Ryle's or plastic tube, preferably passed transnasally, is commenced and continued as long as bile or intestinal contents is withdrawn. With the tube in place the patient is allowed to drink a small quantity (an ounce) of water occasionally, provided the fluid is aspirated promptly. If, after the tube has been withdrawn, vomiting recommences or the patient hiccups, the tube must be reinserted. Throughout the treatment frequent mouth-washes are given.

Intravenous dextrose (4·3 per cent.) **and saline** (0·18 per cent.) **solution** is administered by the drip method at the rate of forty drops a minute. In cases of severe peritonitis associated with paralytic ileus, when the intravenous route is the only one by which the patient can receive nourishment for several days, the addition of a preparation of amino acids for intravenous administration is valuable in maintaining the patient's strength.

Charts.—A two-hourly pulse-rate chart is recorded graphically. In cases causing anxiety, when the advisability of operation is undecided, a one-hourly chart is compiled. The temperature is recorded every four hours. No less important is an intake and output chart, which must be kept for as long as the patient is receiving fluids intravenously.

Sedative Drugs.—While the patient is being watched to determine if there is a favourable response to conservative measures, all sedative drugs should be withheld. Once it has been decided to treat the case conservatively, omnopon $\frac{1}{3}$ grain (20 mg.) is given subcutaneously, or half that dose intravenously.

Electrolytic Balance.—There is no condition in which the maintenance of electrolytic balance is more important than in the case of peritonitis, and the principles set out in Chapter 5 should be followed.

When a patient is being sustained only by parenteral fluids a careful watch must be kept for chloride depletion, which is apt to occur as a result of the initial vomiting or subsequent gastro-intestinal aspiration, and potassium deficiency (see p. 76).

Vitamins, especially B complex and C, should be given parenterally at first and later by mouth. Some of the antibiotics (particularly chloromycetin and aureomycin, given orally) produce vitamin deficiencies, notably of the B complex.

Blood transfusion, preferably fresh blood, is required if the hæmoglobin falls below 70 per cent.

John Alfred Ryle, 1889–1950. Professor of Social Medicine, University of Oxford.

The conservative treatment of peritonitis is considered more fully in relation to late cases of appendicitis (Chapter 27).

Hydrocortisone in Grave Diffuse Peritonitis.—Provided only that a decision has been reached that operation will be performed if and when the patient is fit to undergo it, cortisone can be used to borrow time in which to replace fluid and electrolytes and administer antibiotics. Adreno-corticosteroid and adreno-corticotrophic hormones exert an anti-inflammatory effect, and also a toxin-blocking action. The danger is that they also inhibit the formation of adhesions, and consequently the localisation of peritonitis. So great may be the improvement following the administration of these hormones that a fool's paradise is created at a time when the peritonitis is *in statu quo*, or advancing. Therefore the watchword is *operate while the current serves*, and continue with the hormone for at least two days after the operation. The dosage is similar to that described for grave acute pancreatitis (see p. 467).

Prognosis.—With modern treatment diffuse peritonitis carries a mortality of about 10 per cent. The lethal factors are (*a*) bacterial toxæmia; (*b*) paralytic ileus, and (*c*) bronchopneumonia.

COMPLICATIONS OF PERITONITIS

All the complications of a severe bacterial infection are possible, but the special complications of peritonitis are as follows :

1. Intestinal obstruction (see Chapter 26).
2. Paralytic ileus (see Chapter 26).
3. Residual abscesses.

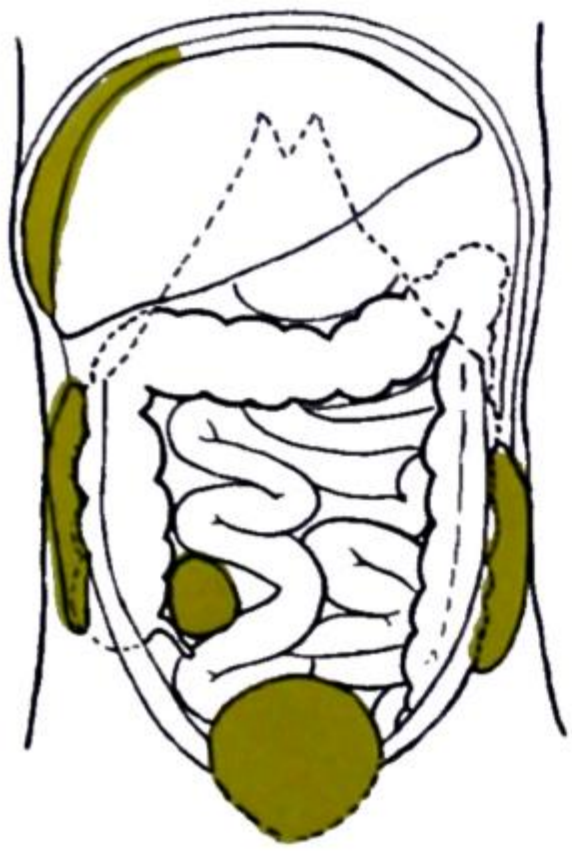

FIG. 643. — Common situations for residual abscesses.

Abscess formation following local or diffuse peritonitis usually occupies one of the situations shown in fig. 643. When palpable, an intraperitoneal abscess should be treated by marking out its limitations on the abdominal wall, and careful daily examination. In a number of instances, with the aid of antibiotic treatment, the mass becomes smaller and smaller, and finally is impalpable. In others the abscess fails to resolve, or becomes larger, in which event it must be opened. In many situations, by waiting a few days the abscess becomes adherent to the abdominal wall, so that it can be drained without opening the general peritoneal cavity.

In the case of a laterally-placed abscess, the incision is made on the lateral side of the swelling. The layers of the abdominal wall are divided until the peritoneum is reached. With the finger, the extraperitoneal tissues are separated from the peritoneum until the abscess is opened. A drainage tube is inserted ; if the path is tortuous, a Penrose drain (a thin tube of latex rubber containing a wick of gauze) is best.

PELVIC ABSCESS

The pelvis is the commonest site of an intraperitoneal abscess, because the vermiform appendix is often pelvic in position and also the Fallopian tubes are a frequent focus of infection. A pelvic abscess can also occur as a sequel to any case of diffuse peritonitis. Pus can accumulate in this area without serious constitutional disturbance, and unless the patient is examined carefully from day to day, such abscesses may attain considerable proportions before being recognised. The most characteristic symptoms of a pelvic abscess are diarrhœa and the passage of mucus in the stools. It is no exaggeration to say that the *passage of mucus, occurring for the first time in a*

Charles Bingham Penrose, 1862–1925. Professor of Gynæcology, University of Pennsylvania, Philadelphia.

patient who has, or is recovering from, peritonitis, is pathognomonic of pelvic abscess. Rectal examination reveals a bulging of the anterior rectal wall which, when the abscess is ripe, becomes softly cystic. It is inaccurate to say that it fluctuates, unless fluctuation can be elicited between it and the abdominal wall. Fluctuation cannot be tested with one finger. Left to Nature, a proportion of these abscesses burst into the rectum, after which the patient nearly always recovers rapidly. It is too hazardous to wait for this possible happy termination. A pelvic abscess should be drained deliberately. In certain cases, notably those where the primary focus is in the Fallopian tubes, vaginal drainage through the posterior fornix is chosen. In other cases, where the abscess is definitely pointing into the rectum, rectal drainage (fig. 644) is employed. If any uncertainty exists as to the presence of pus, an aspirating needle introduced through the rectal wall into the bulging swelling will settle the question. Drainage of a pelvic abscess into the rectum is exceedingly efficacious in selected cases, but occasionally, in the case of a large abscess which can be palpated above the pubes, lower laparotomy should be undertaken in order to be quite certain of the diagnosis. Provided the abscess is shut off from the general peritoneal cavity, a point which can be ascertained undeniably when the abdomen has been opened, rectal drainage of a pelvic abscess is preferable to suprapubic drainage, which in many cases unavoidably breaks down Nature's barriers and exposes the general peritoneal cavity to the dangers of spreading infection.

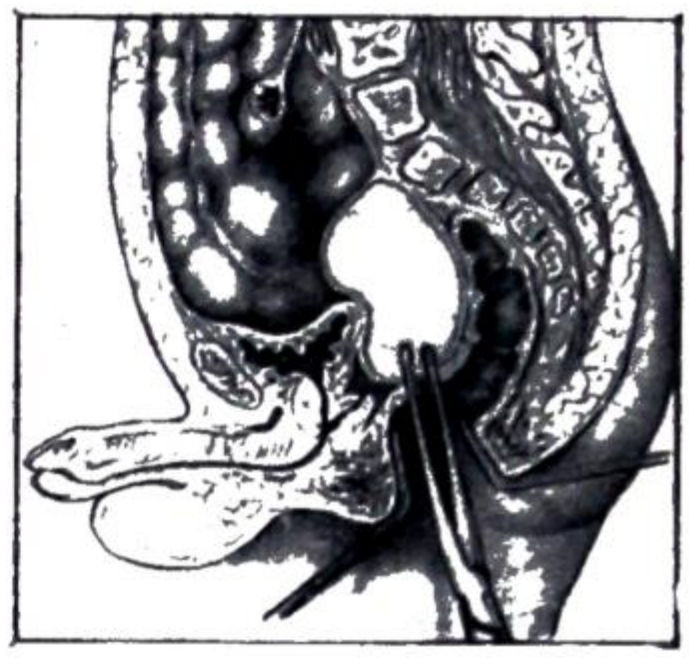

FIG. 644.—Opening a pelvic abscess into the rectum.

SUBPHRENIC ABSCESS

Anatomy.—The complicated arrangement of the peritoneum results in the formation of seven spaces in which pus may collect. Three of these spaces are on either side of the body, and one approximately in the midline (fig. 645).

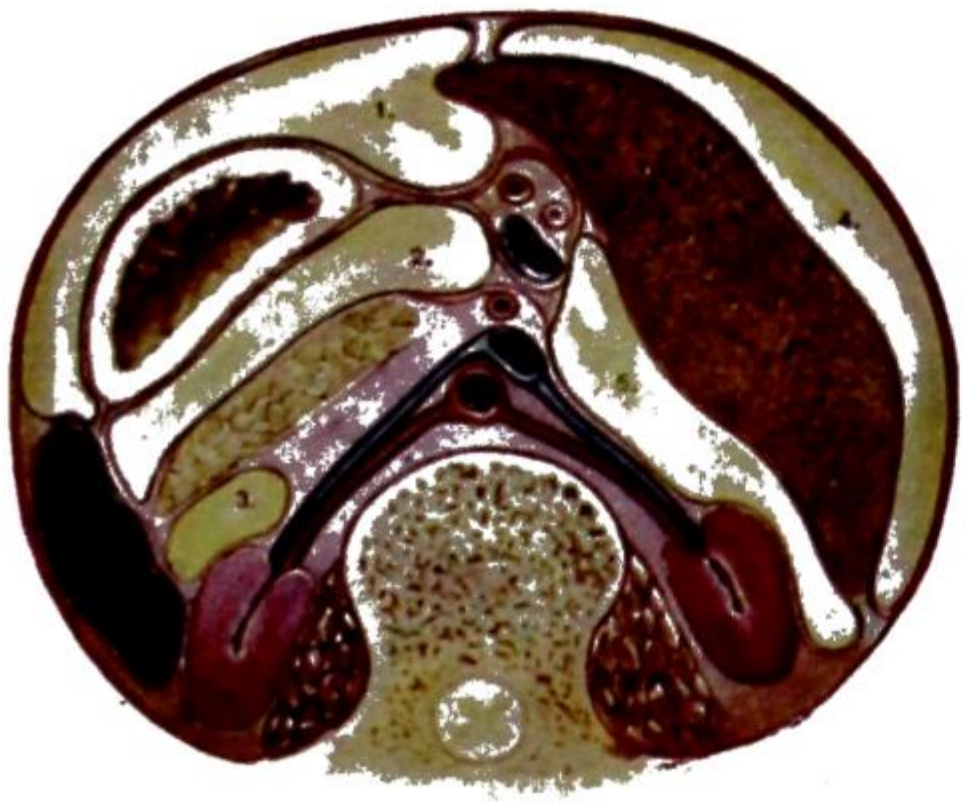

FIG. 645.—Anatomical relationships of five types of subphrenic abscess. 1. Left anterior intraperitoneal; 2. Left posterior intraperitoneal; 3. Left extraperitoneal; 4. Right anterior intraperitoneal; 5. Right posterior intraperitoneal.

Left Side.—*Anterior intraperitoneal*, bounded above by the diaphragm, behind by the left triangular ligament and left lobe of the liver, the gastro-hepatic omentum and anterior surface of the stomach. To the right is the falciform ligament, and to the left the spleen, gastro-splenic omentum, and diaphragm.

Posterior intraperitoneal, which is the upper part of the lesser sac (or omental bursa). Consequently, it is bounded behind by the diaphragm, pancreas, mesocolon, and transverse colon; in front by the caudate (Spiegelian) lobe, gastro-hepatic omentum, and stomach, and to the left by the lieno-renal ligament, spleen, and gastro-splenic omentum. On the right side is the duodenum, above which is the foramen of Winslow.

Extraperitoneal, which is normally only a potential space. When an abscess forms, pus collects near the upper pole of the left kidney, and strips the peritoneum off the diaphragm.

Right Side.—*Anterior intraperitoneal*, which lies between the right lobe of the liver and the diaphragm. It is limited posteriorly by the anterior layer of the coronary and the right triangular ligaments, and to the left by the falciform ligament.

Posterior intraperitoneal (*syn.* Rutherford Morison's kidney pouch) lies transversely beneath the right lobe of the liver. It is bounded on the right by the right lobe of the liver and the diaphragm. To the left is situated the foramen of Winslow, and

Adrian van den Spiegel, 1578–1625. Professor of Anatomy and Surgery, Padua.
Jacob Winslow, 1669–1760. A Dane who migrated to Paris, and there established a School of Anatomy.
Rutherford Morison, 1853–1939. Professor of Surgery, University of Durham.

below this lies the duodenum. In front are the liver and gall-bladder, and behind, the upper part of the right kidney and diaphragm. The space is bounded above by the liver, and below by the transverse colon and hepatic flexure.

Extraperitoneal, which is the ' bare area ' of the liver.

Falciform Ligament.—The two layers are occasionally separated by a collection of pus, which either tracks forwards from the bare area of the liver, or arises from infection around the umbilicus.

Ætiology.—The commonest causes of a subphrenic abscess are appendicitis, a perforated gastric or duodenal ulcer, and leakage or infection after operations upon the stomach. In a series of seventy-eight cases of subphrenic abscess we found that appendicitis was the cause in no fewer than thirty. Although an empyema is frequently associated with a subphrenic abscess, infection of the subphrenic spaces rarely if ever follows an empyema.

Clinical Features.—The symptoms and signs of subphrenic infection are frequently obscure, and it is well to remember the aphorism, " Pus somewhere, pus nowhere else, pus under diaphragm."

Symptoms.—A common history is that when some infective focus in the abdominal cavity has been dealt with, the condition of the patient improves temporarily, but after an interval of a few days or weeks, symptoms of toxæmia reappear. Owing to rapid absorption of toxins, the condition of the patient steadily, and often rapidly, deteriorates. Sweating, wasting, and anorexia are present. There is sometimes epigastric fullness, abdominal discomfort or pain in the shoulder on the affected side, owing to irritation of sensory fibres in the phrenic nerve, referred along the descending branches of the cervical plexus. Persistent hiccup may be a presenting symptom.

Signs.—If the abscess is anterior, abdominal examination will reveal some tenderness, rigidity, or even a palpable swelling. Sometimes the liver is displaced downwards, but more often it is fixed by adhesions. Examination of the chest is important, and in the majority of cases collapse of the lung or evidence of effusion or empyema is to be found.

Accessory Investigations.—(i) *Blood Count*.—A relative and absolute leucocytosis is the rule.

(ii) *X-ray*.—A plain radiograph sometimes demonstrates the presence of gas (fig. 646) or a pleural effusion. On screening, the diaphragm is often seen to be elevated (so-called ' tented ' diaphragm) and its movements impaired.

FIG. 646. — Radiograph showing subphrenic abscess containing gas.

(iii) *Needling*.—Which is the final court of appeal.

If the needle has penetrated a subphrenic abscess, the movements of the diaphragm are transmitted to the needle, which consequently oscillates during respiration. In the case of an empyema these movements are absent. Needling should always be performed in the operating theatre, so that if pus is discovered the needle is left *in situ* as a guide to the abscess, and the operation performed forthwith.

Differential Diagnosis.—Pylephlebitis, tropical abscess, and empyema give rise to most of the diagnostic difficulties.

Treatment.—Many cases of subphrenic infection do not proceed to suppuration, and resolution is encouraged by antibiotic therapy. The clinical course of suspected cases is watched, and blood and radiological examinations are made at suitable intervals. If suppuration seems probable, surgical intervention is indicated.

If a swelling can be detected in the subcostal region or in the loin, an incision is made over the site of maximum tenderness, or over any area where œdema or redness is discovered. The parietes usually form part of the abscess wall, so that contamination of the general peritoneal cavity is unlikely.

If no swelling is apparent, the subphrenic spaces should be explored from behind after removal of the twelfth (and possibly eleventh) rib. The diaphragm is incised below the pleural reflection, and a finger is inserted beneath the diaphragm so as to explore the adjacent area. This method of approach obviates opening either the pleura or peritoneal cavity.

SPECIAL FORMS OF ACUTE PERITONITIS

Meconium Peritonitis.—Meconium is a sterile mixture of epithelial cells, mucin, salts, fats, and bile, and is formed when the fœtus commences to swallow amniotic fluid. By the third month of intra-uterine life the upper third of the small intestine has become filled with meconium ; by the fourth month the accumulation has reached the ileo-caecal valve ; during the remainder of intra-uterine life the colon becomes increasingly filled.

Meconium peritonitis is an aseptic peritonitis which develops late in intra-uterine life or during, or just after, delivery. Meconium enters the peritoneal cavity through an intestinal perforation, and in 50 per cent. of cases the perforation is the result of some form of neonatal intestinal obstruction. In 50 per cent. of cases no cause for the perforation is discernible. When meconium, which is sterile, enters the peritoneal cavity an exudate is secreted that organises rapidly ; matting of intestinal loops occurs, and in many cases in a matter of weeks the extruded meconium becomes calcified.

Meconium remains sterile until about three hours after birth ; thereafter, unless the perforation has become sealed, sterile meconium peritonitis gives place to acute bacterial peritonitis which, unless treated promptly, is rapidly fatal.

Clinical Features.—Meconium peritonitis should always be considered when a baby is born with a tense abdomen. There is vomiting, and failure to discharge meconium. The differential diagnosis between neonatal intestinal obstruction and peritonitis is, in many cases, virtually impossible; indeed, in half the cases both are present. Free fluid in the peritoneal cavity is often sufficient to give a fluid thrill.

Radiography (fig. 647).—Free air in the peritoneal cavity, an abundant quantity of abdominal fluid, fluid levels, calcification (often most distinct on the surface of the liver or the spleen, and most readily seen in a lateral view) are characteristic findings, all of which are unlikely to be present in every case. Meconium peritonitis has been diagnosed by radiography of the fœtus in utero two days before birth.

FIG. 647.—Meconium peritonitis. Free air and fluid in the peritoneal cavity. Intra-abdominal calcification [↖] and on the spleen. Air in the small intestine. Microcolon also shown by a barium enema. (*Dr. Jack Lester, Copenhagen.*)

Treatment.—The prognosis is bad, but recovery has followed prompt operation in a few cases. The greatest chance of survival is in those patients who have an intestinal perforation but no intestinal obstruction, in which case the simple operation of closing the perforation and draining the peritoneal cavity is all-sufficient, and can be performed expeditiously.

Pneumococcal Peritonitis.—There are two forms of this disease :

1. Primary.
2. Secondary to pneumonia.

Primary pneumococcal peritonitis is much the more common. The patient is often an under-nourished girl between three and six years of age, and it is probable that the infection sometimes occurs *via* the vagina and Fallopian tubes, for pneumococci have been cultured from patients' vaginæ. At other times, and always in males, doubtless the infection is blood-borne from the upper respiratory tract or the middle ear. After the age of ten

years pneumococcal peritonitis is most unusual. Children with nephrosis are more liable to this condition than others; possibly a pneumococcal infection was also responsible for the renal condition. During the past thirty years the incidence of pneumococcal peritonitis has declined, perhaps on account of the greater cleanliness and higher standard of living of the poorer classes (Sir Lancelot Barrington-Ward), from which stratum of society patients with this condition are drawn almost exclusively.

Clinical Features.—The onset is sudden, and the earliest symptom is pain localised to the lower half of the abdomen. The temperature is raised to 103° F. (39·8° C.) or more, and there is usually frequent vomiting. Should an inguinal hernia be present it is likely to be distended, but the contents are easily reduced. After twenty-four to forty-eight hours profuse diarrhœa, occasionally blood-stained, is characteristic. There is usually increased frequency of micturition. The last two symptoms are due to severe pelvic peritonitis. Herpes on a lip or nostril is often present. In acute forms of the disease, even in cases where there is no involvement of a lung, there is a tinge of cyanosis of the lips and cheeks, and movement of the alæ nasi is often discernible. On examination rigidity is usually bilateral, and is less than that due to appendicitis.

Differential Diagnosis.—A leucocytosis of 30,000 or more with approximately 90 per cent. polymorphs speaks more for pneumococcal peritonitis than appendicitis. Even so, it is often impossible, especially in males, to exclude with complete certainty perforated appendicitis. The risk of treating expectantly a gangrenous appendix with spreading peritonitis outweighs the risk of a small incision in pneumococcal peritonitis (Sir John Fraser). The other condition which is extremely difficult to differentiate from primary peritonitis in its early stages is pneumonia. An unduly high respiratory rate and the absence of abdominal rigidity are the most important signs supporting the diagnosis of pneumonia, which is usually clarified by a radiograph of the thorax.

Treatment.—When the diagnosis can be made with assurance, the patient should be placed in Fowler's position and nepenthe prescribed in suitable doses, according to age. Intravenous dextrose-saline is administered by the drip method, and penicillin is given in doses of 500,000 units *bis die*. Oxygen therapy is valuable, especially in cases where even faint cyanosis is discernible. Under such treatment resolution often occurs, or after at least fourteen days a localised abscess forms which will require drainage.

Early operation is required when the condition cannot be distinguished from acute appendicitis with peritonitis. Under local infiltration anæsthesia only, if the child's condition is poor, a vertical incision not more than 1 inch (2·5 cm.) in length is made over the middle line of the right rectus abdominis, the fibres of which are split. The peritoneum is incised. Should the exudate be odourless and sticky, the diagnosis of pneumococcal peritonitis is practically certain, but it is desirable to obtain immediate proof without disturbing the intestines. Some of the exudate is removed with a syringe, and

Sir Lancelot Barrington-Ward, 1884–1953. Surgeon, Hospital for Sick Children, London.
Sir John Fraser, 1885–1947. Surgeon, Royal Edinburgh Hospital for Sick Children.

an assistant is in readiness to stain a smear and examine it microscopically. If pneumococci or streptococci are identified, a cigarette drain is passed into the pelvis (fig. 648). The patient is returned to bed and the treatment detailed above is continued. The remainder of the specimen of the exudate is sent to the laboratory for culture and antibiotic sensitivity tests.

FIG. 648.—Soft cigarette drain passed through the wound into the pelvis. Sutures are unnecessary. (*Ladd's technique.*)

In severe cases, in the later stages of treatment, small repeated blood transfusions are given to bring the hæmoglobin level to 100 per cent.

Primary streptococcal peritonitis of infants and children is rather more frequent than the foregoing. When a streptococcus is the infecting organism, the peritoneal exudate is thin, slightly cloudy, and contains flecks of fibrin. From the standpoints of clinical aspects and treatment, streptococcal peritonitis in infants and children does not differ from those detailed in the account of pneumococcal peritonitis (*vide supra*), but the mortality is higher.

Idiopathic streptococcal peritonitis in adults is fortunately rare, for prior to the antibiotic era it was nearly always fatal, and the mortality is still very high. Rightly, in early cases the abdomen is opened, usually on a diagnosis of acute appendicitis. In streptococcal peritonitis the peritoneal exudate is odourless, thin, contains small flecks of fibrin, and may be blood-stained. In these circumstances pus is removed by suction, the abdomen closed with suprapubic drainage, and the measures detailed in the conservative treatment of peritonitis carried out.

Peritonitis following Parturition or Abortion.—When peritonitis follows puerperal infection, it is a notifiable disease. It is more common after first deliveries. Rigidity is seldom much in evidence; this, at any rate in part, is due to the stretched condition of the abdominal musculature. The lochia may be offensive but not necessarily so. Diarrhœa is common.

Provided the infection is limited strictly to the pelvis, the Ochsner-Sherren régime (see Chap. 27) followed, when necessary, by posterior colpotomy, is eminently successful. In cases where the peritonitis is not so limited, it is the considered opinion of those who have had much experience of this condition that operation should be performed as soon as possible.

Operation.—After adequate resuscitative measures and antibiotic therapy have been carried out, the abdomen is opened by a right paramedian incision. When there is an abscess in the wall of the uterus or the broad ligament, after packing off the area the abscess is opened and, if large enough, drained by a tube relegated for that purpose. The peritoneal cavity should be drained by *two* drainage tubes passed into the pelvis, one in front of the uterus and the other into the recto-uterine pouch (pouch of Douglas).

In the pre-antibiotic era the mortality of general peritonitis following parturition or abortion was at least 50 per cent.; with antibiotic therapy and timely operation, when necessary, the mortality has fallen to less than 10 per cent. (R. A. Brews).

Post-operative Peritonitis.—It is hard to decide whether the signs are

James Douglas, 1675–1742. Physician to Queen Caroline. Practised in Red Lion Square, London.
Richard Alan Brews, Contemporary. Obstetric and Gynæcological Surgeon, The London Hospital.

due to operative trauma or to infection. Rigidity, one of the mainstays of the recognition of other forms of peritonitis, is frequently in abeyance. Tenderness, though present, is likely to be attributed to the recent incision. More likely than not a narcotic has been administered and this, of course, masks the signs, indefinite as they are. Absence of bowel sounds, abdominal distension (which is slight in fulminating cases) and the bile-stained gastric aspirate will almost certainly be thought to be due to paralytic ileus. There is often evidence of post-operative pneumonia, to which can be attributed the rise in pulse and temperature.

Peritonitis after abdominal operations occurs more often than is recorded. The principles of treatment in no wise differ from those of peritonitis of other origin. Antibiotic therapy *per se* is insufficient; no antibiotic can stay the onslaught of bacterial peritonitis due to leakage from a suture line.

Periodic peritonitis is characterised by abdominal pain and tenderness, mild pyrexia, polymorphonuclear leucocytosis, and occasionally pain in the thorax and joints. The duration of an attack is twenty-four to seventy-two hours, when it is followed by complete remission, but exacerbations recur at regular intervals. Most of the patients have undergone appendicectomy in childhood. The disease, often familial, is limited principally to Arabs, Armenians, and Jews; other peoples occasionally are affected. At laparotomy, which may be necessary to exclude other causes, the peritoneum—particularly in the vicinity of the spleen and the gall-bladder—is inflamed. There is no evidence that the interior of these organs is abnormal.

Differential Diagnosis.—Patients with abdominal epilepsy do not have physical signs or pyrexia, and their attacks are usually controlled by anti-convulsive medication.

The ætiology of periodic peritonitis is unknown, and no form of treatment has been found to be of the slightest avail.

Bile Peritonitis.—Unless there is reason to suspect that a bile duct was damaged at an operation in which drainage was not provided, it is improbable that bile as a cause of peritonitis will be thought of until the abdomen has been opened and bile is seen therein. The causes of bile peritonitis are as follows:

As a cause of an acute intra-abdominal catastrophe	*Following an abdominal accident*	*Following an abdominal operation*
1. Perforation of a gall-bladder.	1. Rupture of the gall-bladder.	1. Operative damage to the biliary tree during cholecystectomy.
2. Perforation of a bile duct.	2. Rupture of a main bile duct.	2. Leakage from a cut accessory bile duct.
3. Rupture of a choledochus cyst.	3. Rupture of the second part of the duodenum.	3. Accidental dislodgment of a drainage tube placed in the common bile duct.
		4. A main bile duct cut during gastroduodenal resection.

Unless the bile is extravasated slowly, and the collection becomes shut off from the general peritoneal cavity, even if the bile was sterile at the time of extravasation, in addition to signs of diffuse peritonitis, severe toxæmia is

often in evidence. After a few hours a tinge of jaundice is not unusual. Local drainage, and when necessary suprapubic peritoneal drainage, is imperative, and if performed early enough, these measures will save the patient's life. When bile is seen issuing from a perforation of some part of the biliary tree, a drainage tube should be passed through the opening and secured there. A ruptured duodenum must, of course, be repaired.

TUBERCULOUS PERITONITIS

Acute Tuberculous Peritonitis.—It is doubtful if tuberculous peritonitis is ever acute. However, tuberculous peritonitis sometimes has an onset that resembles so closely acute peritonitis that the abdomen is opened. Straw-coloured fluid escapes, and tubercles[1] are seen scattered over the peritoneum and great omentum. Tubercles occasionally simulate fat necroses (see p. 462) or the nodules of peritoneal carcinomatosis (see p. 500). On opening the abdomen and finding tuberculous peritonitis, the fluid is evacuated, a portion of the diseased omentum is removed for histological confirmation of the diagnosis, and the wound closed without drainage.

At other times, although acute abdominal symptoms arise, the presence of ascites makes the diagnosis of acute tuberculous peritonitis tolerably evident.

Chronic Tuberculous Peritonitis.—Although the incidence of tuberculous peritonitis has declined in Britain, in many parts of the world where measures for preventing tuberculosis are enforced less strictly, the condition is still common.

Usually children are affected, but it is not rare for the disease to make its first appearance in early adult life, when females outnumber males by two to one. Exceptionally the disease becomes manifest in patients over forty years of age.

Origin of the infection:

1. From tuberculous mesenteric lymph nodes.
2. From tuberculosis of the ileo-cæcal region.
3. From a tuberculous pyosalpinx.
4. Very occasionally it is due to a blood-borne infection from pulmonary tuberculosis.

There are four varieties of tuberculous peritonitis :

1. **Ascitic Form.**—The peritoneum is studded with tubercles, and the peritoneal cavity becomes filled with pale, straw-coloured fluid. The onset is insidious. There is loss of energy, facial pallor, and some loss of weight. The patient is usually brought for advice because of enlargement of the abdomen (fig. 649). Pain is often completely absent ; in other cases there is considerable abdominal discomfort which may be associated with constipation or diarrhœa. On inspection dilated veins can be seen coursing beneath the skin of the abdominal wall. Shifting dullness can be elicited readily. In the male child congenital hydroceles sometimes appear, due to the patent tunicæ vaginales becoming filled with ascitic fluid from the peritoneal cavity. Because of the increased intra-abdominal pressure,

FIG. 649.—Tuberculous peritonitis, ascitic form. The patient has also left-sided tuberculous epididymitis. *(Mr. F. H. Robarts, Edinburgh.)*

[1] Early tubercles are greyish and translucent. They soon undergo caseation, and then appear white or yellow, and are less difficult to distinguish from carcinoma.

an umbilical hernia commonly occurs. On abdominal palpation a transverse solid mass can often be detected (fig. 650). This is rolled-up great omentum infiltrated with tubercles.

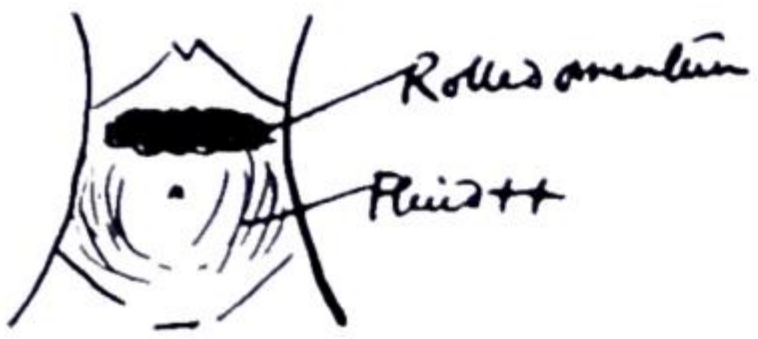

FIG. 650.—Physical signs recorded in a case of the ascitic form of tuberculous peritonitis.

The diagnosis is seldom difficult, except when it occurs in an acute form or when it first appears in an adult, in which case it has to be differentiated from other forms of ascites. A positive Mantoux test in a child with ascites suggests strongly, and a negative test is good evidence against, tuberculosis. In adults this test is of negligible value. The diagnosis of tuberculous peritonitis having been made, it is always important to endeavour to determine if there is tuberculous disease elsewhere, and in this respect the possibility of tuberculous salpingitis in women should be remembered.

Treatment.—Sanatorium treatment, which includes helio- and antibiotic therapy, is usually effective. Evacuation of ascitic fluid is often followed by considerable though sometimes temporary, improvement, probably due to an outpouring of fresh ascitic fluid more plentiful in antibodies : in some institutions it is the practice to evacuate fluid by paracentesis and replace it by oxygen.

Antibiotic Therapy.—If under the age of thirty years the patient is given streptomycin, 1 G. daily intramuscularly, together with isoniazid by mouth, 100 mg. twice a day. If over the age of thirty, it is probably wise, owing to the risk of eighth nerve involvement from streptomycin, to give 1 G. streptomycin three times a week with daily isoniazid in doses already mentioned, and sodium P.A.S. by mouth in doses of 5 G. twice daily. As a rule the treatment is continued for a minimum of six months, after which the P.A.S. and isoniazid are continued for at least another three months. The patient is treated with bed rest until the sedimentation rate has reached normal.

In favourable cases under the triple treatment of (*a*) open air day and night, (*b*) heliotherapy, and (*c*) antibiotic treatment, the ascitic fluid is absorbed gradually, there is a steady gain in weight, and eventual recovery.

If the general condition is good, the patient can return home and, if an adult, to light work, before the course of oral medication has been completed.

2. **Encysted** (*syn.* **loculated**) **form** is similar to the above, but one part of the abdominal cavity alone is involved. So is produced a localised intra-abdominal swelling which gives rise to difficulty in diagnosis. In a female above the age of puberty, when the swelling is in the pelvis, an ovarian cyst will probably be diagnosed. In the case of a child it is sometimes difficult to distinguish the swelling from a mesenteric cyst. For these reasons laparotomy is often performed, and if an encapsulated collection of fluid is found, it is evacuated and the abdomen is closed. The general treatment already detailed is required, but the response to this treatment is more rapid. Late intestinal obstruction is a possible complication.

3. **Fibrous** (*syn.* **plastic**) **form** is characterised by the production of widespread adhesions, which cause coils of intestine, especially the ileum, to become matted together and often adherent to the parietal peritoneum. Typically the condition is accompanied by wasting and attacks of abdominal pain. On examination the adherent intestine with omentum attached, together with the thickened mesentery, may give

Charles Mantoux. 1877-1947 Physician, Le Cannet, France.

rise to a palpable swelling or swellings. The first intimation of the disease may be subacute or acute intestinal obstruction. Sometimes the cause of the obstruction can be remedied easily by the division of bands; more often it can only be overcome by lateral anastomosis between an obviously dilated loop and a collapsed loop of small intestine, or by ileo-colostomy. So great is the danger of recurring attacks of intestinal obstruction and eventual death from this cause that in many cases, during a quiescent period, the involved small intestine should be plicated (see Chapter 27). If the obstruction does not occur or can be overcome in spite of the intraperitoneal chaos, the prognosis is fairly good.

4. **Purulent form** is rare. When it occurs, usually it is secondary to tuberculous salpingitis. Amidst a mass of adherent intestine and omentum, tuberculous pus is present. Sizeable cold abscesses are wont to form, and point on the surface, commonly near the umbilicus, or burst into the bowel. In addition to prolonged general treatment, operative treatment may be necessary for the evacuation of cold abscesses and possibly for intestinal obstruction. If the patient survives long enough to overcome the infection, it may be possible to close a fæcal fistula, which otherwise usually persists because of obstruction distal to it. Closure must therefore be combined with some form of anastomosis between the segment of intestine above the fistula and an unobstructed area below. The prognosis of this variety of tuberculous peritonitis is poor.

PERITONEAL BANDS AND ADHESIONS

Congenital bands and membranes must be distinguished from inflammatory peritoneal adhesions. From time to time the following congenital bands and membranes are encountered:

1. *The cystico-duodenal band* runs from the gall-bladder to the pylorus.
2. *The mesocolic band of Pringle* passes from the mesocolon to the duodeno-jejunal flexure.
3. A congenital band attaching the ileum 4 inches (10 cm.) from the ileo-cæcal junction to the right ovary or the posterior abdominal wall.
4. *Jackson's membrane*, which is comparatively common, runs from the great omentum, enveloping like a shroud an unusually lax, capacious cæcum and ascending colon. When this veil is gently pulled so as to make it move upon the cæcum, its contained blood-vessels become the more apparent (fig. 651). It is attached to the abdominal wall of the right flank and must be divided before the cæcum can be exposed.
5. *Payr's membrane* is a similar structure over the splenic flexure.
6. *Toldt's membrane* is found over the pelvic colon.
7. An obliterated vitello-intestinal duct (see Chapter 29).

None of the above bands and adhesions cause acute intestinal obstruction, except the last.

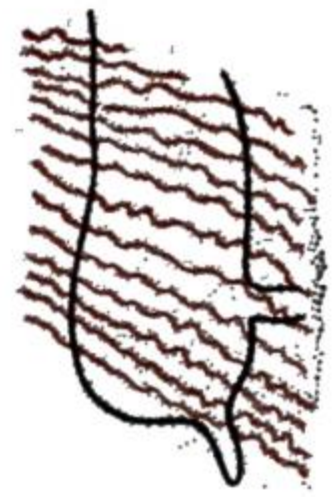

FIG. 651.—Jackson's membrane.

Inflammatory Peritoneal Adhesions.—As a result of peritonitis, adhesions of varying density occur within the peritoneal cavity; as a rule they are more numerous and more dense after peritonitis has been treated by laparotomy, and especially when tube drainage has been employed, than when peritonitis has been treated by the delayed method (see p. 485).

It is not unusual for the greater omentum or a coil of intestine to become adherent to a laparotomy scar. There is little doubt that such adhesion is the result of mild infection. It should be noted that experimental and operative experience dictates that a normal structure will become lastingly adherent to an inflamed one only if the former itself becomes inflamed.

Following Grieg Smith's premise that a serous surface will adhere regularly to a raw surface, there is a widespread belief that raw areas left unperitonealised are the cause of dense adhesions; this is untrue. It has been shown that after complete excision of the pelvic viscera, where no attempt can be made to reperitonealise the pelvic floor, and the small intestine descends into the pelvis and comes in contact with the musculo-

Seton Pringle, 1879–1955. Surgeon, Royal City of Dublin Hospital, Ireland.
Jabez N. Jackson, 1868–1935. Surgeon, Kansas City, U.S.A.
Ewin Payr, 1871–1946. Professor of Surgery, Leipzig.
Carl Toldt, 1840–1920. Professor of Anatomy, University of Vienna.
James Grieg Smith, 1834–1897. Surgeon, The Royal Infirmary, Bristol.

fascial and osseous surfaces of the parietes, adhesions are unusual. Therefore it must be taken for granted that infection, or the irritation produced by glove powder or local bacteriostatic agents, always precede adhesions.

Peritoneal adhesions are a frequent cause of intestinal obstruction. Often the obstructing agent proves to be a single adhesion, sometimes surprisingly thin, perhaps only the thickness of fine sewing cotton. Recent adhesions are separated easily. Old-standing adhesions, tough and sometimes vascular, require division with a scalpel. The resulting raw areas, if circumscribed, can sometimes be buried; otherwise they should be covered with omental patches for, contrary to what has been explained above, given an opportunity one raw surface will adhere readily to another raw surface.

Talc Granuloma.—Talc (silicate of magnesium) should never be used as a lubricant for rubber gloves for it is a cause of peritoneal adhesions and granulomata in the Fallopian tubes. Potassium bitartrate, which is completely soluble, is free from these serious objections.

Lycopodium spores in glove dusting-powder are also a cause of peritoneal adhesions, and sometimes the spores produce multiple nodules on the peritoneal surface which simulate those occurring in carcinoma peritonei or tuberculous peritonitis.

ASCITES

Ascites, an excess of serous fluid within the peritoneal cavity, can be recognised clinically only when the amount of fluid present exceeds 1,500 ml.; in the obese a greater quantity than this is necessary before there is clear evidence of the presence of intraperitoneal fluid. Although ascites is a symptom, and not a disease, it is convenient and instructive to consider the common causes of the condition together. Experimentally it has been shown that partial ligation of the thoracic portion of the inferior vena cava causes ascites, whereas similar ligation of the portal vein or the abdominal portion of the inferior vena cava fails to produce ascites. This observation proves that congestion of the liver is a factor in the production of ascites; indeed, systemic venous congestion is the only source of mechanical ascites.

FIG. 652.—The absence of any cause for ascites made the diagnosis of ovarian cyst probable in this case. Diagnosis confirmed by operation.

Clinical Features.—The abdomen is distended evenly, with fullness of the flanks, which are dull to percussion. Usually shifting dullness is present, but when there is a very large accumulation of fluid, this sign is absent. In such cases, on flicking the abdominal wall, a characteristic fluid thrill is transmitted from one side to the other. In the female, ascites must be differentiated from an enormous ovarian cyst (fig. 652).

Type 1. *Due to Congestive Heart Failure.*—This, the commonest cause of ascites, is due to chronic venous stasis in the thoracic segment of the inferior vena cava, and consequent obstruction to the venous outflow from the liver. There is stasis also in the superior vena cava, as evinced by engorgement of the veins of the neck—a

striking sign in this condition. The ascitic fluid is a light yellow serum of low specific gravity, about 1010.

Type 2. *Due to Hepatic Cirrhosis.*—In cirrhosis of the liver there is obstruction to the venous outflow of the liver due to obliterative fibrosis of the intrahepatic venous bed. Enlargement (later contraction) of the liver, enlargement of the spleen, spider nævi, and possibly a history of alcoholism are often in evidence. The serum is darker in colour than the foregoing, and the specific gravity is about 1·015.

Type 3. *Due to Tuberculous Peritonitis.*—In this instance the ascites is due to excessive outpouring of peritoneal fluid consequent upon inflammation of the endothelial lining of the peritoneum. The fluid is pale yellow, usually clear, and rich in lymphocytes. The specific gravity is comparatively high, often 1·020 or over. Even after centrifugation, rarely can the *mycobact. tuberculosis* be found, but its presence can be demonstrated by culture or by guinea-pig inoculation.

Type 4. *Secondary Carcinoma of the Peritoneum.*—Again the ascites is due to excessive outpouring, this time due to irritation of the peritoneum by the parasitic neoplastic cells. The fluid is dark yellow and frequently blood-stained. The specific gravity is high—1·020 or over. Microscopical examination often reveals cancer cells.

Type 5. *Chronic Constrictive Pericarditis* (syn. *Pick's Disease*).—In addition to the peritoneal effusion, effusions occur into the pleural cavity. These effusions are due to engorgement of the venæ cavæ consequent upon diminished capacity of the right side of the heart.

Type 6. *Due to depletion of blood protein* consequent upon albuminuria or starvation. The ascites in this instance is due to alterations in the osmotic pressure of the capillary blood.

Type 7. *Due to profound anæmia.*

Type 8. *Transient Ascites.*—Occasionally no cause for the ascites can be found, and after paracentesis it does not re-form. Probably such cases of benign ascites, as it is sometimes called, are due to portal hypertension that is rectified by naturally-occurring portacaval shunt (see Communicating Channels between the Portal and Systemic Venous Channels, p. 416).

Treatment.—Repeated tappings of the ascitic fluid leads to loss of valuable protein, and tends to induce low serum sodium levels as the fluid re-accumulates. Particularly when ascites is due to a non-malignant condition, medical treatment is frequently successful. Dietary sodium restriction to 200 mg. per day will usually control ascitic fluid formation, and will sometimes lead to prompt diuresis and abatement of the ascites. Many patients, however, do not have prompt diuresis and loss of ascites when such a régime is instituted, but continue to harbour ascitic fluid which, however, does not increase in amount. In selected cases an effort is made to increase its excretion with mercurial diuretics used with great care. If medical treatment is unsuccessful, paracentesis (tapping) becomes necessary.

Paracentesis Abdominis.—The bladder having been emptied by a catheter, under local anæsthesia puncture of the peritoneum is carried out with a moderate-sized trocar and cannula at one of the points shown in fig. 653. In cases where the

Friedel Pick, 1867–1926. Director of the Laryngological Institute, Prague.

effusion is due to cardiac failure the fluid must be evacuated slowly. In other circumstances this precaution is unnecessary. If the cannula becomes blocked with fibrin, it is cleared with a probe. After the fluid has been evacuated the puncture is sealed, and a tight binder is applied to the abdomen.

FIG. 653.—Usual points of puncture for tapping ascites. The bladder must be emptied by a catheter before the puncture is made. Note the relationship of the sites of puncture to the deep epigastric artery.

Permanent Drainage of Ascitic Fluid into the Urinary Bladder.—In some cases where ascites accumulates rapidly after paracentesis, drainage of the ascitic fluid into the bladder renders the patient more comfortable. Paracentesis is done on the operating table, the bladder is then distended with fluid, and the peritoneal cavity is opened in the midline of the hypogastrium. A long whistle-tipped catheter, with two or three lateral apertures cut in the upper third to provide extra drainage, is anchored to the peritoneum, and the butt end of the catheter is passed to the right flank. The lower end of the catheter is threaded through a small puncture in the lowest part of the peritoneum and the main peritoneal incision is closed. A tiny puncture is made in the bladder, and the eyed end of the catheter is introduced for 1½ inches (3·8 cm.). The catheter is fastened to the bladder, and the bladder is anchored to the sheath of the rectus. The incision is closed. A self-retaining catheter is kept in place for ten days.

PERITONEAL LOOSE BODIES

Peritoneal loose bodies almost never cause symptoms. Not infrequently one or more are found in a hernial sac. The probable origin of a peritoneal loose body is an appendix epiploica that has undergone axial rotation, followed by necrosis of its pedicle, and detachment. These hyaline bodies rarely attain the size of a pea. A large loose body has caused retention of urine by becoming impacted in the pelvis.

NEOPLASMS OF THE PERITONEUM

Carcinoma peritonei is a common terminal event in many cases of carcinoma of the stomach, colon, ovary, or other intraperitoneal organ and also of the breast. The peritoneum, both parietal and visceral, is studded with secondary growths, and the peritoneal cavity becomes filled with clear, straw-coloured, or blood-stained ascitic fluid.

The main forms of peritoneal metastases are :

1. Discrete nodules (fig. 654). This is by far the most common variety.
2. Plaques varying in size and colour.
3. Diffuse adhesions. This form occurs at a late stage of the disease, and gives rise, for instance, to a 'frozen pelvis.'
4. Pseudo-myxoma peritonei.

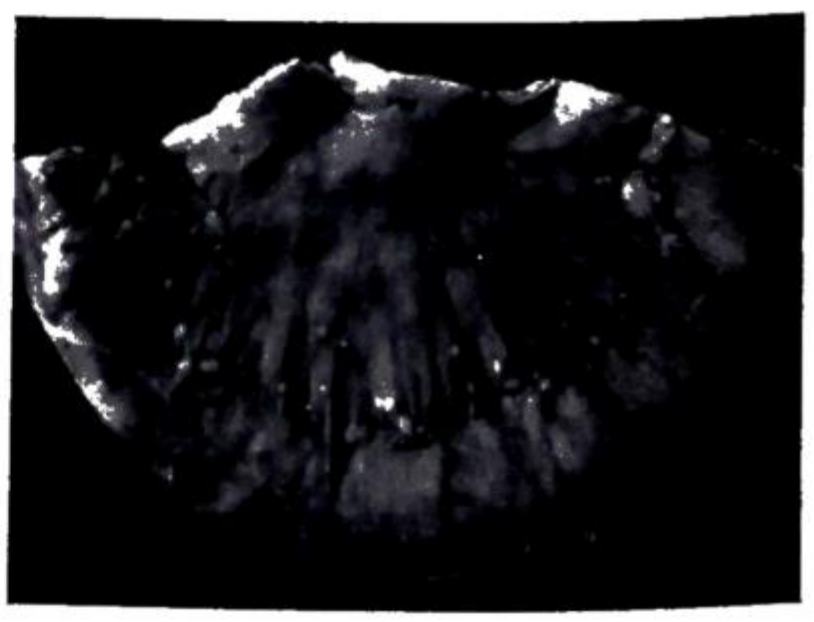

FIG. 654.—Peritoneal metastatic nodules secondary to carcinoma of the pelvic colon. (*Mr. Owen Daniel, Sheffield.*) (*British Journal of Surgery.*)

Gravity probably determines the distribution of free malignant cells within the peritoneal cavity. Cells not caught in peritoneal folds along the attach-

ments of mesenteries gravitate into the pelvic pouches or into a hernial sac, enlargement of which is occasionally the first intimation of the condition. Implantation occurs also on the greater omentum, the appendices epiploicæ, and the inferior surface of the diaphragm.

Differential Diagnosis.—Early discrete tubercules common in tuberculous peritonitis are greyish and translucent, and closely resemble the discrete nodules of peritoneal carcinomatosis, but the latter feel hard when rolled between the finger and the thumb, making the differential diagnosis tolerably simple. Fat necroses usually can be distinguished from carcinomatous nodules by their opacity. Peritoneal hydatids can also simulate malignant disease after rupture of a hydatid cyst.

It is remarkable how often a patient riddled with intraperitoneal carcinoma preserves her nutrition, and looks and feels comparatively well until the terminal stage.

Treatment.—Ascites due to carcinomatosis of the peritoneum can often be considerably ameliorated by instillations of radio-active gold.

Radio-active gold (^{198}Au) has a half-life of two and a half days, and is supplied as a purple colloidal solution. One hundred millicuries or more of the solution are introduced into the peritoneal cavity after paracentesis. To improve distribution the foot of the bed is raised ; then the patient lies on one side and then on the other, and finally prone, each for fifteen minutes. There follows a period of nausea, but approximately half the patients so treated are benefited for a time. The treatment is of no avail if, after paracentesis, the secondary deposits can be palpated, as the isotope can only penetrate to a depth of 1 millimetre.

Radio-active chromic phosphate (^{32}P) is also supplied as a colloid solution, and is as effective as gold. Not only is it less expensive, but this solution requires none of the troublesome precautions connected with the protection of personnel. It emanates pure beta radiation, and has a longer half-life, viz. 14·3 days. In its administration rubber gloves are the only protection needed.

Sixty per cent. of patients are improved by treatment with these isotopes.

Pseudo-myxoma Peritonei.—This rare condition occurs more frequently in females. The abdomen is filled with a yellow jelly, large quantities of which are often more or less encysted. The condition arises in one of two ways. More often from rupture of a pseudo-mucinous cyst of the ovary not otherwise malignant ; less often from rupture of a mucocele of the appendix. The condition is painless, and there is no impairment of general health for a long time. When the condition arises from the appendix the mass is often more localised, but in cases of ovarian origin the whole peritoneal cavity is involved. Although an abdomen distended with what seems to be fluid that cannot be made to shift should suggest the possibility, it is highly improbable that a correct pre-operative diagnosis will be made. At laparotomy masses of jelly are scooped out, and the primary focus, if it can be found, is removed. Unfortunately, recurrence is usual. In one of our patients laparotomy and evacuation of quarts of jelly was undertaken on four occasions in a little over a year. When pseudo-myxoma peritonei arises from a mucocele of the appendix, repeated recurrence is less common. Pseudo-myxoma peritonei is locally malignant, but does not give rise to metastases. Occasionally the condition responds to radio-active isotopes, which certainly should be employed in recurrent cases.

PERITONEOSCOPY

For many years peritoneoscopy has been advocated for inspecting intraperitoneal organs without the necessity of laparotomy, but comparatively few surgeons employ it. The chief value of the method is to visualise the available portion of the liver in cases of hepatomegaly of undetermined origin. It use has been extended to inspect the gall-bladder and the stomach, as also the uterus and anexæ, in subjects in whom laparotomy is inadvisable.

The peritoneoscope resembles a cystoscope. The abdomen is prepared as for laparotomy. Preferably under general anæsthesia, an incision ¼ inch (6 mm.) long is

made in the middle line close to the umbilicus. A small trocar and cannula are pushed into the peritoneal cavity and the requisite amount of air (1 to 1½ litres) is introduced with a pneumoperitoneum apparatus. The patient is tilted so that the air will rise into the upper abdomen or the pelvis, as required. A special trocar and cannula is then introduced, the trocar withdrawn, and the telescope inserted. Biopsy of a particular area of the liver is possible, but hæmorrhage from it is not infrequent. Air embolism is another possible danger.

THE GREATER OMENTUM

Rutherford Morison called the greater omentum 'the abdominal policeman.' Relatively larger and structurally more substantial in the adult than

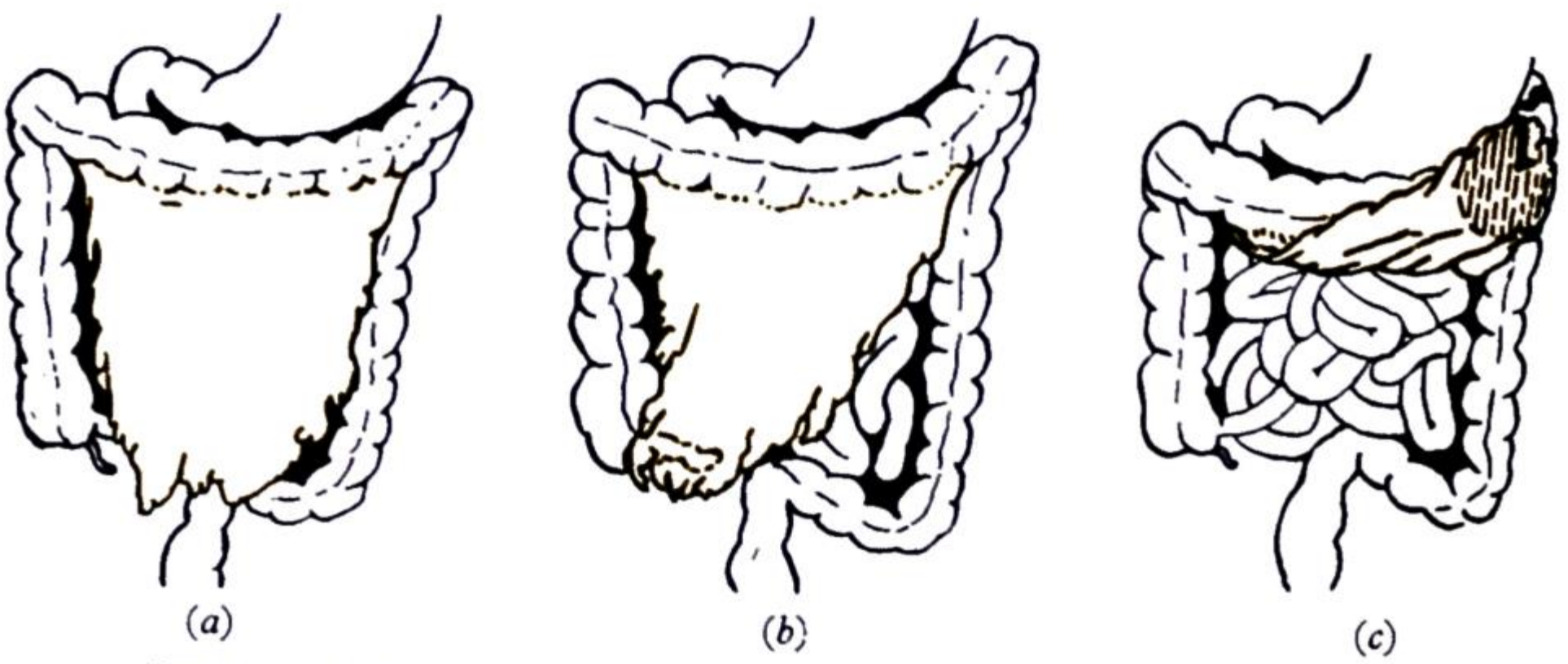

FIG. 655.—The greater omentum. (*a*) Normal. (*b*) In appendicitis. (*c*) In a (comparatively small) laceration of the spleen.

in the child, the discharge of its life-saving constabulary duties becomes more effective after puberty, and remains unabated throughout life. The greater omentum attempts, often successfully, to limit intraperitoneal infective and other noxious processes (fig. 655). For instance, an acutely inflamed appendix is often found wrapped in omentum, and this saves many a patient from developing diffuse peritonitis. Sufferers from herniæ are also greatly indebted to this structure, for it often plugs the neck of a hernial sac and prevents a coil of intestine from becoming strangulated.

Apart from a small portion of it becoming gangrenous while performing the last-mentioned duty (strangulated omentocele), this good Samaritan of the peritoneal cavity seldom itself becomes diseased; when it does become overwhelmed, as in tuberculous peritonitis and carcinomatosis peritonei, it becomes rolled like a scroll.

FIG. 656.—Torsion of the greater omentum. Specimen removed by operation. (*Mr. Archibald Ronald, Barrow-in-Furness.*)

Torsion of the Omentum.—Torsion of the omentum (fig. 656) is a rare emergency, and consequently is seldom diagnosed correctly. It is usually mistaken for appendicitis with somewhat abnormal signs. It may be primary or secondary to an adhesion of the omentum, to an old

Rutherford Morison, 1853–1939. Professor of Surgery, University of Durham.

focus of infection, or to a hernia. Not infrequently patients who have suffered from torsion of the omentum have had an external hernia. Successive herniations of a portion of the omentum into a hernial sac of irregular bore are credited with giving the necessary impetus to omental torsion in a manner similar to that of a bullet travelling along the barrel of a rifle.

The patient is most frequently a middle-aged, obese male, but a number of cases have occurred during childhood. A tender lump may be present in the abdomen. The blood supply having been jeopardised, the twisted mass sometimes becomes gangrenous, in which case bacterial peritonitis soon follows.

Treatment.—The abdomen having been opened, the pedicle above the twist is ligated securely and the mass removed.

Omental Cyst (see p. 509).

A wound of the mesentery can follow a severe abdominal contusion, and is a cause of hæmo-peritoneum. In about 60 per cent. of cases the mesenteric laceration is associated with a rupture of the intestine. If the tear is a large one, and especially if it is transverse (fig. 657), the blood

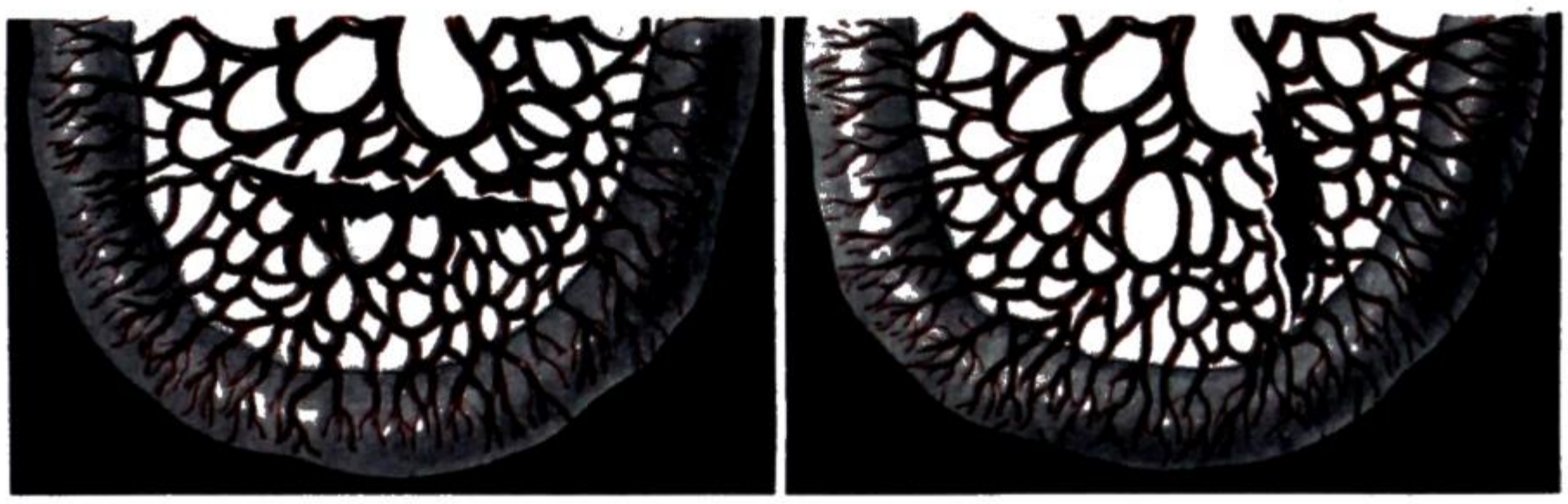

FIG. 657. FIG. 658.

Laceration of the mesentery. A transverse tear (fig. 657) often imperils the blood supply of a segment of intestine, making resection necessary. A longitudinal tear (fig. 658) can be closed by suture.

supply to the neighbouring intestine is cut off, and a limited resection of gut is imperative. Small wounds and wounds in the long axis (fig. 658) should be sutured.

Torsion of the Mesentery (see Volvulus Neonatorum, Chapter xxvi, and Volvulus of the Small Intestine, Chapter xxvi).

Embolism and Thrombosis of the Mesenteric Vessels (see Chapter xxvi).

ACUTE NON-SPECIFIC MESENTERIC ADENITIS

Ætiology.—Non-specific mesenteric adenitis was so named in 1920, to distinguish it from specific (tuberculous) mesenteric adenitis. Despite much investigation, the ætiology remains unknown. As so often happens in other inflammatory diseases when no causative bacterium can be found, an unidentified virus is blamed. In about 25 per cent. of cases a respiratory infection precedes an attack of acute non-specific mesenteric adenitis. In spite of the fact that the vermiform appendix is not diseased in this condition, which is definitely recurrent, appendicectomy does reduce the incidence of further attacks, perhaps by removing what is sometimes known as 'the abdominal tonsil.' This self-limiting disease is never fatal.

Living Pathology.—There is a small increase in the amount of peritoneal fluid. As seen and felt between the leaves of the mesentery, the mesenteric lymph nodes are enlarged, being firmly elastic and usually about the size of a haricot bean. In very acute cases they are distinctly red, and many of them are the size of a walnut. The nodes nearest the attachment of the mesentery are the largest. The nodes are not adherent to their peritoneal coats, and if a small incision is made through the overlying peritoneum, a node is extruded easily. The adenitis is most in evidence in the lower third of the mesentery.

Clinical Features.—During childhood, acute non-specific mesenteric adenitis is a common condition, the ratio of acute appendicitis to acute non-specific adenitis being about 3 : 1. The age incidence of the disease is shown in fig. 659. While the condition has been recorded in adults, so rare is it after the age of puberty that it is highly improbable that the diagnosis is even entertained pre-operatively in those past school age. On the other hand, frequently it is possible to arrive at the correct pre-operative diagnosis in children, although admittedly often it is difficult to rule out the possibility of anomalous acute or subacute appendicitis. The typical history is one of attacks of abdominal colic, varying in severity. In 30 per cent. of cases the patient has experienced similar, less severe attacks lasting a few days within the past few months or a year. Vomiting is usual, but there is no characteristic alteration of bowel habit.

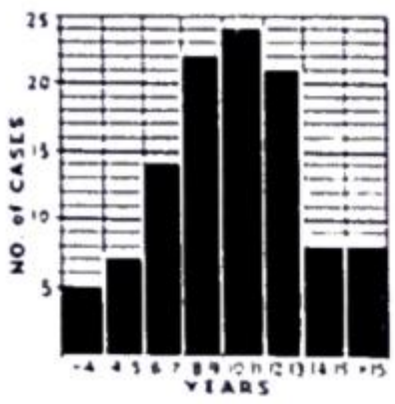

FIG. 659.—The age incidence in 109 cases of acute non-specific mesenteric adenitis. (*After M. J. T. Fitzgerald.*)

On Examination.—There are severe spasms of general abdominal colic, usually referred to the umbilicus, with intervals of complete freedom, which never appertains in obstructive appendicitis. The patient seldom looks ill. In more than half the cases the temperature is elevated; in very acute examples it exceeds 101° F. (38·3° C.). Extremely characteristic is a malar flush, which is present irrespective of pyrexia. The site of maximum tenderness is shown in fig. 660. When present, shifting tenderness is a valuable sign for differentiating the condition from appendicitis. After lying the patient on the left side for a few minutes, the maximum tenderness moves to the left of the original site (Klein's sign) : it must however be realised that a positive Klein's sign does not rule out the possibility of acute Meckelian diverticulitis.

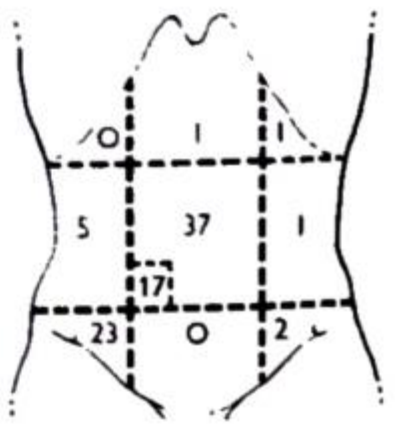

FIG. 660.—Sites of maximum tenderness in acute non-specific mesenteric adenitis. (*After M. J. T. Fitzgerald.*)

The enlarged lymph nodes are palpable in about 30 per cent. of cases if deep bimanual palpation is employed. The pelvic peritoneum is tender to rectal palpation in a like number of cases. The neck, axillæ, and groin should be palpated for enlarged lymph nodes—if these nodes are enlarged, brucellosis should come to mind (see p. 506).

Leucocyte Count.—There is a leucocytosis of 15,000 or more on the first day of the attack, but this falls on the second day.

William Klein, Contemporary. Consulting Surgeon, Morrisania City Hospital, New York.
Johann Meckel, 1781–1833. Professor of Botany, Anatomy, and Obstetrics, Berlin.

Treatment.—When the diagnosis can be made with assurance, bed rest for a few days is the only treatment necessary. If at a second examination acute appendicitis cannot be excluded, it is safer to perform appendicectomy.

SUPPURATING MESENTERIC LYMPH NODES

It would seem likely that infection of the mesenteric lymph nodes by enterococci and other intestinal organisms occurs rather frequently, but suppuration of these nodes is comparatively rare. When a palpable abscess develops from a breaking-down mesenteric lymph node or nodes it is likely to be diagnosed as an appendix abscess ; more often while such an abscess is small, coils of intestine become adherent to it and the signs are those of subacute intestinal obstruction. On other occasions the contents of the abscess burst into the peritoneal cavity and diffuse peritonitis results. The treatment is to drain the abscess by the most direct route. In cases where an abscess has burst, drainage of the peritoneal cavity, if performed early, is usually successful.

TUBERCULOSIS OF THE MESENTERIC LYMPH NODES

Although still fairly common, tuberculous mesenteric lymphadenitis is considerably less common than acute non-specific lymphadenitis, and it has become increasingly less frequent in Britain during the past thirty years. Tubercle bacilli, usually but not necessarily bovine, are ingested, and enter the mesenteric lymph nodes by way of Peyer's patches. It is possible for one draught of raw milk to start the infection ; it is equally possible that a toddler can become infected with human tubercle bacilli by placing one dust-covered small object in its mouth. Sometimes only one lymph node is infected ; usually there are several; occasionally massive involvement occurs (fig. 661).

FIG. 661.—Massive tuberculosis of the lymph nodes of the mesentery.

Clinical Features :

1. **Demonstrated Radiologically.**—The shadows cast by one or more calcified tuberculous lymph nodes are seen frequently in a plain radiograph of the abdomen (fig. 662). Often the shadow cast by such a node or nodes is situated in the ileo-cæcal region, but nearly as many are displayed along the line of attachment of the mesentery (fig. 663). Usually the radiological characteristics are unmistakable. Each node is round or oval, not homogenous, but mottled, and its outline is not regular, but bosselated like a blackberry. Calcification of these lymphatic nodes occurs at the earliest in eighteen months. It is often assumed that because a tuberculous lymph node is calcified, the

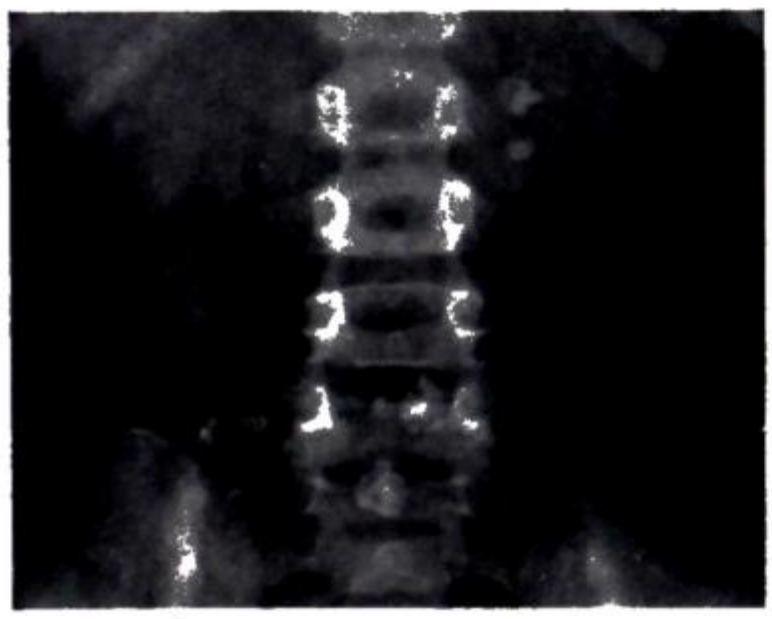

FIG. 662.—Radiograph showing calcified tuberculous lymph nodes of the mesentery. (*Dr. H. R. E. Wallis, Bath.*)

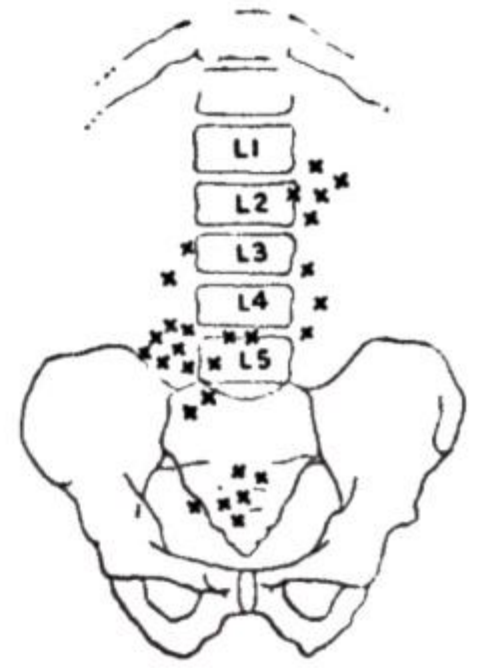

FIG. 663.—The site of calcified tuberculous mesenteric lymphadenitis; twenty cases aggregated. (*After H. R. E. Wallis.*)

Johann Conrad Peyer, 1653–1712. Professor of Medicine, Schaffhausen, Switzerland.

infection is necessarily defunct. Especially in children, this sweeping assumption is not factual. Firstly, radio-opaque nodes (see fig. 662) do not become white in a single night,[1] and secondly, calcification does not eliminate the presence of uncalcified tuberculous sister nodes. A good criterion is that while the lesion is active, the patient is tuberculin-positive.

Not infrequently the presence of calcified tuberculous mesenteric nodes first becomes apparent during the course of an abdominal operation for another condition, or at necropsy.

2. **As a Cause of General Symptoms.**—Less frequently the tuberculous process is more active, and gives rise to general symptoms. The patient, usually a child under ten years of age, loses appetite, looks pale, and there is some loss of weight ; sometimes evening pyrexia occurs. In children with these symptoms, especially those who live in the country, if the Mantoux test is negative, brucellosis, the ' disease of mistakes,' should be thought of, and an intradermal test with brucellin performed (H. R. E. Wallis).

3. **As a Cause of Abdominal Pain.**—Sometimes abdominal pain is the cause of the patient being brought for advice; usually this pain is central, not severe, but rather a discomfort, and is often constant. On examination the abdomen is somewhat protuberant and there is tenderness on deep pressure to the right of the umbilicus. In these circumstances the condition has to be distinguished from acute non-specific mesenteric lymphadenitis. On deep palpation inflamed mesenteric nodes sometimes are palpable as firm, discrete, tender bean-like objects most frequently to the right of and near the umbilicus. In both conditions a normal leucocyte count favours tuberculosis, and in a child a positive Mantoux test is confirmatory evidence of tuberculosis.

4. **Symptoms Indistinguishable from those of Appendicitis.**—On occasions the abdominal pain is acute and may be accompanied by vomiting. This, combined with tenderness and some rigidity in the right iliac fossa, makes the diagnosis from subacute appendicitis almost impossible. When, as is sometimes the case, the tuberculous infection of the mesenteric lymph nodes becomes reactivated in adolescent or adult life, the diagnostic difficulties are even greater. A radiograph may show calcified lymph nodes, but as such a condition can co-exist with appendicitis, in some cases laparotomy for appendicectomy and visualisation of the lymph nodes is necessary. If the mesentery is found to be in an inflamed state with caseation of some of the lymph nodes, the diagnosis of active tuberculosis of the nodes is confirmed.

Treatment.—Unless the home conditions are poor or general symptoms are in evidence, sanatorium treatment, although advantageous, is not essential. There is no justification for treating the condition with streptomycin, P.A.S., or isoniazid. In the majority of cases the lesion heals spontaneously.

As a means of assisting healing by calcification, the administration of

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[1] My hair is grey, but not with years,
Nor grew it white
In a single night,
As men's have grown from sudden fears :
Byron, *The Prisoner of Chillon.*

Sir David Bruce, 1855–1931. Major-General, Army Medical Service.
Hugh R. E. Wallis, Contemporary. Consultant Pædiatrician, Bath.

calciferol is logical. The dose should be calculated by the weight of the child—1,100 units of calciferol per Kg. daily is recommended : above this level it is liable to give toxic symptoms. Ultra-violet irradiation should also be employed.

The prognosis with this treatment is excellent. From time to time a local abscess forms, usually in the right iliac fossa, when the tuberculous pus should be evacuated and the abdomen closed without drainage (see also Pseudo-mesenteric Cyst, below).

Tuberculous Mesenteric Lymph Nodes as a Cause of Intestinal Obstruction.—Remote, rather than recent, tuberculous mesenteric adenitis can be the cause of intestinal obstruction. For instance, a coil of small intestine becomes adherent to a caseating node, and is thereby angulated (fig. 664), or a free coil may become imprisoned in the tunnel beneath the site of adherence and the mesentery.

FIG. 664.—Obstruction by angulation. The gut had become adherent to a tuberculous lymph node.

As a Cause of Pseudo-mesenteric Cyst.—When tuberculous mesenteric lymph nodes break down, the tuberculous pus may remain confined between the leaves of the mesentery, and a cystic swelling having the characteristics of a mesenteric cyst is found. When such a condition is confirmed at operation the tuberculous pus should be aspirated without soiling the peritoneal cavity, the wound closed, and general treatment continued until the infection has been overcome.

Calcifying Mesenteric Lymph Nodes as a Confusing X-ray Shadow.—Sometimes the shadow cast by a calcified mesenteric lymph node or nodes simulates that of a ureteric calculus or renal calculi. A change of posture often causes a lymph-node shadow to alter in position. Pyelography clarifies the diagnosis in doubtful cases.

MESENTERIC CYSTS

Classification
- Chylolymphatic.
- Enterogenous.
- Urogenital remnant.
- Dermoid (teratomatous cyst).

Cysts arising from a urogenital (Wolffian or Müllerian) remnant are essentially retroperitoneal, but they are included in the classification because it is not impossible for such a cyst to project forward into the mesentery.

The following, while not being mesenteric cysts in the academic meaning of the term, give rise to the same physical signs. From the practical point of view they *are* mesenteric cysts :

Serosanguineous cyst is probably traumatic in origin, but a history of an accident is seldom obtained.

Tuberculous Abscess of the Mesentery (see above).

Hydatid Cyst of the Mesentery.

Kaspar Friedrich Wolff, 1733–1794. Professor of Anatomy and Physiology, St. Petersburg.
Johannes Müller, 1801–1858. Professor of Anatomy and Physiology, Bonn, Germany.

Chylolymphatic cyst, the commonest variety of mesenteric cyst, probably arises in congenitally misplaced lymphatic tissue that has no efferent communication with the lymphatic system : it arises most frequently in the mesentery of the ileum. The thin wall of the cyst, which is composed of connective tissue lined by flat endothelium is filled with clear lymph or, less frequently, with chyle varying in consistency from watered milk to cream. Frequently the cyst attains a great size (fig. 665). More often unilocular than multilocular, a chylolymphatic cyst is almost invariably solitary, although there is an extremely rare variety in which myriads of cysts are found in the various mesenteries of the abdomen. A chylolymphatic cyst has a blood supply independent of that of the adjacent intestine, and the cyst wall, although often contiguous, is not continuous with that of the intestine, thereby making enucleation without the necessity of resection of gut possible.

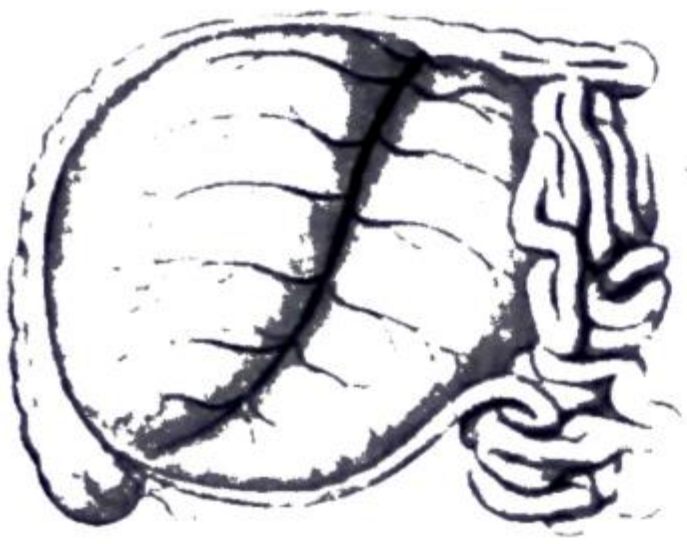


FIG. 665.—A large chylolymphatic mesenteric cyst. (*Higgins and Lloyd.*) (*British Journal of Surgery.*)

Enterogenous cyst is believed to be derived either from a diverticulum of the mesenteric border of the intestine which has become sequestrated from the intestinal canal during embryonic life, or from a duplication of the intestine. An enterogenous cyst has a thicker wall than a chylolymphatic cyst, and it is lined by mucous membrane, sometimes ciliated. The content is mucinous, and is either colourless or yellowish-brown from bygone hæmorrhage into the cyst. As can be seen at operation, the muscle in the wall of an enterogenous cyst and the bowel with which it is in contact have a common blood supply ; consequently removal of the cyst always entails resection of the related portion of intestine.

Clinical Features of a Mesenteric Cyst.—A mesenteric cyst is encountered most frequently in the second decade of life, less often between the ages of one and ten years, and infrequently in infants under one year.

The patient presents on account of :

(*a*) *A Painless Abdominal Swelling.*—A cyst of the mesentery presents characteristic physical signs. These have been well summarised by Tillaux, and are known as Tillaux's triad.

1. There is a fluctuating swelling in the abdomen near the umbilicus.

2. The swelling moves freely in a plane at right angles to the attachment of the mesentery (fig. 666).

FIG. 666.—A mesenteric cyst moves freely in the direction of the arrows, i.e. at right angles to the attachment of the mesentery.

3. There is a zone of resonance around and, classically, a belt of resonance across the cyst.

(*b*) *Recurrent attack of abdominal pain* with or without vomiting. The pain results from recurring temporary impaction of a food bolus in a segment

Paul Tillaux, 1834–1904. Professor of Surgery, University of Paris.

of bowel narrowed by the cyst or possibly from tension at the root of the mesentery.

(*c*) *An acute abdominal catastrophe* arises (1) as a result of torsion of that portion of the mesentery containing the cyst; (2) rupture of the cyst, often due to a comparatively trivial accident; (3) hæmorrhage into the cyst; (4) infection.

Radiography.—In most instances the patient should be submitted to X-ray after a barium meal. The hollow viscera will be found to be displaced around the cyst, and not infrequently some portion of the lumen of the small intestine will be narrowed. In order to exclude or confirm the diagnosis of a hydronephrosis an excretory pyelogram should not be omitted. In cases of painless enlargement of the abdomen this examination should be undertaken first.

Treatment.—As has been indicated already, many chylolymphatic cysts can be enucleated *in toto*.

When, after aspiration of about half the contents of the cyst, the major portion of the cyst has been dissected free but one portion abutting the intestine or a major blood-vessel seems too dangerous to remove, this portion can be left attached and its lining destroyed by swabbing with pure carbolic acid, the excess of which is removed by alcohol.

In the case of an enterogenous cyst, enucleation must not be attempted. If a comparatively short segment of the intestine is involved, resection of the cyst with the adherent portion of the intestine, followed by intestinal anastomosis, is the correct course. Should a very large segment of small intestine be implicated, an anastomosis should be made between the apex of the coil of small intestine and the cyst wall which, in this instance, holds sutures well.

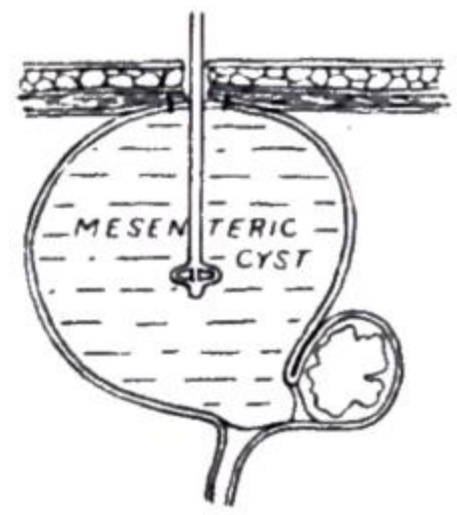


FIG. 667.—Method of treating a large mesenteric cyst by marsupialisation.

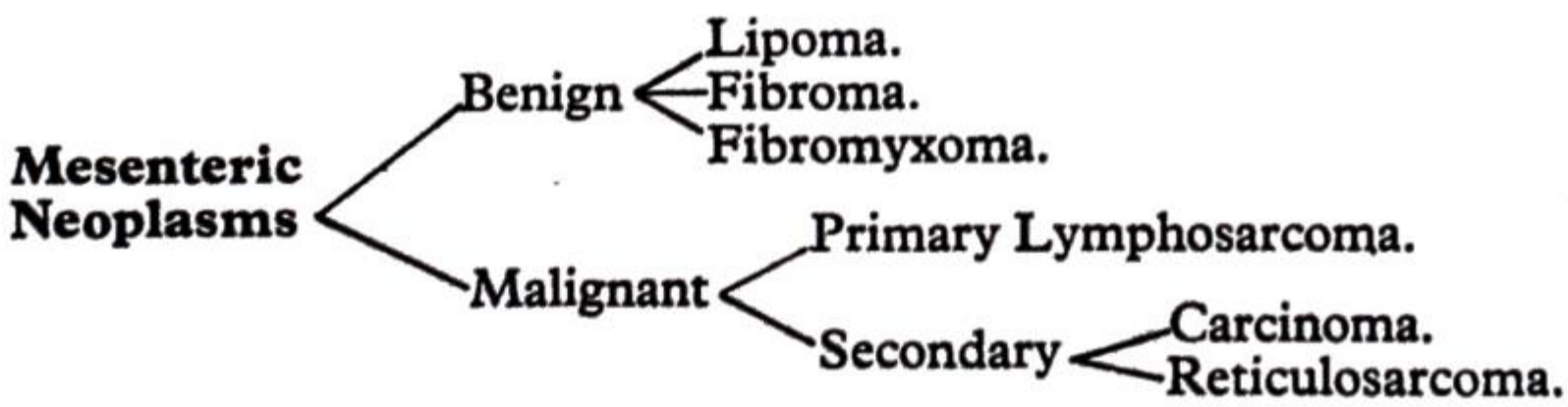
The older treatment of marsupialisation of a mesenteric cyst (fig. 667) has little to recommend it, for a fistula or recurrence sometimes results. However, occasionally, on account of its simplicity, it is a good standby in a poor-risk subject.

Omental cyst occurs nearly as frequently as a mesenteric cyst. Preoperative differentiation is possible because a lateral radiograph shows the cyst in front of the intestines. Treatment is ultra-simple—omentectomy.

Cyst of the mesocolon is uncommon, and it is differentiated from a mesenteric cyst only at operation. The treatment is similar.

NEOPLASMS OF THE MESENTERY

Tumours situated in the mesentery give rise to physical signs similar to those of a mesenteric cyst, the sole exception being that they sometimes feel solid.



A benign tumour of the mesentery is excised in the same way as an enterogenous mesenteric cyst, i.e. with resection of the adjacent intestine. When possible, a malignant tumour of the mesentery is subjected to the same treatment. In inoperable cases radiotherapy can be employed if the biopsy specimen reveals that the growth is probably radio-sensitive.

THE RETROPERITONEAL SPACE

Pus or blood in the retroperitoneal space tends to track to the corresponding iliac fossa. If a retroperitoneal hæmatoma or an abscess develops, it should be evacuated by an incision through the abdominal wall, meticulously avoiding opening the peritoneum. Should the retroperitoneal collection be found at laparotomy, it must be drained by a counter-incision in the flank, and only after gloves, etc., have been changed, is the laparotomy incision closed.

Retroperitoneal Cyst.—A cyst developing in the retroperitoneal space often attains very large dimensions, and has at first to be distinguished from a hydronephrosis. After the latter condition has been eliminated by pyelography, because it presents the same clinical features a retroperitoneal cyst can seldom be diagnosed with certainty from a retroperitoneal tumour, until displayed at operation. The cyst may be unilocular or multilocular. Many of these cysts are believed to be derived from a remnant of the Wolffian duct, in which case they are filled with clear fluid. Others are teratomatous, and are filled with sebaceous material.

Excision of these and other retroperitoneal swellings is best performed through a transperitoneal incision (see below).

PRIMARY RETROPERITONEAL NEOPLASMS

Although neuroblastomata and ganglioneuromata of the adrenal gland are retroperitoneal tumours, they have now been segregated as clinical entities, and have been dealt with on p. 266. Of the other retroperitoneal neoplasms, the most frequent innocent tumour—indeed, for practical purposes, the only innocent tumour—is a lipoma. Of the malignant varieties, lymphosarcoma, liposarcoma, leiomyosarcoma, Hodgkin's disease, and embryonal carcinoma occur in that order of frequency (L. V. Ackerman).

Retroperitoneal lipoma, in the first instance, is usually mistaken for a hydronephrosis, a diagnosis which is ruled out by pyelography. Women are more often affected. These swellings sometimes reach an immense size. We have removed such a tumour weighing 5½ lb. (2·5 kgm.), and much larger specimens have been recorded. A retroperitoneal lipoma sometimes undergoes myxomatous degeneration, a complication which does not occur in a lipoma in any other part of the body. Moreover, undoubtedly a retroperitoneal lipoma sometimes becomes malignant (liposarcoma) (fig. 668).

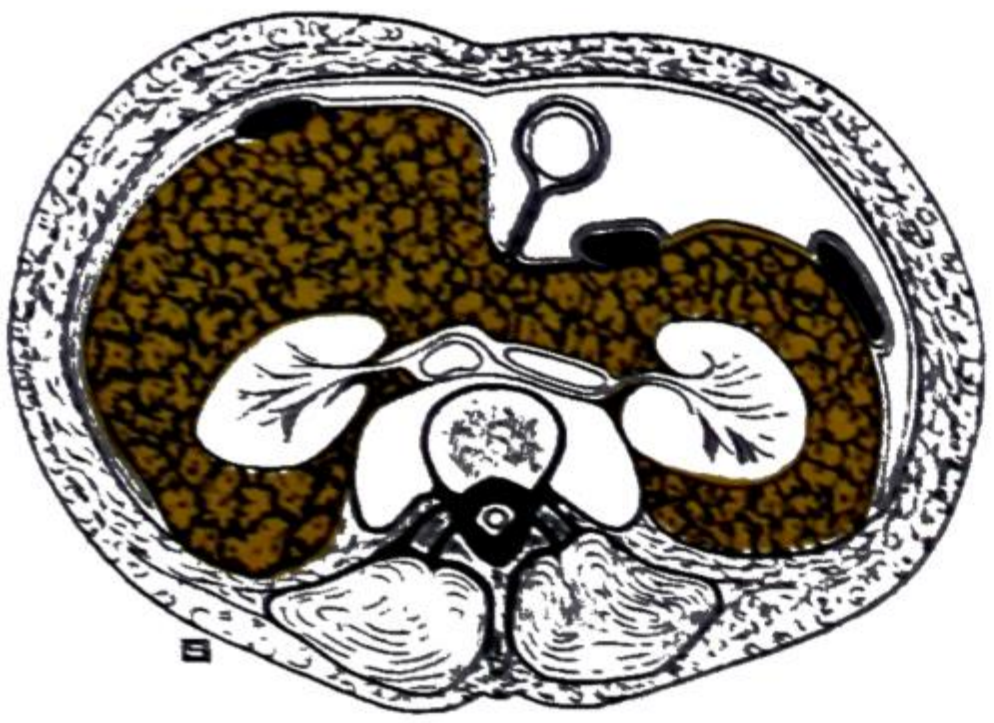


FIG. 668.—Rapidly growing retroperitoneal liposarcoma.

Retroperitoneal sarcoma presents signs similar to a retroperitoneal lipoma. The patient may seek advice on account of a swelling or because of indefinite abdominal pain. On other occasions the tumour, by pressure on the colon, causes symptoms of subacute intestinal obstruction. On examination a smooth fixed mass, which is not tender, is palpated. The most probable original diagnosis is that of a neoplasm of the kidney. This is ruled out by pyelography. The ureter, however, is liable to become displaced by the tumour. Exploratory laparotomy should be performed, and when possible the tumour is removed. Often it is found widely disseminated in the retroperitoneal space, rendering complete removal impossible, in which case a portion is excised for

Thomas Hodgkin, 1798–1866. Curator of the Museum, Guy's Hospital, London.
Lauran V. Ackerman, Contemporary. Professor of Pathology and Surgical Pathology, Washington University, St. Louis, Missouri.

microscopy. Even when excised at a comparatively early stage, recurrence always takes place, and these tumours must be looked upon as being necessarily fatal. Deep X-ray therapy sometimes keeps recurrences in abeyance for a time.

Removal of a Retroperitoneal Cyst or Neoplasm.—After the anterior abdominal wall has been opened and the diagnosis of a retroperitoneal tumour has been confirmed, the incision is extended as necessary. The small intestine is packed away in the upper abdomen, and the cæcum and the sigmoid are relegated to their respective fossæ. The retroperitoneum is then incised throughout its length over the area to be exposed, the incision paralleling the medial border of the aorta. The retroperitoneum is dissected from the tumour. If the dissection is likely to be an extensive one, it is advisable to unite temporarily the two layers of the peritoneum on either side of the midline superiorly, packs being removed in the course of the process. This leaves a small opening at the upper angle of the wound which can be protected by a pack. This procedure permits the retroperitoneum to be exposed without hindrance from protrusion of intestines (G. H. Pratt).

Gerald H. Pratt, Contemporary. Surgeon, St. Vincent's Hospital, New York.

CHAPTER XXV

THE INTESTINES

HAMILTON BAILEY

Embryology.—Beneath the notochord appears the primitive alimentary canal, which is continuous with the yolk-sac (fig. 669). This canal becomes differentiated into:

The fore-gut, from which are developed the pharynx, œsophagus, and stomach, together with the duodenum as far as the ampulla of Vater.

The hind-gut, which is the forerunner of the descending colon, pelvic colon, rectum, and the anal canal as far as the pectinate line.

The mid-gut, which gives origin to the remainder of the intestinal canal.

FIG. 669. — Embryo showing the primitive alimentary canal and the yolk-sac.

As development proceeds, the communicating channel between the mid-gut and the yolk-sac dwindles into the *vitello-intestinal duct*. Later still the duct disappears entirely, except in 2 per cent. of individuals, where its inner extremity is represented by a pouch, so well known as Meckel's diverticulum (see p. 521). The mid-gut grows apace, and becomes differentiated into the small intestine and the proximal half of the colon, the junction being indicated by an outgrowth that later becomes the cæcum. The gut, now suspended by a mesentery, elongates and during the fourth week of intrauterine life the greater part of the mid-gut is extruded from the cœlom, and comes to lie within the umbilical cord. This is a temporary physiological hernia, which sometimes persists (see Exomphalos, p. 699). Normal rotation of the gut occurs during the process of reduction of the physiological hernia, and continues when the mid-gut regains the cœlomic cavity.

There are three stages of rotation :

First stage occurs while the mid-gut loop occupies the umbilical cord. During this stage the loop elongates and rotates counter-clockwise 180° on the axis of the mesenteric vessels.

Second stage comprises the return of the mid-gut with a further rotation of 90°.

Third stage.—Further rotation occurs and the ultimate position is a total counter-clockwise rotation of 270° from the saggital plane (fig. 670). During this stage the duodenum, mesentery and descending colon become fixed to the posterior abdominal wall while the cæcum comes to rest in the right iliac fossa.

FIG. 670.—Rotation of portions of the alimentary canal on either side of the middle line.

Surgical Anatomy.—It is of great practical importance to be able :

1. To distinguish various portions of the intestinal canal at sight.
2. To know in which part of the abdomen the upper coils, as opposed to the lower coils, of the small intestine lie in relationship to the anterior abdominal wall.
3. To be able to tell which is the proximal and which the distal end of any coil under consideration.
4. To distinguish irrefutably large from small intestine.

For practical purposes these problems are settled as follows :

(*a*) The mesentery of the jejunum has only two series of arches of blood-vessels, whereas the lower ileum has several series of arches.

(*b*) Monks's method of intestinal localisation roughly indicates the disposition of the upper, middle, and lower thirds of the small intestine (fig. 671).

Abraham Vater, 1684–1751. Professor of Anatomy and Botany, Wittenburg.
George H. Monks, 1853–1933. Surgeon-in-Chief, City Hospital, Boston, U.S.A.

(*c*) The mesentery, after being made taut, is examined. As the mesenteric attachment runs from left to right (fig. 672), if palpation reveals the mesentery is not twisted, then the upper end of the bowel in the wound is the proximal end. Such

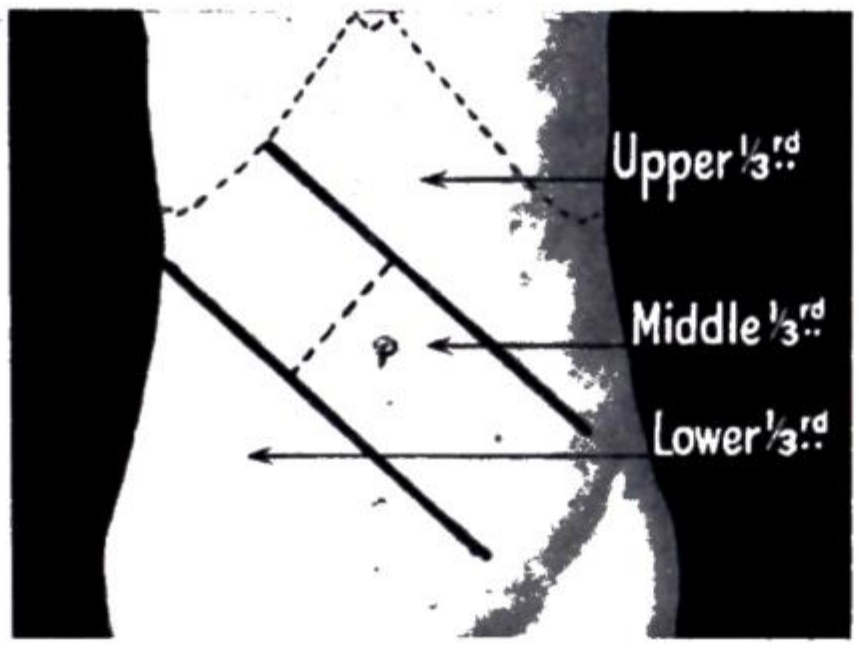


FIG. 671. — Monks's method of localising the small intestine upon the surface.

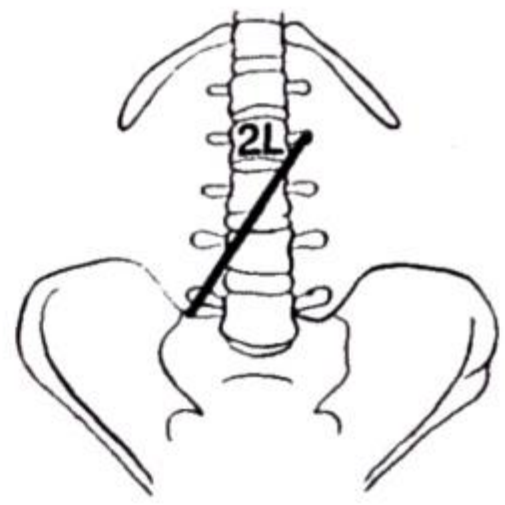


FIG. 672. — The attachment of the mesentery.

a test is useful, but not as easy to perform in the living as in the dissecting-room subject.

(*d*) As the 'small' intestine is sometimes found enormously distended and the 'large' intestine entirely collapsed, size is no criterion. The large intestine is characterised by its tæniæ coli and appendices epiploicæ.

Length of the Intestines.—After death, the average length of the intestines is as follows :

Small Intestine		*Large Intestine*	
Men	*Women*	*Men*	*Women*
21 ft.	19½ ft.	6 ft.	5 ft.

(*Betty M. L. Underhill.*)

There are racial differences in intestinal length, the average being higher in India than in Europe, and higher still among American negroes, in whom lengths of 40 feet (12 metres) are not unknown. It should be noted carefully that all the foregoing measurements refer to the cadaver. In life, intestinal length is astonishingly less. Thus the whole alimentary tract, from the mouth to the anus, is usually only 8 to 10 feet (2·4 to 3 metres) long (W. C. Alvarez). This statement has been proved conclusively by the use of an intestinal aspiration tube in cases of intestinal obstruction. In a number of instances the inflatable balloon has been extruded from the anus, and the measurement from the naris to the anus has been, on an average, 9 feet. Conclusive experimental and radiographic evidence has shown that the gut is not telescoped upon the tube, but the tube lies evenly along the mesenteric border of the intestine. Loss of intestinal tone is responsible for the lengthening that takes place after death. In a small percentage of persons the small intestine is much shorter than the average.

The Main Arteries of the Large Intestine.—The right and middle colic arteries are branches of the superior mesenteric artery, and supply the ascending colon, hepatic flexure, and transverse colon. The left colic artery and the sigmoidal arteries are branches of the inferior mesenteric artery, and supply the left colon and the upper rectum respectively (fig. 673(*a*)). An interesting and not infrequent

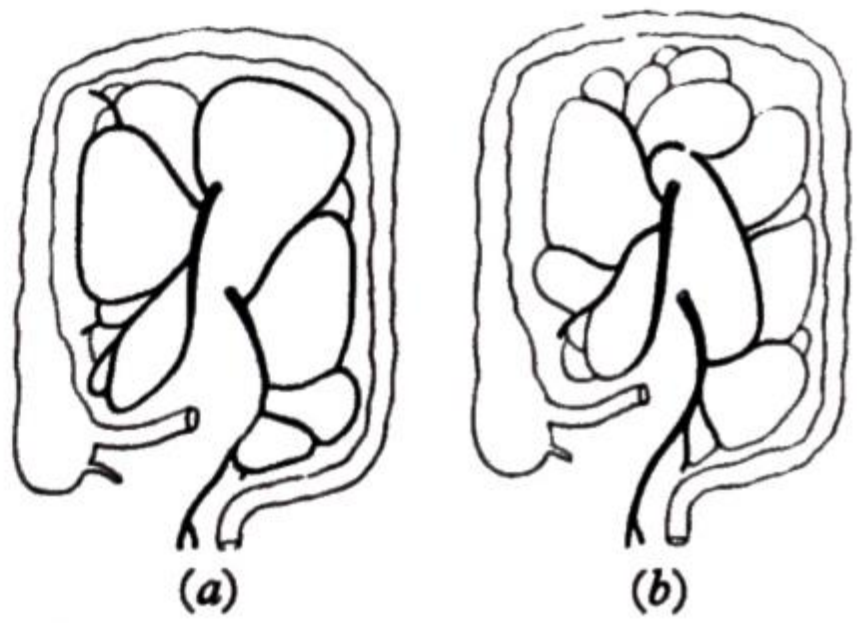


FIG. 673.—(*a*) The main arteries of the large intestine. (*b*) Showing the arc of Riolan. (*After J. V. Basmajian.*)

Betty M. L. Underhill, Contemporary. Surgical Registrar, Chase Farm Hospital, Enfield, Middlesex.
Walter Clement Alvarez, Contemporary. Consulting Physician, The Mayo Clinic, Rochester, Minnesota.
Jean Riolan, 1577–1657. Professor of Anatomy and Botany, Paris. He bitterly opposed William Harvey's views on the circulation of the blood.

anomaly is the arc of Riolan (fig. 673(*b*)) which is a loop running retroperitoneally from the root of the superior mesenteric artery, or one of its primary branches, to the inferior mesenteric artery, or one of its primary branches. The superior mesenteric artery is sometimes double. Except in the rectosigmoid area, where it is often absent, anastomosis between the arteries supplying the colon is generally good or excellent, but this is not by any means always the case. The inferior mesenteric artery occasionally is absent, in which event the entire intestinal tract is supplied by the superior mesenteric artery. At times there is failure of anastomosis between the left and middle colic arteries, and other abnormalities are common (I. S. Ravdin). Amidst these inconsistencies of the normal arterial arrangement it is imperative that resection of lengths of the colon with anastomosis should not be stereotyped, but planned for each individual at points where each end of the remaining bowel receives an adequate blood supply.

CONGENITAL MALFORMATIONS OF THE INTESTINES

Congenital Atresia of the Duodenum (see p. 364).
Congenital Atresia of the Small Intestine (see p. 567).
Volvulus Neonatorum (see p. 569).

Failure of Descent of the Cæcum.—The cæcum remains under the right lobe of the liver—a normal situation of the structure in the mangabey monkey. This anomaly, which is not infrequent, is of importance, as it is associated with displacements of the vermiform appendix.

HIRSCHSPRUNG'S DISEASE (*syn.* CONGENITAL AGANGLIONIC MEGACOLON)

Pathology.—Hirschsprung's disease is characterised by enormous dilatation and hypertrophy of the pelvic colon, which sometimes extends into the descending colon but rarely involves the more proximal parts of the large intestine. The pelvic mesocolon is elongated and thickened and its blood-vessels are large and prominant. All coats of the dilated intestine show gross pathological changes. The serous coat is thickened, the circular fibres of the muscle coat are greatly hypertrophied, whereas the tæniæ coli are unaffected and relatively inconspicuous. The mucous lining is chronically inflamed and frequently ulcerated. In Hirschsprung's disease there is a terminal constricted, non-hypertrophied, segment of bowel involving the anal canal, the rectum, and a variable part of the large intestine. In nine out of ten cases the upper limit of the contracted segment is the pelvi-rectal junction; exceptionally the deficiency extends to a higher level (fig. 674). It is in this

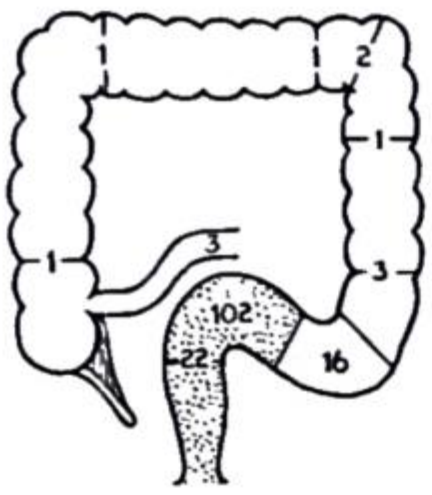


FIG. 674.—Extents of aganglionic segments in 152 cases of Hirschsprung's disease treated by operation at the Hospital for Sick Children, Great Ormond Street. (*After G. G. Wyllie.*)

contracted segment that physiological obstruction lies, and the dilatation hypertrophy of the colon above is due to absence of peristalsis in the spastic segment.

On histological examination the cause of the immotility of the spastic

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In 1898 Sir Frederick Treves attributed the cause of Hirschsprung's disease to congenital spasm of the distal segment, thus preceding the re-discovery of this concept by fifty years.

Isidor Schwaner Ravdin, Contemporary. Professor of Surgery, Philadelphia.
Harald Hirschsprung, 1830–1916. Physician, Queen Louise Hospital for Children, Copenhagen.
Sir Frederick Treves, 1853–1923. Surgeon, The London Hospital.

segment is evident; there is a complete absence of parasympathetic ganglion cells, and this ganglion deficiency extends for a distance of 0·4 to 2 inches (1 to 5 cm.) into a transitional zone (fig. 675) between the terminal spastic

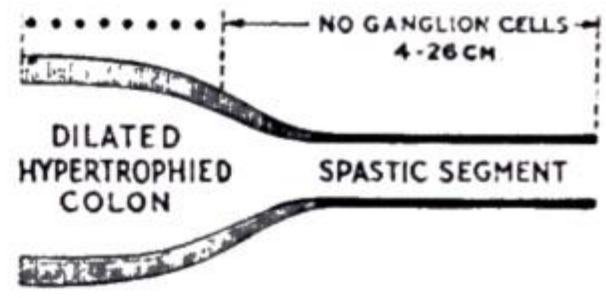


FIG. 675.—Showing the essential pathological histology in congenital Hirschsprung's disease. (*After M. Bodian.*)

segment and the hypertrophied portion (M. Bodian). Above the transitional zone, parasympathetic ganglion cells are present as in normal intestine.

Clinical Features.—Hirschsprung's disease shows a familial tendency. It is much more common in males than females. In 90 per cent. of cases symptoms appear within three days following birth. In 5 per cent. they develop within the first three months. In 4 per cent. they pass unnoticed until between three and twelve months after birth. In only 1 per cent. of cases are symptoms delayed past the first birthday.

Constipation.—The infant fails to pass meconium during the first two or three days of life, and then only after the insertion of a little finger or a tube into the rectum. Subsequently motions are tooth-paste-like, and inadequate in amount; straining is in evidence during their passage.

Vomiting.—Some regurgitation of feeds is accepted as normal in the neonatal period. In about 30 per cent. of cases of Hirschsprung's disease bile-stained vomiting occurs.

Abdominal distension is usually unmistakable by the third day. In a proportion of cases the abdominal distension progresses, and sometimes it is evident that the colon is obstructed (fig. 676): in others it is impossible clinically to differentiate large from small intestinal obstruction. Loud borborygmi and visible peristalsis are much in evidence.

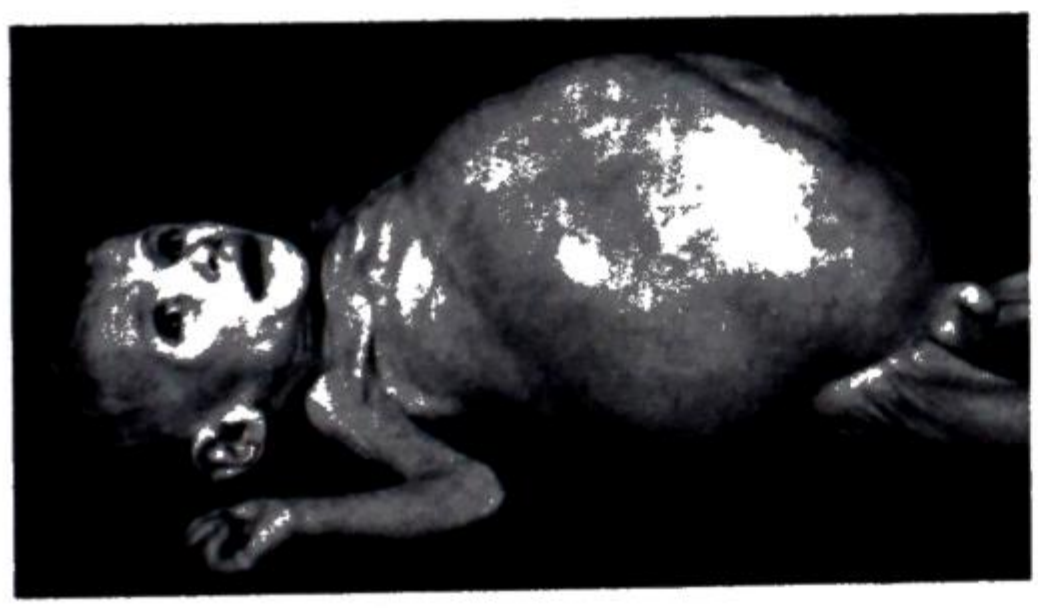


FIG. 676.—Hirschsprung's disease showing enormous colonic dilatation and a wave of visible peristalsis. (*Mr. Denis Browne, London.*)

Rectal examination.—In neonatal cases the anus looks smaller than normal, but it admits the 5th digit. The rectum is empty, and the constricted recto-sigmoidal region can be appreciated. Usually, after withdrawal of the finger flatus and meconium are passed. In older children the anus admits an index finger, and in 98 per cent. of cases the essential findings are similar to those just described. In 2 per cent. of cases the rectum is loaded, therefore a loaded rectum does not preclude the possibility of Hirschsprung's disease.

Acute intestinal obstruction supervenes in about 25 per cent. of cases—quite frequently within a few days of birth. As a rule the constipation continues, or becomes complete. In a few, the constipation is interrupted by attacks of diarrhœa, when fluid fæces are passed from above the inspissated

Martin Bodian, Contemporary. Morbid Anatomist, Hospital for Sick Children, London.

masses; vomiting with diarrhœa simulates gastro-enteritis. The infant becomes toxic and dehydrated, and if fluid balance is not restored quickly and the obstruction relieved, death occurs. Reverting to patients with the more usual chronic symptoms: provided a congenital stricture of the anal canal is ruled out, a history of constipation dating from birth can be regarded as indicative of Hirschsprung's disease.

Radiography: *A plain X-ray* of the abdomen often confirms the diagnosis, for gas-filled small and large intestine can be seen above the aganglionic segment. However, in infants haustrations are ill-defined, and for this reason, and especially if the aganglionic segment extends into or past the transverse colon, sometimes it is impossible to differentiate Hirschsprung's disease from other causes of neonatal intestinal obstruction (see p. 567).

FIG. 677.—Radiological appearances in Hirschsprung's disease; coning, as well as dilatation, is diagnostic. (*After B. C. H. Ward.*)

Barium Enema.—When the clinical findings are atypical, a barium enema is often helpful in confirming the diagnosis. The objective is to demonstrate the contracted aganglionic segment, if such be present, and to this end preliminary wash-outs are withheld. For reasons explained below, the barium should be admixed with normal saline solution, not tap-water. By using only a little dilute barium emulsion—just sufficient to run over the fæcal masses—a good outline of the bowel can be obtained (fig. 677).

Biopsy of the ano-rectal wall is employed in order to prove or disprove the presence of ganglia in the myenteric plexus. A specimen is obtained by a small incision through the mucosa between the columns of Morgagni to secure a specimen of the circular muscle fibres, together with the longitudinal fibres externally. The presence or absence of ganglion cells between these layers can then be determined. The indications for biopsy are:

1. When the barium enema findings are inconclusive.
2. When the whole colon, or the greater part thereof, is spastic.
3. When the symptoms include diarrhœa instead of the conventional constipation.

Laparotomy to establish the diagnosis is required when the patient has acute-on-chronic intestinal obstruction, and other causes cannot be eliminated. If the case proves to be one of Hirschsprung's disease (fig. 678), as a rule it is advisable to establish a temporary transverse colostomy or, in the rare event of the whole colon being involved in spasticity, an enterostomy.

FIG. 678.—Hirschsprung's disease; laparotomy findings. (*After A. Jolleys.*)

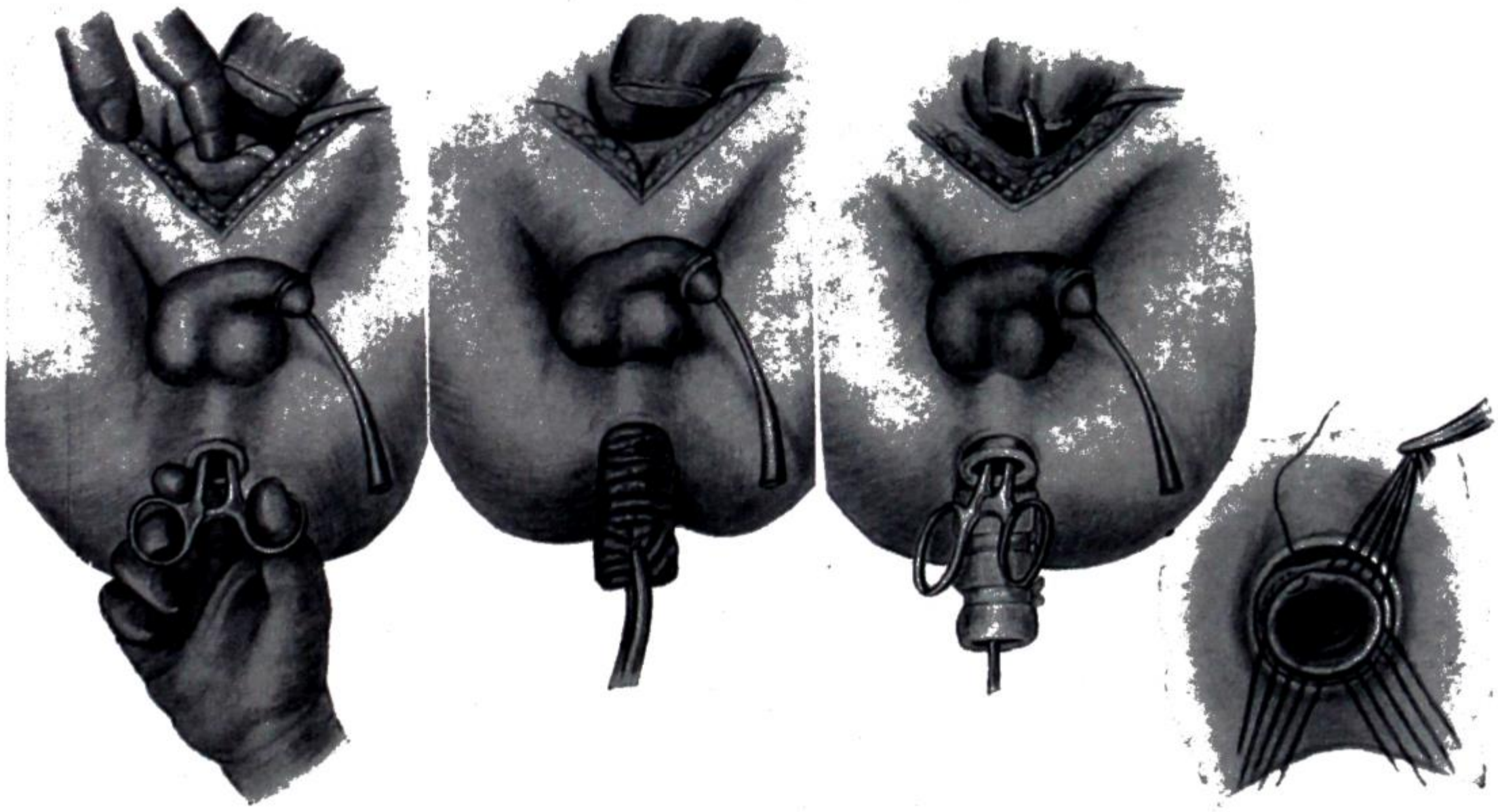
Pre-operative Treatment.—Colonic lavage in Hirschsprung's disease is dangerous, for the wall of the aganglionic segment is weak, and is perforated easily. Therefore enemata should be administered only by a very highly-skilled person. Another danger that needs to be known widely is that the

Giovanni Morgagni, 1682–1771. Professor of Surgery, Padua.

use of tap-water for enemas in this condition is liable to result in water intoxication; the megacolon absorbs water much more rapidly than does a normal colon, and the disturbance of electrolytic balance, especially of sodium, may prove fatal. Provided the general condition is good, normal saline solution can be used with safety. In patients with impaired cardiac or renal reserve, 7 per cent. gelatine solution should be substituted. Unless measurements show that distension is increasing, four weeks' pre-operative preparation is desirable (O. Swenson).

Treatment.—The only curative treatment of Hirschsprung's disease is excision of the entire aganglionic segment. So it comes about that the teaching of Sir Frederick Treves has, after 50 years of trial and tribulation of other methods, now come to be the standard treatment. It is interesting to record that a patient operated upon by Sir Frederick Treves for Hirschsprung's disease in 1897 is alive and well, and has excellent anal continence (H. D. Johnson).

Operation.—The patient is placed in a semi-lithotomy position, which gives good access to the abdomen and the peri-anal region. Through a left lower paramedian incision (surrounding the colostomy, if such has been performed), the rectum, the contracted part of the adjoining colon, and the commencement of the hypertrophied portion are freed from their attachments as far downwards as possible towards the anal canal. In contrast to excision for carcinoma of the rectum, the dissection must be kept immediately outside the fascia propria. In this way the seminal vesicles and the autonomic nerve trunks to the bladder are protected. Should doubt exist as to whether the aganglionic segment has been encompassed, frozen section biopsy will provide the answer. The intestine is then transected at an indubitably



(*a*) (*b*) (*c*) (*d*)

FIG. 679.—(*a*) Long hæmostat introduced through the anus so that the top of the closed bowel can be grasped. (*b*) Mobilised aganglionic segment is everted through the anus. (*c*) Long hæmostat introduced through the incision in the rectal wall to grasp the proximal end of the divided colon. (*d*) Anastomosis of the colon to the commencement of the anal canal. (*After O. Swenson.*)

Orvass Swenson, Contemporary. Surgeon, The Boston Floating Hospital for Infants and Children, Boston, Mass.
Harold Daintree Johnson, Contemporary. Surgeon, Royal Free Hospital, London.

motile level, and the distal end is closed with a purse-string suture. The mobilised aganglionic segment is then everted through the anus (fig. 679(*a*) and (*b*)). The mucosa thus exposed is painted with antiseptic solution, and dried. The anterior half of the junction of the inverted rectum with the anal canal is opened transversely, and the proximal colon is pulled through the opening (fig. 679(*c*)). End-to-end anastomosis between the colon and the anal canal is carried out (fig. 679(*d*)) as the everted aganglionic segment is excised. The union having been completed, it is reduced into the anal canal. Gloves having been changed, the abdominal incision is repaired.

By two teams working simultaneously, one abdominally and one perineally, the operation can be performed expeditiously.

Cases of Hirschsprung's disease involving the entire colon have been treated successfully by total colectomy and ileoproctostomy.

PSEUDO-HIRSCHSPRUNG'S DISEASE (*syn.* ACQUIRED MEGACOLON)

In these cases dilatation and hypertrophy of an otherwise normal large bowel extends to the anal canal. The source of the obstruction is fæcal impaction. Frequently a spastic anal sphincter is encountered, but in the absence of an anal fissure it is uncertain whether the spasticity is the cause or the result of the impaction. As a rule faulty bowel care and training are the source of the trouble, and usually they can be traced to infancy; the onset, however, is never from birth.

Dolichocolon of Martinotti.—Occasionally a patient is encountered in whom a long redundant colon is present, the excessive absorptive surface of which removes too much water from the fæces, and chronic constipation results. Sometimes the first recognised indication of the presence of a dolichocolon is volvulus of the sigmoid in adult life. Elongation of the colon is more often the result of constipation and obstructive impaction than the cause of it.

Symptomatic megacolon has long been recognised as separate from idiopathic cases. A cause of the obstruction is demonstrable; occasionally an anal fissure causing spasm of the external sphincter is present, but the most common single cause is stricture following an operation for imperforate anus. As fæcal impaction develops and persists, an extensive megacolon results.

Rectal Examination.—Almost at once the finger encounters a scybalous mass, which is contrary to what is found in Hirschsprung's disease.

Radiography.—In all cases of acquired megacolon the dilatation as shown by a barium enema ends at the anal canal (fig. 680).

FIG. 680.—Radiological appearances of acquired megacolon. (*After B. C. H. Ward.*)

Biopsy of the Ano-rectal Wall.—When acquired megacolon cannot be differentiated from Hirschsprung's disease by clinical and radiological means, biopsy is required. The presence of ganglion cells in the myenteric plexus is the signal for careful and prolonged conservative treatment.

Conservative Treatment.—Should an anal stricture or a fissure-in-ano be present, these lesions must be treated appropriately. Whether or not such a lesion exists, regular saline enemata and the ingestion of liquid paraffin-containing preparations, together with training in bowel habit, will cure the condition.

GENERAL VISCEROPTOSIS

All the intra-abdominal organs are ptosed. The patient is nearly always a woman. T. Rovsing recognised two types of general visceroptosis :

Guiseppe Martinotti, Contemporary. Professor, Institute of Radiology, University of Turin.
Thorkild Rovsing, 1862–1927. Professor of Surgery, University of Copenhagen.

Maternal Visceroptosis.—As a result of repeated pregnancies, the abdominal wall loses its tone and the intra-abdominal organs slide downwards.

Virginal Visceroptosis.—From childhood the patient shows signs of ptosis. She has a long, narrow, upper abdomen and tends to stoop. She is of the ' drooping lily ' type, and, as she grows older, is constantly ailing. Although, no doubt, there are uncomfortable sensations from the ptosed organs, only too often the patient becomes hypochondriacal.

Treatment is unsatisfactory. The symptoms are relieved by recumbency and in advanced stages of pregnancy. Dry meals help to obviate a heavy stomach dragging on the gastro-hepatic omentum. A well-fitting abdominal belt, put on each morning before the patient arises, and worn all day, is of great service. Massage and exercises are helpful, especially in the maternal type.

TRAUMATIC RUPTURE OF THE INTESTINE

The intestine can be ruptured without any external wound. The most frequent cause of such an accident is a kick in the abdomen, the rupture probably being produced by a coil of intestine being crushed against the sacral promontory. Rupture of the intestine is also liable to occur where a fixed part of the alimentary tract joins a free portion, such as the duodeno-jejunal flexure. The latter type of lesion is sometimes met with after run-over accidents.

In small perforations the mucosa prolapses and tends partially to seal the rent (fig. 681); consequently the early signs are misleading. In general it may be stated that the signs simulate closely those of a perforated peptic ulcer.

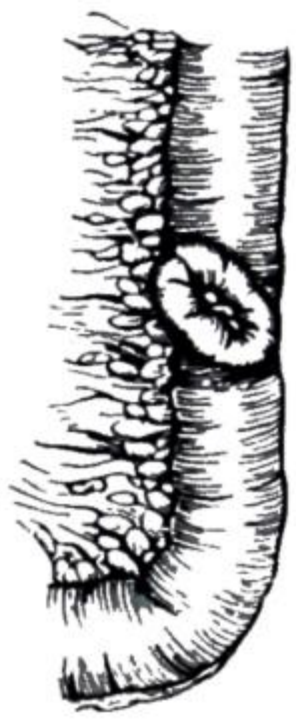


FIG. 681.—Traumatic rupture of the small intestine. Note the prolapse of the mucous membrane.

Laceration of the mesentery is a frequent operative finding in the type of injury under consideration. The intestine itself is not necessarily damaged, but, owing to devascularisation, its viability may be so imperilled as to render resection of the infarcted segment (fig. 682) imperative.

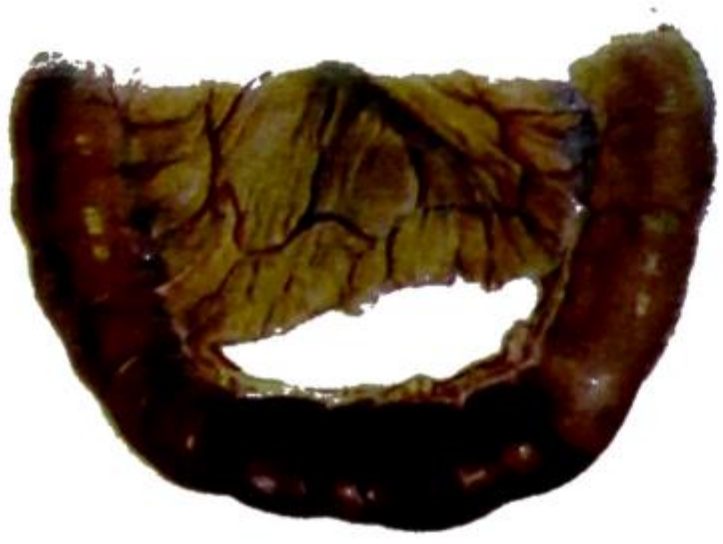


FIG. 682.—Laceration of the mesentery resulting in infarction of the associated portion of gut.

Traumatic rupture of the large intestine is very much less frequent. Compressed-air rupture of the colon is sometimes the result of a damnable form of practical joke, whereby a hose, carrying air under considerable pressure, is turned on near the victim's anus.

Blast injuries of the abdomen sustained during air-raids resulted in a number of cases of traumatic rupture of the intestine. The pelvic colon was found to be injured more frequently than other segments of the intestine. Rupture of the upper reaches of the rectum is not unknown during sigmoidoscopy. In ulcerative lesions the air insufflation has been sufficient to perforate the intestinal wall.

Treatment.—In all cases of rupture of the intestine immediate laparotomy must be performed. In many instances simple closure of the perforation is all that is required. In others, e.g. where the mesentery is lacerated, resec-

tion may be required. In the case of the large intestine, exteriorisation, if possible, is often the procedure of choice ; if this is not feasible, after closure of the perforation proximal colostomy is performed. Except in early cases of high jejunal perforation, the general peritoneal cavity must be drained. The administration of an antibiotic intravenously is an important detail in all cases of ruptured intestine.

INTESTINAL DIVERTICULOSIS AND DIVERTICULITIS

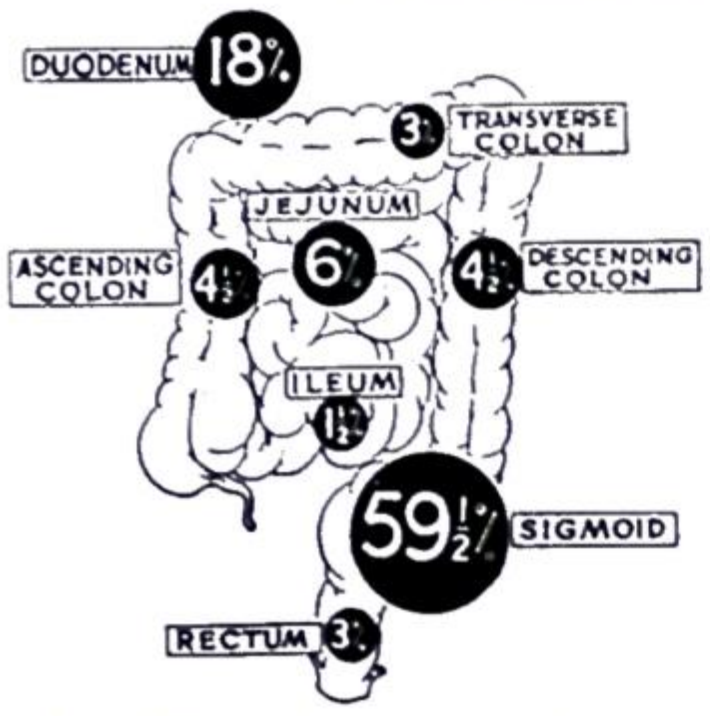


FIG. 683.—Incidence of diverticula in various portions of the intestine. Meckel's diverticulum and diverticulum of the cæcum, both definitely congenital conditions, are excluded. *(After Dixon, Deuterman, and Weber.)*

Diverticula occur in many parts of the alimentary canal from the duodenum to the rectum, but the incidence in the various parts of the intestine varies greatly (fig. 683).

All the diverticula are divided into two varieties:

(*a*) *Congenital.*—All three coats of the bowel are present in the wall of the diverticulum.

(*b*) *Acquired.*—The wall of the diverticulum lacks a muscular coat. In spite of the absence of demonstrably increased intraluminar pressure, most alimentary diverticula are thought to be acquired (H. Edwards).

Literally, a diverticulum means a wayside house of ill-fame, and these wayside houses certainly live up to their evil reputation.

DIVERTICULA OF THE SMALL INTESTINE

The belief that a diverticulum of the small intestine originates as a mucosal herniation through a point of entrance of blood-vessels into the wall of the intestine is founded upon the fact that most of these diverticula arise from the mesenteric side of the bowel (fig. 684).

FIG. 684.—Diverticulum at the *mesenteric* border of small intestine.

Duodenal Diverticulum (see p. 367).

Jejunal Diverticula vary in size, are sometimes single, but more often several are present. Usually they are symptomless, but occasionally a solitary diverticulum (fig. 685) gives rise to two main symptoms : (*a*) vague abdominal discomfort and sometimes pain, particularly after meals ; (*b*) flatulence, sometimes very pronounced and associated with loud borborygmi. It is possible that the closure of the mouth of one of these diverticula is the explanation of the formation of a mesenteric cyst—a condition that otherwise is difficult to explain (see p. 507).

FIG. 685.—Diverticulum of the jejunum three hours after ingestion of barium. The patient was a woman of fifty-six, with a fourteen-years' history. The symptoms were ameliorated by excision of the diverticulum.

Harold Edwards, Contemporary. Surgeon, King's College Hospital, London.

Jejunal Diverticulosis and Steatorrhœa.—Jejunal diverticula are sometimes associated with macrocytic anæmia, steatorrhœa, and glossitis—a syndrome that has recently attracted considerable attention, but is mainly of medical interest.

Meckel's Diverticulum.—It will be recalled that this structure is present in 2 per cent. of the human race; that it is situated upon the anti-mesenteric border of the small intestine, 2 feet (60 cm.) from the ileo-cæcal valve, and that it is usually 2 inches (5 cm.) long. Useful as is this mnemonic, in the first place one need not be acquainted with abdominal surgery for any length of time to realise that the diverticulum may be of much greater length, sometimes up to 2 feet (60 cm.). Secondly, and of great importance, is the fact that in about 30 per cent. of cases the diverticulum occurs at a point between 3 and 5 feet (0·9 and 1·5 m.) proximal to the ileo-cæcal valve. Consequently it is unsafe to pronounce that the diverticulum is absent unless the last 5 feet of the ileum have been inspected.

In nearly 90 per cent. of cases the diverticulum arises from the anti-mesenteric border of the ileum, and being congenital this diverticulum possesses all three coats of the intestinal wall. In 20 per cent. of cases the mucosa of the diverticulum contains heterotopic epithelium, viz. gastric, colonic, or sometimes pancreatic tissue. When present, heterotopic tissue lines the greater part of the proximal end of the pouch, and not infrequently extends for a short distance into the nearby ileum. Although Meckel's diverticulum occurs with equal frequency in both sexes, symptomatic cases, due almost entirely to the epithelium contained in the diverticulum, are predominant in males. In order of frequency, these symptomatic cases are as follows:

1. **Severe hæmorrhage per rectum,** due to peptic ulceration, occurs most frequently between the ages of 10 and 15 years, but it is a mistake not to keep in mind the possibility of this source of hæmorrhage in adults. The blood passed is neither the bright red of a colonic lesion nor the typical, almost black, stool of melæna from a bleeding gastro-duodenal ulcer ; it is intermediate (maroon) in colour. A feature of value in diagnosis is that although the patient frequently vomits, the vomitus contains no blood. Seldom is the hæmorrhage preceded by pain ; sometimes bleeding precedes perforation. When operation for serious progressive hæmorrhage per rectum is required, and no lesion in the stomach or duodenum is found, always the next step should be the examination of the terminal ileum—in cases of bleeding from Meckel's diverticulum the neighbouring ileum, and the colon, contain much blood.

2. **As a Cause of Intussusception.**—In the majority of cases the leading point of the intussusception is swollen, inflamed, heterotopic epithelium at the mouth of the diverticulum—not inversion of the diverticulum, as is commonly stated. Intussusception due to Meckel's diverticulum is discussed on p. 573.

3. **Meckelian diverticulitis** with or without perforation. An important factor in the production of acute inflammation in the diverticulum is lodgement of coarse food residue or a sharp foreign body. The symptoms of Meckelian diverticulitis without perforation are those of acute appendicitis, and unless the appendix has been removed previously the diagnosis is impossible before operation. When a diverticulum perforates, so rapid is the onset of peritonitis that the symptoms simulate those of a perforated duodenal ulcer. Whether or not the diverticulum has perforated, urgent operation is required. In non-perforated cases an inflamed diverticulum should be sought as soon as it has been ascertained that the vermiform appendix (and, in the case of a female, the Fallopian tubes) is not culpable.

4. **Chronic Peptic Ulceration.**—The pain is similar to that of a duodenal ulcer, but unless the patient is a child, and some blood has been passed per rectum, usually

Johann Friedrich Meckel (The Younger), 1781–1833. Professor of Anatomy and Surgery, Halle.

the condition remains undiagnosed for long periods or until a serious complication arises, the reason for this being as follows :

Radiography.—In cases of Meckel's diverticulum giving rise to symptoms, failure to visualise the diverticulum by radiography after a barium meal, which is very common, is of no significance, because so often the entrance of the diverticulum is blocked by œdema.

' Silent ' Meckel's Diverticulum.—It is, of course, true that a Meckel's diverticulum may remain symptomless throughout life, and is found only at necropsy. When a silent Meckel's diverticulum is encountered in the course of an abdominal operation, the prevailing modern view is that provided the diverticulum can be excised without appreciable additional risk, this should be done. The wisdom of this advice has been confirmed by the frequency and severity of the complications that are liable to ensue in connection with the anomalous structure.

Exceptionally a Meckel's diverticulum is found in an inguinal or femoral hernial sac, when it is known as a Littre's hernia.

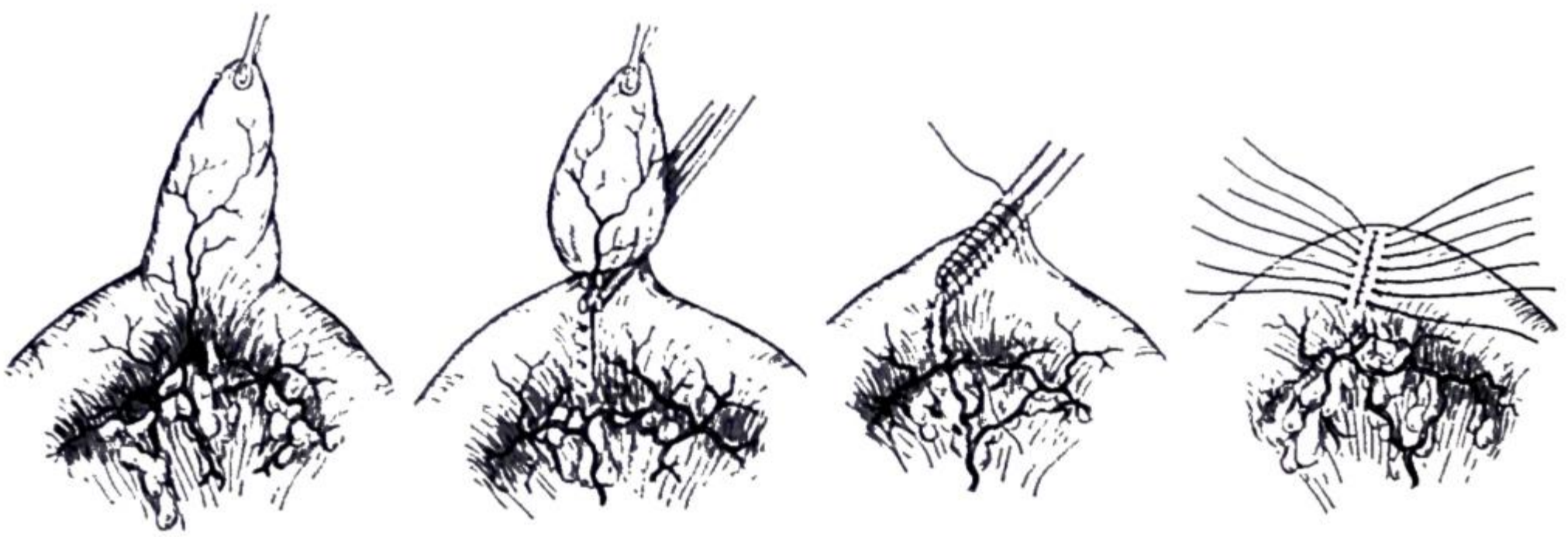
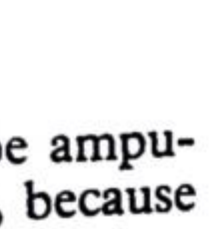


FIG. 686.—Steps in the performance of Meckelian diverticulectomy.

Meckelian Diverticulectomy.—A Meckel's diverticulum should not be amputated at its base and invaginated in the same way as a vermiform appendix, because often this is a cause of considerable narrowing of the intestine. Moreover, it does not remove completely heterotopic epithelium, if such be present. The steps of Meckelian diverticulectomy are clearly displayed in fig. 686, making further description unnecessary. Should there be considerable induration of the base of the diverticulum, and particularly when such induration extends into the neighbouring ileum, it is advisable to resect a short segment of the ileum containing the diverticulum and to restore the continuity of the bowel by anastomosis.

DIVERTICULA OF THE LARGE INTESTINE

Diverticulum of the cæcum is congenital, for it has a complete muscular coat. It is solitary, and situated on the medial aspect of the intestine just above the ileocæcal valve, viz. 

Its neck is narrow, and the diverticulum is subject to attacks of acute inflammation indistinguishable from acute appendicitis. When chronically inflamed it produces gross thickening of the ileo-cæcal region and dense adhesions, the cause of which may not be evident until the cæcum has been resected.

Cæcal Diverticulosis.—It is exceptional for colonic diverticulosis to extend into the cæcum.

Colonic Diverticulosis.—The presence of diverticula in the colon can be demonstrated in at least 5 per cent. of persons of both sexes over forty

Alexis Littre, 1658–1725. Surgeon and Anatomist, Paris.

years of age subjected to radiological examination after a barium enema. The primary seat of colonic diverticulosis is the pelvic colon (fig. 687); it never occurs below the peritoneal reflection. It is not uncommon for the diverticulosis to extend into the distal half of the descending colon, but proceeding upwards along the large intestine from that point, the diverticula become less numerous, and by the time the splenic flexure is reached, usually there are none. Sparse diverticulosis of the transverse colon is not a rarity. A diverticulum of the ascending colon is found in one per cent. of persons; its structure and ætiology are similar to that of a diverticulum of the cæcum.

FIG. 687.—Pelvic colon with multiple diverticula.

Pathogenesis.—Colonic diverticula contain no muscle in their walls. They are herniations of mucous membrane through the circular muscle coat between the mesenteric and the two antimesenteric tænia (fig. 688) at a point where an arteriole pierces the muscular wall. Consequently most of them are situated nearer the mesenteric than the antimesenteric border of the colon.

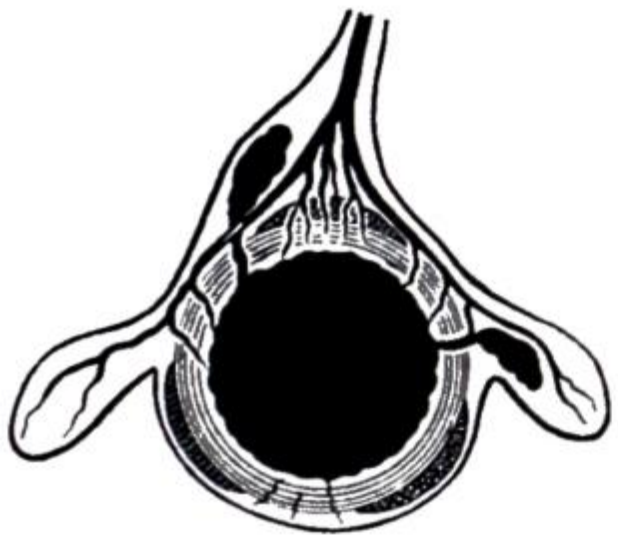


FIG. 688.—The usual sites of diverticulation of the colon. (*After Hamilton Drummond.*)

Sometimes the diverticulum passes into an appendix epiploica. As a rule, appendices epiploicæ of a diverticula-bearing pelvic colon are luxuriant and contain fat of a bright yellow hue. A segment of colon bearing the diverticula is always spastic with a diminished lumen.

Ætiology.—The cause of the diverticulosis has been attributed to increased intracolonic pressure due to chronic constipation or purgation and also to degeneration occurring in the parasympathetic ganglion cells of the affected large intestine.

Radiological Findings.—Colonic diverticula, as such, give rise to no symptoms. On radiographic examination after a barium meal, or better after a barium enema, the affected colon is more contracted than normal, and globular shadows in relation to the colon are apparent (see fig. 689). Sometimes only two or three are present; more often upwards of a score are visible. In early cases the diverticula empty as the colon empties. In cases of longer standing the shadows persist for a considerable time after the barium enema has been evacuated. Sometimes the diverticula are filled with fæcal concretions, in which case they may give a crescentic shadow, due to the interior of the diverticulum being filled only partially with barium. It is exceptional for colonic diverticula to be present in children and young adults.

COLONIC DIVERTICULITIS

Diverticulitis is a comparatively rare complication of colonic diverticulosis. With the increasing span of life, colonic diverticulitis has become more common during the past twenty-five years : the recorded death-rate from this condition in England and Wales has risen from 15 in 1930, to 725 in 1955.

Pathogenesis.—Many colonic diverticula are filled with inspissated fæcal matter. Should the neck of a diverticulum become partially blocked, chronic diverticulitis is likely to supervene. Similarly, if the products of inflammation cannot escape freely into the intestinal lumen, acute diverticulitis supervenes. Colonic diverticulitis is always occlusive in origin. The inflammatory process commences in *one* diverticulum : the resulting œdema is sufficient to occlude the openings of a number of diverticula adjacent to it. As a consequence, they too become secondarily infected. Incidentally, the occlusion of the orifice prevents barium entering the diverticula of the inflamed area (see fig. 689). The length of the colon thus involved is usually 4 to 10 inches (10 to 25 cm.).

In contradistinction to appendicitis, which is seldom chronic, colonic diverticulitis usually presents in a chronic form.

Chronic Colonic Diverticulitis.—By the time symptoms are sufficient to cause the patient to seek advice, chronic colonic diverticulitis is accompanied by peridiverticulitis.

Clinical Features.—The patient is over forty years of age; 75 per cent. are over the age of sixty. Men and women are affected equally; formerly it was stated that male patients predominated.

The clinical course is one of exacerbations and remissions. While the exacerbations last for a few days to more than a week, remissions for months or even a few years are not infrequent. In early cases flatulent dyspepsia and constipation are the main symptoms. As the condition progresses these give place to :

Pain is the most common complaint. During the attack it is generally constant, is situated in the left iliac fossa, but is not intense unless the associated peri-diverticulitis has caused irritation of the parietal peritoneum. The pain is worse on defæcation, or on being jolted, as when riding in a vehicle. Often there is a sense of fullness in the rectum, unrelieved by defæcation.

Periodic loose stools with the passage of some mucus is rather common at some time during the attack.

Hæmorrhage Per Rectum.—The old dictum ' if it bleeds, it must be a carcinoma ' must be abandoned; 20 per cent. of patients with colonic diverticulitis pass blood per rectum. Of recent years a large number have been reported in whom there was a short history of lower abdominal pain, followed by severe continued bleeding per rectum.

On Examination.—Tenderness in the left iliac fossa is characteristic. Sometimes a thickened, tender pelvic colon can be palpated in the left iliac fossa, or on bimanual pelvic examination. Frequently, however, the patient is obese, and deep tenderness in the left iliac fossa is the only physical sign that can be elicited.

Radiography.—Obviously the presence of colonic diverticula is no criterion that the patient is suffering from diverticulitis. What is extremely suggestive, if not diagnostic, is an area bereft of diverticula interposed in a segment of diverticula-bearing colon (fig. 689). The reason for this phenomenon is given on p. 524. Fixity of the segment, and tenderness over it, are also helpful radiological signs.

FIG. 689.—An area devoid of diverticula in the midst of the diverticula-bearing region of the colon is indicative that the patient has diverticulitis as well as diverticulosis. (*The late Dr. G. R. Mather Cordiner London.*)

Sigmoidoscopy. — Narrowing, rigidity, and a considerable excess of mucus in the sigmoid are the usual findings. The orifices of the diverticula are seldom observed because œdema of the mucous membrane occludes them.

Differential Diagnosis.—Many of the manifestations of chronic diverticulitis closely resemble those of a carcinoma of the colon. A long history favours diverticulitis. Abdominal pain is more common in cases of diverticulitis than when a carcinoma of the colon is present. Rectal bleeding occurs in 65 per cent. of cases of carcinoma, and in 20 per cent. of cases of colonic diverticulitis. A palpable mass is present in 25 per cent. of patients in each group, but tenderness, both abdominal and rectal, is more common in diverticulitis. Colonic diverticula revealed by a barium enema can, of course, be concomitant with carcinoma of the colon. If the growth takes the form of an ulcer or is of the papilliferous variety, radiological appearances are characteristic, as also are the sigmoidoscopy findings. The radiological appearances of an inflammatory and a malignant annular stricture are not always distinctive. Sigmoidoscopy shows a stricture in both peridiverticulitis and annular carcinoma. The absence of bleeding when the walls of the strictured portion are swabbed favours diverticulitis. On laparotomy there is no distinguishing feature and enlargement of the paracolic lymph nodes may be inflammatory. Even when the affected portion of the colon has been excised it is difficult to determine which of the two conditions is present, but should the mucous surface be ulcerated it usually proves to be a carcinoma. The final verdict in such cases rests on microscopical examination.

Similar difficulties occur when a vesico-colic fistula is present (see Chapter xxxiii).

Treatment:

(*a*) **Medical.**—In early cases medical treatment is sometimes effective. Purgatives are discarded and liquid paraffin substituted, and the diet should contain little residue. Three ounces (90 ml.) of liquid paraffin instilled into the rectum at bed-time, and retained all night, if possible, is helpful. Low-pressure saline enemata are useful, but they must be given by a skilled person. Courses of sulphathalidine or sulphasuccidine are given.

(*b*) **Operative Treatment:** *One-stage Resection.*—Many surgeons now

accept that the most satisfactory treatment for colonic diverticulitis is resection of the affected portion of the intestine, and not infrequently a one-stage operation is possible. This is suitable especially as an interval procedure. It is, perhaps, also indicated somewhat urgently in a patient with diverticulitis who recently has developed urinary symptoms, but in whom an actual colovesical fistula has not yet formed. Prompt primary resection in such cases prevents the occurrence of a fistula, which a simple defunctioning transverse colostomy cannot be relied upon to do (J. C. Goligher).

Most cases of acute hæmorrhage occurring as a complication of colonic diverticulitis cease with conservative treatment, allowing resection to be undertaken during a quiescent period. Occasionally the bleeding necessitates urgent resection of the involved bowel.

The technique of resection does not differ from that for carcinoma of the colon (see p. 549).

Complications.—The complications of colonic diverticulitis are rather frequent—all are serious. It is not unusual for symptoms to remain sufficiently in abeyance for the disease to progress to a stage of complication before the patient presents. Such complications are :

Chronic or acute-on-chronic intestinal obstruction due to peridiverticulitis of long standing resulting in stenosis of the affected segment of bowel. Intestinal obstruction can also arise from pericolonic adhesions implicating the small intestine.

A vesico-colic fistula is the most common variety of fistula associated with chronic colonic diverticulitis. It occurs as the result of the inflamed pelvic colon becoming adherent to the bladder. Often there are premonitory signs of frequency and dysuria, but it may occur without warning, the patient presenting entirely on account of symptoms of cystitis accompanied by pneumaturia. Vesico-colic fistula is considered in Chapter xxxiii.

Fistulous communications between the colon and the small intestine, or the skin, are by no means rare complications, but are encountered less commonly than in regional ileitis. The former is a cause of persistent diarrhœa.

It should be noted that there is not the slightest evidence that chronic diverticulitis is a precursor of colonic carcinoma.

Acute Diverticulitis.—In typical cases the pain commences at the umbilicus and passes to the left iliac fossa, where the maximum tenderness is situated. This makes the diagnosis from appendicitis simple. Even those very rare cases of left-sided vermiform appendicitis can be eliminated because in these, although the appendix is situated on the left, the pain and usually the tenderness is situated on the right. However, cases occur where the inflamed diverticulum is situated in a loop of colon lying in the pelvis, and tenderness is elicited only by pelvic examination. In such cases the differentiation between acute appendicitis and colonic diverticulitis is not apparent until laparotomy has been performed.

Treatment of uncomplicated acute colonic diverticulitis. When the diagnosis can be made with assurance, conservative treatment similar to that of delayed treatment of appendicitis (see p. 604) should be instituted. With

John Cedric Goligher, Contemporary. Professor of Surgery, University of Leeds.

such treatment resolution nearly always occurs. About a week later it is safe to give a barium enema. Assuming that diverticulosis of the pelvic colon can be demonstrated radiologically, and the patient is at least fairly robust, because it is almost certain that the patient will have future attacks of diverticulitis, perhaps with perforation, interval resection of the affected segment of the colon should be advised. Should, however, the patient be old and enfeebled, perhaps a watching policy under the care of a physician is the best course. In cases where the abdomen has been opened on the mistaken diagnosis of acute appendicitis, and acute colonic diverticulitis is found, what to do for the best is an occasion for exercising considerable surgical judgment. When the patient is obese and the colon fixed, undoubtedly the best course is to close the abdomen (it is justifiable to remove the vermiform appendix first) and return the patient to bed and to institute a strict Ochsner-Sherren régime. In the happy circumstances where the patient is not obese and the pelvic colon is mobile, to proceed to exteriorise it is a meritorious decision.

Acute free perforation of an inflamed colonic diverticulum is not a rarity. Free perforation is responsible for the majority of deaths from this disease, and a quarter of the patients have no premonitory symptoms of diverticulitis. The signs are those of diffuse peritonitis, and the radiological findings of a pneumoperitoneum tend to alter the original diagnosis of probable perforated appendicitis to one of perforated peptic ulcer. The fact that perforated colonic diverticulitis gives rise to a pneumoperitoneum even more readily than a perforated peptic ulcer should be appreciated more widely. With flatus and fæces pouring into the peritoneal cavity, most obviously operation with as little delay as possible will give the patient his only chance. In many instances, under the anæsthetic it is possible to feel a lump in the left iliac fossa, a cardinal sign that was not vouchsafed at the bedside because of overlying rigidity. The abdomen having been opened, the best course to adopt will vary with circumstances: if the colon is mobile and the abdominal wall not excessively fat-laden, exteriorisation of the loop and suprapubic drainage of the peritoneal cavity fulfils the highest objectives. When, as is frequently the case, exteriorisation is impractical, suture of the perforation, local sump drainage, and drainage of the peritoneal cavity is probably the best course ; some advocate a defunctioning transverse colostomy in addition. It is not infrequent to encounter an indurated colonic perforation where an attempt at suture, owing to the friability of the tissues, results in making the perforation larger. There is then but one very sound, fairly expeditious course—to resect the affected segment, to close the distal end, and bring the proximal end on to the surface at the upper end of the wound, and keep it there by transfixion of the mesocolon with a spigot. As before, local and suprapubic drainage is provided.

Localised peridiverticular abscess is a common complication of acute diverticulitis. As a rule, if sufficient time has elapsed to allow overlying rigidity to abate, a mass can be felt in the left iliac fossa.

Treatment.—In the first instance, the patient should be treated by the delayed method, the details of which are set out on p. 605. The indications

for opening a peridiverticular abscess do not differ from those of an appendix abscess. Sump drainage is, however, strongly recommended so that the suction can carry off any fæcal discharge that leaks from the perforation. A fæcal (colocutaneous) fistula is the usual sequel of draining a peridiverticular abscess, and in due course, when it is adjudicated that resolution of the inflammation is complete, resection of the affected segment and the colocutaneous fistula is to be recommended. In old and enfeebled patients, and others in whom, by reason of their poor general condition, resection is contraindicated, a colocutaneous fistula is some guarantee against the development of further abscesses (H. Edwards).

ULCERATIVE COLITIS

There has been a considerable increase in the number of cases of ulcerative colitis in England and Wales during the past decade, and a corresponding elevation in the death-rate from this disease. An increased incidence among Western peoples seems to have been fairly general ; however, it is surprising to learn that ulcerative colitis is rare in France.

Ætiology.—The cause is unknown. In spite of intensive bacteriological studies, no organisms or group of organisms can be incriminated. Evidence is accumulating that the disease is linked with the stress of modern life, and is psychosomatic in origin. In cases of extensive ulceration secondary infection plays a large part.

Pathology.—In the great majority of cases the disease commences in the rectosigmoid, and spreads upwards along the colon. Especially when the ileocæcal valve is incompetent, retrograde ileitis involving the last foot (30 cm.) of the ileum is liable to supervene ; this occurs in about 17 per cent. of cases, but it does not appear to influence prognosis.

Essentially the disease is characterised by the appearance of multiple minute ulcers—sometimes the ulcers are discrete ; at others there is a sea of ulceration. Microscopical evidence nearly always proves that the ulceration is more severe and extensive than the gross appearance indicated (Cuthbert Dukes). As time goes on the small ulcers are apt to coalesce to form larger ones, mainly due to the crypts of Lieberkühn becoming distended with pus and bursting into the bowel. When the ulceration extends into the submucosa it causes reflex muscle spasm, and the appearance of a stricture. In long-standing cases there is always considerable intramural fibrosis, causing the affected part of the colon to become permanently contracted.

Clinical Features.—Women are attacked somewhat more often than men. The onset of the disease is in the 3rd, 4th, and 2nd decade, in that order : exceptionally it is encountered in childhood. The first symptom is watery diarrhœa occurring in a person of previously normal bowel habit. Mucus (sometimes blood-stained) is present in the stools, and in cases of some standing pus from secondary infection of the ulcers is often detected on microscopical examination. Pain as an early symptom is unusual. The disease progresses by relapses and remission.

The course, and therefore the prognosis, of ulcerative colitis is unpre-

Cuthbert Dukes, Contemporary. Director of the Research Laboratories, St. Mark's Hospital, London.
Johann Lieberkühn, 1711–1756. Anatomist, Berlin. He demonstrated his anatomical preparations in London, and was awarded the F.R.S.

dictable. The fluctuations from week to week are remarkable, and there are but few diseases where the patient's condition can alter from week to week for better or worse with such rapidity (T. L. Hardy).

Two types are encountered :

Fulminating type (5 per cent.) is ushered in with a temperature of 102° to 103° F. (38·9° to 39·4° C.) and incessant diarrhœa containing blood, mucus, and pus ; the patient looks and feels very ill. In such circumstances the condition must be differentiated from bacillary or amœbic dysentery and typhoid. These infections are eliminated by bacteriological examinations of the stools, blood culture, and a Widal reaction. A scraping from the rectal wall obtained through a proctoscope may be necessary to rule out amœbic dysentery.

Chronic Type (95 per cent.). As a rule the initial attack is of moderate severity, but exacerbations at intervals of weeks, months or, in mild cases years, occur. As the disease progresses the patient becomes wasted from diarrhœa, and severely anæmic from loss of blood. Often during the attacks there are ten to twenty stools a day, accompanied by tenesmus. The frequency of the motions and the degree of invalidism go hand in hand, and are proportional to the extent of the involvement of the colon. Lesions of limited length are commonly found in milder cases ; when the whole colon is involved, seldom can the patient work, and in extreme instances she is rendered so weak as to be partially or wholly bed-ridden.

Radiology after a barium enema shows one of the following :

1. The earliest sign is loss of normal mucosal haustra in the pelvic colon. As the disease progresses, more of the colon is thus affected.

FIG. 690.—Advanced ulcerative colitis. Showing tubular contraction and shortening of the colon. *(Mr. W. B. Gabriel, London.)*

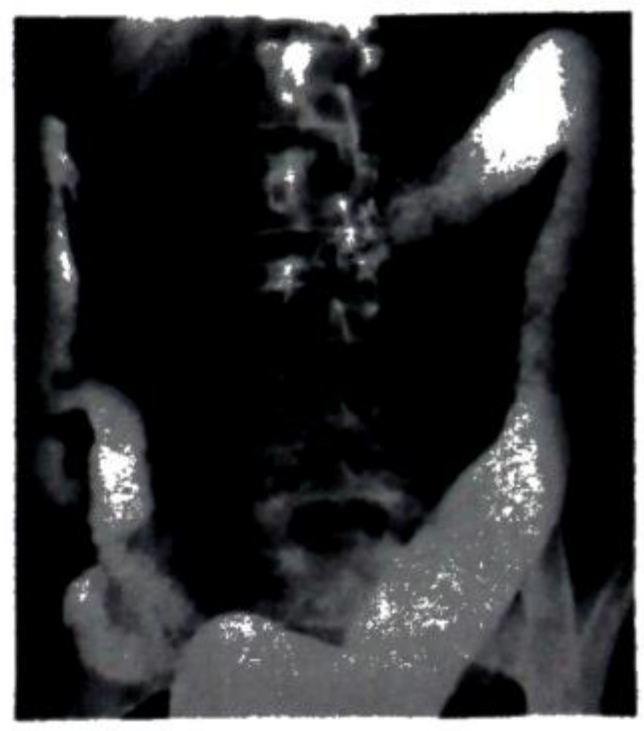


FIG. 691.—Ulcerative colitis showing pseudopolyposis. *(Dr. Clifford Hawkins, Birmingham.)*

2. The colon becomes narrower and much less distensible. Ulcer cavities can be demonstrated in severe cases.

3. The colon is reduced to a contracted tube (fig. 690).

4. In 10 per cent. of cases regeneration of islets of mucous membrane produce a condition of pseudopolyposis (fig. 691).

Thomas Lionel Hardy, Contemporary. Professor of Gastroenterology, University of Birmingham.
Fernand Widal, 1862–1929. Professor of Pathology and Physician, Hôpital Cochin, Paris.

Sigmoidoscopy is indispensable in the diagnosis of early cases and in mild cases when the pathological effects of the disease are insufficient to alter the barium shadow. The granular, reddened mucosa dotted with tiny ulcers (fig. 692) is very different from the picture seen in amœbic dysentery, where there are large deep ulcerations with the intervening mucosa comparatively healthy. As ulcerative colitis progresses, the ulceration may become so severe that practically no normal mucous membrane remains. In mild cases, when the passage of occasional blood and mucus per rectum is the only symptom, sigmoidoscopy may be the means of differentiating the condition from a carcinoma of the pelvic colon.

FIG. 692.—Ulcerative colitis. The granular inflamed appearance of the mucosa is characteristic. (*After the late J. P. Lockhart-Mummery.*)

A barium enema and sigmoidoscopy should be avoided during the acute phase.

Exfoliative Cytology.—The epithelial cells shed in ulcerative colitis are normal in one-third of cases, but in two-thirds large cells with large and prominent nuclei abound. To obtain specimens of colonic epithelial cells S. C. Truelove employs a long metal rod with a perspex head (fig. 693).

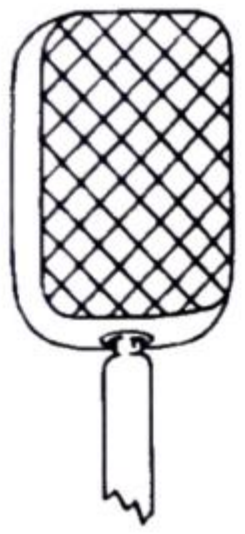


FIG. 693.—Perspex head for obtaining a specimen of colonic epithelium. (*After S. C. Truelove.*)

Complications :

Pseudo-polyposis occurs in 10 per cent. of cases, and only in those of considerable standing. Pseudo-polyps are not neoplastic and are not liable to undergo malignant degeneration (Cuthbert Dukes).

Carcinoma.—An adenocarcinoma supervenes in about 5 per cent. of cases of ulcerative colitis of long standing. The probable cause is continual regeneration of epithelium in excess of normal requirements, and such a carcinoma grows and metastasises with exceptional rapidity.

Fibrous stricture follows successful medical treatment, with or without ileostomy, in 12 per cent. of cases. Most usually the stricture is situated at the recto-sigmoid junction or in the anal canal. In the colon itself, should a stricture develop, it is nearly always malignant.

Perforation of an ulcer into the general peritoneal cavity occurs usually in the fulminating type of the disease. Excessive steroid therapy is a predisposing cause of perforation, especially slow perforation.

Massive hæmorrhage from the bowel is rare, but in half the cases in which it occurs, in spite of blood transfusion, the patient succumbs. Once again excessive steroid therapy predisposes to this serious complication.

Recto-vaginal fistula, or a colo-vaginal fistula, sometimes complicates long-standing cases in females.

Perianal fistula, preceded by an ischio-rectal abscess, occurs in about 5 per cent. of late cases, and follows perforation of the rectal wall by an ulcer.

Fatty infiltration of the liver is not uncommon. The heavy drain of protein in the fæcal discharges is probably the explanation.

Polyarthritis occurs in 10 per cent. of cases. Pain and swelling of some joints may occur during any episode of ulcerative colitis. Usually it subsides during the phase of remission, but there is progressive joint damage with each attack. Another form is slow, progressive insidious arthritis, often accompanying moderate colonic symptoms.

Dermatological Lesions.—Urticaria, erythematous and pustular rashes, purpura, erythema nodosum and ulcers of the leg occur with considerable frequency, especially in patients in whom arthritis has developed.

Sidney Charles Truelove, Contemporary. Assistant Physician, The Radcliffe Infirmary, Oxford.

Treatment.—The correction of fluid and electrolytic depletion, particularly that of potassium, is important. Intravenous dextrose-saline solution is required in severe cases. In necessary cases potassium deficiency is corrected by adding potassium chloride in appropriate quantities to the dextrose-saline solution. Potassium loss is a pre-operative problem that ileostomy tends to correct.

When necessary, blood transfusion should be instituted and continued until the hæmoglobin has reached a level of about 80 per cent. Liver extract, given parenterally, is also indicated. There must be a high caloric intake (at least 3,000 calories per day). The aim is to provide a large amount of protein and carbohydrates with a minimum of fat and a low residue. Fluids thickened with skimmed milk powder provide increased protein. In those with anorexia a fortified intragastric drip should be given at night.

Dextrinised starch, malt, lactose, and chocolate are valuable. In cases of extreme wasting casein hydrolysate, 100 G. (3½ ounces) daily, is given, preferably by mouth but, if necessary, intravenously so that amino-acids can be absorbed without the necessity of being digested previously. Animal charcoal in 2-drachm (8-ml.) doses, or kaolin ½ ounce (15 G.), helps to relieve the discomfort from flatulence. Vitamins are given to supplement those contained in the diet. The administration of vitamin B_{12} is valuable. Vitamin D is especially required because of the low content of fat in the diet. Possibly vitamin K_1 will help to control the bleeding. To combat anæmia, which is often serious, multiple small transfusions are more effective than large, less frequent ones. Phthalylsulphathiazole is administered in large doses, 1 G. (15 grains) four-hourly for six weeks. Chloromycetin is helpful in some cases because of its bactericidal effect on streptococci and coli groups of organisms. Aureomycin and other antibiotics have been given, with a view to reducing infection without, as a rule, any lasting beneficial effect.

Morphine is necessary in early severe cases. Later a combination of phenobarbitone, codeine, and belladonna is useful.

Cortisone therapy appears to exert a beneficial influence in acute and severe cases of fairly recent onset. The usual dose is 100 mg. a day for three weeks, followed by smaller doses (say 75 mg.) a day for a similar period : sometimes initial doses up to 300 mg. a day are given. However, its effect is far from permanent, and it is often negligible in the established disease. The main value of cortisone therapy is as an adjuvant measure in the pre-operative preparation of the patient.

Dangers of Prolonged Cortisone Therapy.—1. Not only is there an increased risk of massive hæmorrhage and of free perforation into the peritoneal cavity, but in patients who have not responded to a course of cortisone or similar steroid over a period of four to six weeks, the colonic wall becomes excessively friable and in some areas disintegrates, its place being taken by the parietes or adjacent viscera, usually the small bowel. In such cases the surgeon may then be unable either to remove the colon or even to perform ileostomy (B. N. Brooke).

2. Cortisone therapy renders the patient more susceptible to pyogenic infection ; therefore it is wise to administer penicillin in addition.

3. J. A. Bargen has little use for steroids ; he finds that patients become addicted to them.

The chief indication for the exhibition of steroid therapy is in fulminating cases, as an alternative to ileostomy performed during the ultra-acute phase (G. Crile Jnr.).

Bryan Nicholas Brooke, Contemporary. Reader in Surgery, University of Birmingham.
Jacob Arnold Bargen, Contemporary. Chief of the Department of Intestinal Diseases, Mayo Clinic, Rochester, Min.
George Crile Jnr., Contemporary. Chief Surgeon, Cleveland Clinic, Cleveland, Ohio.

Salicylazosulphapyridine (4 to 6 G. daily) is far better than cortisone for general use. Indeed, it is apparently a specific remedy (J. A. Bargen).

Operative treatment.—*Indications:*

1. Acute fulminating cases, after a trial of steroid therapy.
2. Pseudo-polyposis, or possible malignant change.
3. Strictures or fæcal fistula have occurred.
4. Serious hæmorrhage.
5. Perforation.
6. Associated arthritis.
7. Skin complications, especially dermatitis and ulceration of the legs.
8. The most usual of all—when medical treatment has failed.

At the present time between 25 and 50 per cent. of sufferers are recommended for surgical treatment by their physicians.

In cases of perforation into the general peritoneal cavity, provided the patient can withstand it, urgent colectomy and ileostomy probably hold out the best chance of survival (H. E. Lockhart-Mummery). As is more usual when the patient's condition is parlous, closure of the perforation, peritoneal toilet, drainage of the peritoneal cavity (particularly in the region of the perforation), plus ileostomy, should be carried out. In desperate cases exteriorisation of the affected segment (should it be mobile) with drainage of the peritoneal cavity, is the best that can be done.

In all other instances when surgical treatment is advised, about six days' intensive pre-operative preparation by the methods already enumerated is carried out. During this time the skin of the abdominal wall is toughened by the application of tannic-acid jelly, in order to withstand excoriation by escaping intestinal juices.

Ileostomy *per se*.—As a rule, ileostomy is performed with a view to resection of the colon when the patient is fit to withstand that procedure. In the majority of instances the ileostomy is permanent, and its success depends a great deal on certain principles in technique.

A disc of skin the size of a florin (3 cm. in diameter) is removed at least 2 inches (5 cm.) below and external to the umbilicus (fig. 694). The abdomen is opened by a left paramedian incision and the ileum is divided near its termination, together with its mesentery. The distal end is closed and invaginated into the cæcum. Alternatively, some surgeons prefer to divide the ileum more proximally (a most desirable step if the terminal ileum is diseased also), and, avoiding all stitches, to bring the distal cut end out on to the surface through a stab incision 2 inches (5 cm.) above the ileostomy opening.

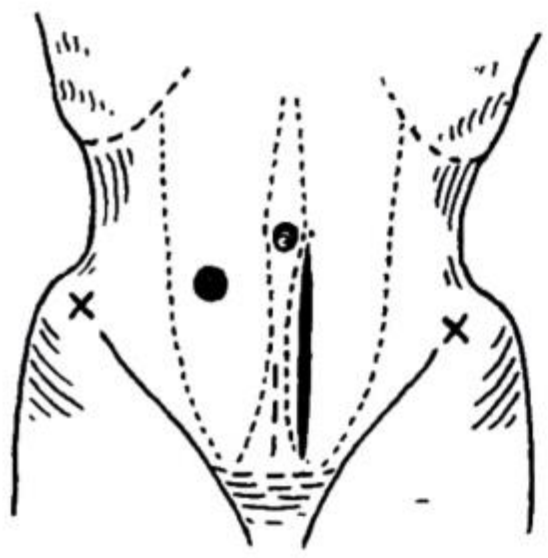


FIG. 694.—Site of the ileostomy. The 'trephine' wound should be at least 2 inches (5 cm.) from all scars and bony points.

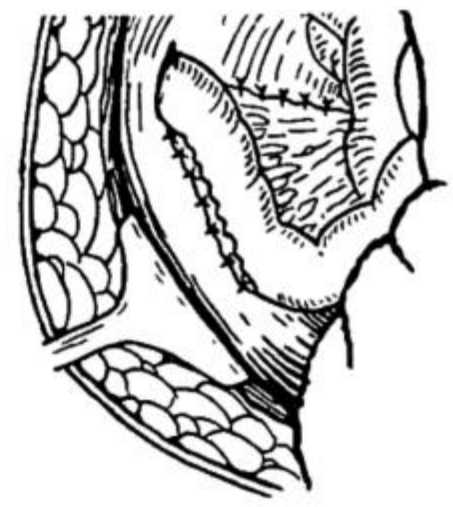


FIG. 695.—Suture of the mesentery to the parietal peritoneum, to prevent prolapse. (*After J. C Goligher.*)

Important steps in the performance of ileostomy are (*a*) to close the peritoneal space on the external side of the ileostomy (otherwise there will be a tunnel here that invites intestinal obstruction), and (*b*) to anchor the anterior edge of the mesentery to the parietal peritoneum (fig. 695). The latter prevents prolapse of the mucous membrane. About 2 inches of the divided ileum (closed temporarily with a non-crushing clamp) is brought through the split rectus

Hugh Evelyn Lockhart-Mummery, Contemporary. Surgeon, St. Mark's Hospital, London.

muscle, and its periphery is stitched to the skin edges prepared previously for its reception (fig. 696). The abdomen is closed. An ileostomy appliance is affixed to the skin forthwith.

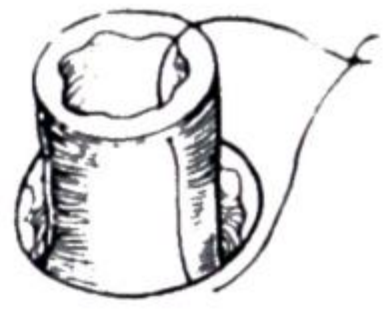


FIG. 696.—Suturing the free extremity of the proximal ileum to the skin edges.

During the early post-operative period fluid balance must be adjusted with great care. Excoriation of the skin by liquid fæces can be minimised by giving isogel by mouth, and by the continued use of an ileostomy bag (fig. 697). This bag,[1] which is fixed to the skin by latex rubber adhesive, has greatly reduced the tribulations inseparable from an ileal abdominal anus.

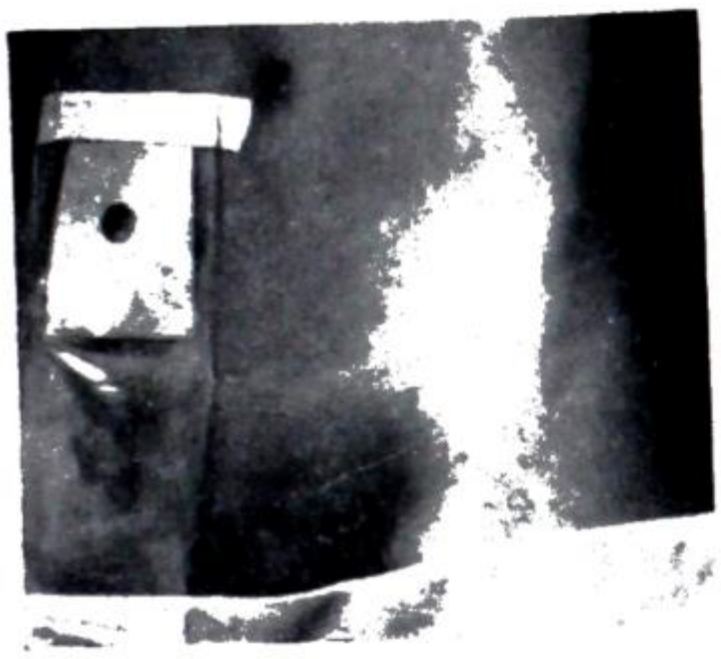


FIG. 697.—Disposable ileostomy bag.

The skin of the abdominal wall requires great attention to prevent excoriation, some degree of which is inevitable. A paste of aluminium 10 parts and zinc oxide 90 parts is perhaps the best of many applications. The stools thicken in a few weeks, and are usually semi-solid in a few months. The patient should restrict his diet slightly to eliminate fruit pips and skins, that are liable to cause abdominal colic.

Subsequent colectomy is undertaken as soon as improvement is maintained.

The previous scar having been excised, the abdomen is opened through a left paramedian incision, freeing the *distal* ileal stump from its stab wound exit, if such be present. The cæcum and ascending colon are detached from their mesenteries, and the colectomy is continued. The rectum being freed as far as the pelvic diaphragm (levatores ani), it is transected at this point. The large intestine having been removed, the distal end of the rectum is closed and the pelvic peritoneum united over it.

Provided the patient is even moderately fit, the modern trend is to undertake colectomy in addition to ileostomy at the primary operation. Colectomy is not a difficult operation when it is performed on an emaciated individual whose peritoneal cavity is free from adhesions. After a successful ileostomy both these desiderata are absent; firstly, the patient has gained weight, and secondly, adhesions are bound to be present at least in the right iliac fossa.

The Stump of the Rectum.—Whether the patient should be left with a permanent ileostomy, or whether ileo-rectal anastomosis should be carried out either at the time of the colectomy or at a later period, remains highly controversial. The majority of surgeons do not favour the anastomosis; they find that intractible proctitis often mars this otherwise desirable procedure.

CHRONIC MUCOMEMBRANOUS COLITIS (*syn.* MUCOUS COLITIS)

Chronic mucomembranous colitis is a definite clinical entity characterised by obstinate constipation with mucus in the stools. In this condition *there is no ulceration of the colonic mucous membrane.* Mucomembranous colitis is usually encountered in women about forty years of age. The patient is rich

[1] Mr. Koenig, Contemporary, Chemist, and W. A. Rutzen, Surgical Instrument Manufacturer of Chicago, invented this type of bag, modifications of which are now made in Great Britain.

rather than poor, and there is always a neurotic element. In advanced cases the patient's whole life is mainly concerned with her bowels, often because she has little else to occupy her mind. Not infrequently there is a spastic condition of the colon, which can often be palpated in the left iliac fossa. This tonic contraction of the colon is aggravated by purgation, and is a cause of abdominal pain, situated mainly in the left iliac fossa.

Sigmoidoscopy reveals normal mucous membrane. Spasm of the circular muscle throws the mucous membrane into folds and tends to make the instrument difficult to advance.

Treatment is entirely medical, and often it is unsatisfactory. At least temporary benefit is obtained by a visit to one of the spas, such as Plombières, where colonic wash-outs are given.

ACUTE PSEUDOMEMBRANOUS (POST-OPERATIVE) ENTEROCOLITIS (*syn.* STAPHYLOCOCCAL ENTEROCOLITIS)

There has been a disconcerting increase in the incidence of this alarming, and frequently fatal, variety of enterocolitis: an increase that coincides with the advent of broad spectrum antibiotics. None the less, post-operative pseudomembranous enterocolitis was known in the pre-antibiotic era; consequently the oral administration of antibiotics cannot be held entirely responsible, although there is little doubt that it accounts for the increased frequency of the condition.

Ætiology.—Prevailing opinion indicates that the initial, and occasionally the only cause is intense vasoconstriction of intestinal blood-vessels consequent upon shock; this vasoconstriction imperils the vitality of the intestinal mucous membrane to such an extent that some of its cells perish. The other cause (which in some instances operates single-handed) is suppression of normal intestinal flora by antibiotics resulting in a preponderance of one or more species that assumes the rôle of a pathogen. Resistant staphylococci overwhelmingly predominate, but on occasions *B. proteus* cannot be exonerated.

Pathology.—In the most severe form, areas of necrotic mucous membrane are shed, or they remain as a pseudomembrane attached to the deeper layers of the intestinal wall. The lower ileum is chiefly affected.

Clinical Features.—The diarrhœa is often cholera-like, and commences abruptly or, in patients undergoing gastro-intestinal aspiration, the aspirate becomes foul and alarmingly excessive. Collapse in the peripheral circulation follows so rapidly that, without effective treatment, death may occur within seventy-two hours. Advanced age, lowered resistance, and malnutrition each can play an important part.

Treatment.—*Firstly* the broad spectrum antibiotic having been discontinued, no effort is spared to restore the blood pressure and the fluid and electrolytic balance. The amount of fluid loss in these cases is prodigious (10–20 litres per day) and this must be replaced. The administration of noradrenaline in adequate doses to maintain blood pressure is important. Erythromycin is the most effective drug against the staphylococcus, although there is a rapid increase in the number of strains resistant even to this anti-

biotic. Bacitracin can also be used. When the organism is isolated and its sensitivity investigated, the bacteriologist's report should influence the selection of an appropriate antibiotic.

Secondly the great loss of protein requires blood transfusion. The caloric requirements can be met in part by intravenous 10 per cent. dextrose. The danger of disseminating the infection precludes the use of ACTH in the early phases, but it can be administered with safety and advantage when culture of the stools indicates that infection has been controlled. The oral administration of a preparation containing lactobacillus helps in the restoration of the normal intestinal flora.

THE SURGICAL ASPECTS OF INTESTINAL AMŒBIASIS

Amœbiasis denotes an infestation with *Entamœba histolytica*. Contrary to general belief, *E. histolytica* is not strictly a tropical parasite; it has a world-wide distribution, and is found in about 3 per cent. of the inhabitants of Great Britain. It is surprisingly common in overcrowded institutions. The reason why in some cases it becomes pathogenic is unknown.

Life History of the Parasite.—The active form of the parasite or trophozoite lives in the intestinal mucous membrane, where it ingests red blood corpuscles and other cells, and multiplies by mitosis. Should the parasite become pathogenic, it makes its way into the follicles of Lieberkühn, and by dissolving interglandular tissue by cytolysins, submucous loculi are produced. Some of these burst through the mucous membrane to become amœbic ulcers. While the trophozoites continue their activities in the base of the ulcer, others cease to feed, migrate towards the surface,

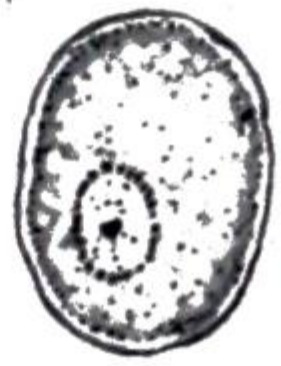


FIG. 698.—*Entamœba histolytica*, cystic stage. In this form, usually in drinking water, the parasite is transferred from one host to another. (*After Sir Philip Manson-Bahr.*)

and become transformed into cysts (fig. 698). In due course the cysts are swept into the lumen of the bowel and pass into the outer world with the fæces. Amœbiasis is transmitted mainly in drinking water.

Pathology.—The ulcers, which have been described as 'bottle-necked' because of their considerably undermined edges, have a yellow necrotic floor (fig. 699), from which blood and pus exude. While on rare occasions the ulcers are scattered throughout the large intestine, in 75 per cent. they are confined to the lower sigmoid and the upper rectum. In rare instances they are limited to the cæcum.

FIG. 699.—Sigmoidoscopic findings in amœbic dysentery (*After M. A. Arafa.*)

Biopsy material is obtained by scraping the suspected area of mucosa with a long-handled Volkmann's spoon; this removes a thin slice of the diseased mucosa, which is examined after covering it with a cover glass that is pressed so as to squash the specimen. The examination must be made immediately. It should be noted particularly that the finding of *Entamœba histolytica* is not conclusive evidence that the symptoms are due to amœbiasis. On numerous occasions a positive slide examination has caused the clinician to jump to the conclusion that the diagnosis has been established when, in point of fact, there has been an early carcinoma further up the colon and the *Entamœba histolytica* have been non-pathogenic and incidental.

Richard von Volkmann, 1830–1889. Professor of Surgery, Halle.

Clinical Features.—Dysentery is only one manifestation of the disease. In various guises amœbiasis obtrudes itself into the surgeon's diagnostic arena :

Appendicitis or Amœbic Typhlitis[1] *?*—In tropical countries where amœbiasis is endemic, this is a constantly recurring problem requiring considerable surgical judgement. To operate upon a patient with amœbic dysentery without the precautions described below is to invite an exacerbation of amœbiasis that may prove fatal. Especially in cases where a palpable mass is present, the bowel is friable and satisfactory closure of the appendix stump becomes difficult or impossible. The death-rate from peritonitis and wound infection in the notorious Chicago epidemic of amœbiasis in 1933 was appalling, which emphasises that surgeons in temperate climates should be familiar with the condition. In the case of amœbic typhlitis there is rarely rigidity, and pain commences in the right iliac fossa. In amœbic typhlitis there are two characteristic and localised zones of tenderness on deep palpation—one over the cæcum and one over the sigmoid. The latter is sharply defined, and being comparable to McBurney's point on the right side, it has been named aptly ' *the amœbic point* ' by Sir Philip Manson-Bahr. Routine sigmoidoscopy is of great value. If real doubt exists as to the differential diagnosis, 1 grain (60 mg.) of emetine hydrochloride in 20 ml. (5 drachms) of normal saline solution given intravenously very slowly is likely to produce substantial amelioration of symptoms within two hours (A. T. Andreasen).

Perforation.—The most common sites are the cæcum and recto-sigmoid. Usually perforation occurs into a confined space where adhesions have previously formed, and a pericolic abscess results, which eventually needs draining. When there is sudden fæcal flooding of the general peritoneal cavity, often life must be despaired of, although A. T. Andreasen and others have had success by free drainage of the region of the perforation, followed by gastro-intestinal aspiration, intravenous fluid replacement, antibiotic therapy, and a full course of emetine.

Alarming rectal hæmorrhage due to the separation of slough is liable to occur.

Granuloma.—Progressive amœbic invasion of the wall of the rectum or colon, with secondary inflammation, may produce a granulomatous mass indistinguishable from a carcinoma. The exhibition of emetine as a therapeutic test will prevent mistakes in diagnosis. Amœbiasis and carcinoma occasionally co-exist (Naunton Morgan).

Fibrous stricture may follow the healing of extensive secondary infection of amœbic ulcers.

Intestinal obstruction is a common complication of amœbiasis, and the obstruction is due to the adhesions associated with pericolitis and a large granuloma.

Paracolic abscess, ischio-rectal abscess, and fistula from perforation by amœbæ of the intestinal wall followed by secondary infection.

Hæmorrhoids frequently become acutely inflamed during an exacerbation of dysentery. The amœbiasis should be treated before the hæmorrhoids.

Treatment.—There is no remedy that can compare with emetine injections in the acute stage. In the chronic stages emetine bismuth iodide, given in gelatin capsules in amounts to a total of 30 grains over a period of ten days is usually extremely effective. It should be noted that enteric-coated capsules are useless—they pass through the intestine unchanged. The quinoxyl group of drugs, including diiodoquin, are also effective in the chronic stage, and can be given with advantage during the interval between courses of emetine.

THE SURGICAL COMPLICATIONS OF TYPHOID AND PARATYPHOID

Chloromycetin exerts a rapidly curative effect on typhoid and paratyphoid infections ; consequently complications are less frequently encountered than formerly. When any of the surgical complications of typhoid arise, chloromycetin should be given in addition to other necessary treatment, not forgetting that this antibiotic destroys the organisms responsible for the production of vitamin B complex which must be replaced.

1. *Paralytic ileus* is the commonest complication of typhoid. The treatment does not differ from that described on p. 582.

2. *Intestinal hæmorrhage* may be the leading symptom. In three cases in our practice torrential rectal hæmorrhage has been the first indication of a typhoid infection. The

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[1] Typhlitis = inflammation of the cæcum.

Charles McBurney, 1845-1913. Surgeon, Roosevelt Hospital, New York.
Sir Philip Manson-Bahr, Contemporary. Consulting Physician to the Hospital for Tropical Diseases, London.
Anthony Turner Andreasen, Contemporary. Formerly Professor of Surgery, University of Calcutta.
Clifford Naunton Morgan, Contemporary. Surgeon, St. Mark's Hospital, London.

condition must be distinguished from purpura with intestinal symptoms, and intussusception. A Widal reaction should be employed and, if negative, repeated in suspected cases. Urgent blood transfusion will be required.

3. *Perforation.*—Perforation of a typhoid ulcer usually occurs during the third week; occasionally it is the first intimation of the disease (ambulatory typhoid). The ulcer is longitudinal to the long axis of the gut (fig. 700A), and in the case of

FIG. 700A.—A typhoid ulcer is longitudinal to the long axis of the gut. (Peyer's patch necrosis.)

FIG. 700B.—A tuberculous ulcer is transverse (because it follows the lymphatics).

typhoid it is situated in the lower ileum. In paratyphoid B, perforation of the large intestine sometimes occurs. The usual treatment is to perform laparotomy under local anæsthesia, and to close the perforation. The results have been so poor that the conservative treatment of peritonitis plus chloromycetin has been tried, and yields better results.

4. Spontaneous rupture of the enlarged *spleen* has been recorded.

5. *Liver abscess* is a rare complication of paratyphoid fever.

6. *Cholecystitis.*—Acute typhoid cholecystitis is common among typhoid patients (p. 443). Gall-stones occasionally contain typhoid bacilli. Chronic typhoid cholecystitis can result in the patient becoming a typhoid carrier.

7. *Phlebitis.*—Venous thrombosis, particularly of the left common iliac vein, is a not very infrequent complication of typhoid fever.

8. *Genito-urinary Complications.*—Typhoid cystitis, pyelitis, bacilluria, and epididymo-orchitis all occur.

9. *Joints.*—All degrees of arthritis, from a mild effusion to suppuration, occur rather commonly as a complication of this disease.

10. *Bone.*—Typhoid osteomyelitis and typhoid spine are discussed in Chap. XLVII.

11. *Larynx.*—Typhoid perichondritis is met with occasionally, and typhoid laryngitis has been known to obstruct the airway.

REGIONAL ILEITIS (*syn.* REGIONAL ENTERITIS; CROHN'S DISEASE)

Ætiology.—No causative organism has been found in the lesion or in the stools. Dogs receiving finely powdered silicates admixed with their food for long periods develop an identical condition, and it seems possible that the ingestion by the ileum of fine, insoluble particulate matter such as toothpaste is the cause of regional ileitis. A disease similar to regional ileitis occurs in swine. The difficulty of distinguishing hypertrophic ileocæcal tuberculosis from regional ileitis on clinical, radiological, and even histopathological bases, has suggested that regional ileitis may be a stage in the healing process of tuberculosis (S. S. Anand).

Pathology.—Regional ileitis is essentially a cicatrising inflammation with ulceration of the mucosa. It usually commences at or near the ileo-cæcal valve, and extends upwards along the ileum for about 12 inches (30 cm.), but as little as 2 inches (5 cm.), and, more often, as much as 4 feet (1·2 metres) may be implicated. In acute cases the affected intestine is seen to be swollen, bright pink in colour, and with a fibrinous exudate on its peritoneal surface; in chronic cases hyperæmia is less in evidence. On palpation the intestinal wall feels like a hose-pipe. The mesentery of the involved intestine is exceedingly thickened, œdematous, and contains enlarged and fleshy lymph nodes. Unlike tuberculosis, the affected lymph nodes neither break down nor calcify. Tracing the diseased ileum upwards, it terminates abruptly in normal intestine. Above this commonly there is another (short) area of diseased intestine: this is a so-called 'skip' lesion. Doubtless a comparatively inconspicuous additional lesion is sometimes overlooked, and

Johann Conrad Peyer, 1653-1721. Successively Professor of Logic, Rhetoric, and Medicine at Schaffhausen, Switzerland.
Burrill B. Crohn, Contemporary, Physician, Mount Sinai Hospital, New York, first described this disease in 1932.
Santokh Singh Anand, Contemporary. Professor of Surgery, Amritsar, India.

is one cause of recurrence after resection. In 6 per cent. of cases there is an extension into the cæcum and at times the ascending colon is implicated in the interrupted manner referred to. Examples of primary Crohn's disease of the colon, jejunum, duodenum, and even of the stomach have been reported, which justifies the newer term, regional enteritis.

Pathological Histology.—A characteristic finding is granulomatous infiltration of lymphatics of the submucosa with the presence of non-caseating giant-celled systems. In the late stages of the disease fibrosis extends into and obliterates the submucosa, but usually giant-celled systems can be found in the related mesenteric lymph nodes.

Clinical Features.—The disease, which is independent of age, sex, social and economic conditions, or geographical location, is increasing in frequency. To some extent it is familial.

Acute regional ileitis occurs only in 5 per cent. of cases. The symptoms and signs resemble those of acute appendicitis, with one notable exception, viz. almost invariably diarrhœa precedes the acute attack. Exceptionally, perforation of the intestine, resulting in local or diffuse peritonitis, occurs.

Chronic regional ileitis is the usual form of the disease. It can be divided into three stages, but sometimes the second stage is lacking.

First Stage.—There is a history of mild diarrhœa extending over months or years, occurring continuously or in bouts accompanied by intestinal colic, relieved by defæcation. Intermittent pyrexia, seldom more than 99° F. (37·2° C.) is usual, but some patients are afebrile throughout. As a rule a tender mass can be felt in the right iliac fossa, and frequently by a pelvic examination also. There is often a moderate secondary anæmia. Occult blood and some mucus is present in the stools; two-thirds of the patients have some degree of steatorrhœa. A perianal abscess is a frequent accompaniment of early Crohn's disease. The cause is probably an infected anal crypt associated with the concomitant diarrhœa. The high incidence and diagnostic significance of perianal and perirectal abscesses and fistulæ in patients with regional ileitis has become incontestable.

FIG. 701.—Crohn's disease. Stage of cicatricial contracture. (*Sir Lancelot Barrington-Ward and R. E. Norrish.*)

Second stage is characterised by symptoms of acute or chronic

intestinal obstruction. Cicatrisation of the granulomatous area has progressed to such an extent that the lumen of the affected portion of the intestine is narrowed (fig. 701).

Third stage is that of adhesions sometimes accompanied by slow perforation of the intestinal wall. Adhesions are dense, abscess formation is common, and fistulous tracts are wont to develop :

(*a*) Internally into neighbouring hollow viscera, notably into a redundant pelvic colon, but occasionally into the bladder. In all cases of enterocolic and vesico-intestinal fistulæ the possibility of regional ileitis should be considered.

(*b*) Externally, nearly always through the scar of a previous operation for the condition, e.g. appendicectomy, resection of implicated intestine.

Radiological Diagnosis.—X-ray examination after a barium meal often shows lack of segmentation and feeble or absent peristalsis in the affected portion of the intestine, the lumen of which remains constant in diameter. Radiologically, cases can be divided into stenosing and non-stenosing. In the non-stenosing phase straightening of the valvulæ conniventes is characteristic. When ulceration has occurred multiple defects (cobblestone reticulation) can be seen after the barium has been evacuated from the segment in question. When cicatrisation has occurred the radiograph is particularly characteristic ; sometimes the terminal ileum is so constricted that what is known as the ' string ' sign of Kantor (fig. 702) is present.

FIG. 702.—The 'string' sign of Kantor.

It should be noted that detection of regional ileitis radiologically is not in itself an indication for operation.

Treatment:

Medical Treatment.—In the early stages of the disease medical treatment should be given an extended trial. It consists of a long period of strict rest in bed and a high protein diet with vitamin supplementation. Exacerbations are treated with sulphathaladine or chloramphenicol orally, or streptomycin intramuscularly. Cortisone or ACTH sometimes bring about dramatic symptomatic improvement, but the effect on the long-term course of the disease is insignificant.

Operation.—Should the abdomen be opened on the mistaken diagnosis of acute appendicitis and acute regional ileitis is found, the one thing *not* to do is to remove the appendix. Appendicectomy frequently determines the development of an external fistula (see above). The correct procedure is to close the abdomen forthwith. Occasionally the condition resolves completely ; more often chronic ileitis supervenes.

In chronic cases operation usually is required eventually. Intestinal obstruction, internal or external fistulæ, or failure of the patient to thrive after a long period of medical treatment, all call for operation. Resection of the

John Leonard Kantor, Contemporary. Gastroenterologist, Presbyterian Hospital, New York.

affected segment of intestine and restoration of the continuity of the apparently healthy intestine has been given an extended trial, and is still practised in some clinics : there is an appreciable mortality and an inordinate recurrence rate.

Consequently, unless circumstances, such as an enterocolic fistula, leave no other alternative, more and more surgeons are following the advice of Dr. Crohn and his surgical colleagues, who for many years have recommended a more conservative course. It is division of healthy ileum 6 in. (15 cm.) above the diseased portion, with closure of both ends, followed by ileo-transverse colostomy. The mortality of this operation is almost zero, and the results are at least as good as those following resection. In the few cases that do not improve or respond in spite of this short circuit, resection still can be undertaken.

TUBERCULOSIS OF THE INTESTINE

In Britain tuberculosis of the intestine has become infrequent. Nevertheless, in countries where the Public Health control of tuberculosis is less strict, the disease is still common.

1. **Ulcerative type** is always secondary to pulmonary tuberculosis, and probably arises as the result of swallowing tubercle bacilli. It is characterised by the presence of multiple ulcers in the terminal ileum, the long axis of each ulcer lying transversely (fig. 703). The serous coat overlying the ulcerated segment is thickened, injected, and sparsely bespattered with tubercles. Perforation is rare, but in patients who overcome the infection late stricture or strictures of the ileum are rather frequent.

FIG. 703. — Tuberculous ulcer of the ileum. Placed transversely, it is known as the 'girdle' ulcer. (*Professor A. K. Toufeeq, Lahore, Pakistan.*)

Clinical Features.—Diarrhœa is the over-riding symptom ; there is also loss of weight. The stools have a fœtid odour, and contain peptone, pus, and occult blood. Often the patient has received, or is receiving, treatment for pulmonary tuberculosis ; more rarely pulmonary tuberculosis is disclosed for the first time in the course of the investigations.

Radiology.—A barium meal often discloses complete absence of filling of the lower ileum, the cæcum, and most of the ascending colon (Stierlin's defect) due to hypermotility of the ulcerated segment.

Treatment is sanatorium régime and antituberculous chemotherapy, and provided the intestinal ulceration is not a terminal event of advanced pulmonary tuberculosis, healing often occurs. Operation is required in the rare event of perforation. Remotely, from time to time cicatrisation causes intestinal obstruction, and calls for surgical intervention.

2. **Hyperplastic tuberculosis** occurs most commonly in the ileo-cæcal region, although solitary or multiple lesions of the lower ileum are met with occasionally. This form of intestinal tuberculosis is consequent upon the ingestion of *Mycobacterium tuberculosis* by a patient with a high resistance

Eduard Stierlin, 1878–1919. Professor of Surgery, Munich.

to the organism. In Western countries *mycobacterium tuberculosis bovis* is accepted as the causative organism, while in the East the human variety is the culprit (A. K. Toufeeq). The infection establishes itself in lymphoid follicles, and spreads to the submucous and subserous planes. The resulting chronic inflammation causes much thickening of the intestinal wall, and consequent narrowing of its lumen. The vermiform appendix, which becomes involved in the mass, occasionally is the seat of the primary infection. There is early involvement of regional lymph nodes, which on occasion reveal caseation. Unlike regional ileitis (which in many respects this disease simulates) abscess and fistula formation is rare.

Untreated, sooner or later subacute or acute intestinal obstruction supervenes, and in the East, not infrequently impaction of an enterolith within the narrowed lumen is the precipitating cause.

Clinical features.—Attacks of abdominal pain with intermittent diarrhœa and constipation are the premonitory symptoms. Rarely is there

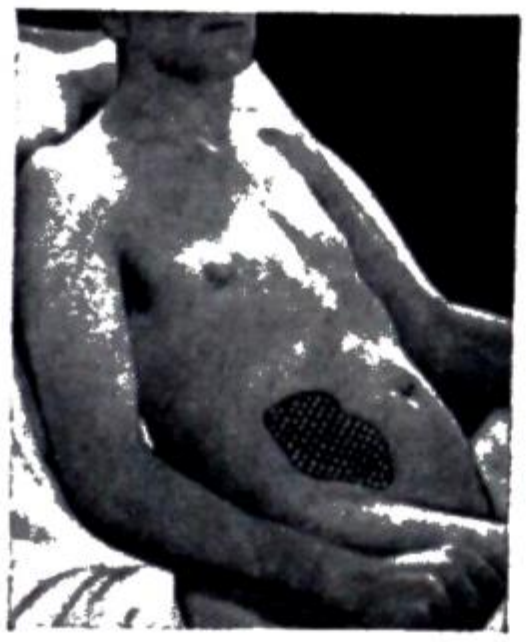

FIG. 704.—The difficulties in diagnosis of a mass connected with the cæcum. In this case the lump outlined on the skin was thought at first to be an appendix abscess. After the cæcum and ascending colon had been excised the specimen had many of the appearances of tuberculosis. Histologically the mass proved to be a carcinoma.

evening pyrexia, as is so usual in other manifestations of tuberculosis. Early in the course of the disease a mobile swelling becomes palpable in the right iliac fossa; later the lump becomes fixed to the posterior parietes. At this stage the nature of the mass often presents a perplexing diagnostic problem (fig. 704).

FIG. 705.—Ileocæcal tuberculosis. The defect seen occurs also in carcinoma of the cæcum and is known as Stierlin's filling defect. (*Professor A. K. Toufeeq, Lahore, Pakistan.*)

Radiography.—In a well-established case a barium meal reveals a long, narrow filling defect consisting of the terminal ileum and ascending colon, lying vertically. The cæcum becomes subhepatic (fig. 705).

Treatment.—When the diagnosis is certain, and the patient has not yet developed obstructive symptoms, sanatorium treatment with antituberculous chemotherapy is advised, but in all cases operative treatment is required. Right hemicolectomy with removal of the diseased segment of ileum is the treatment of choice. In patients with intestinal obstruction, and those in poor general condition, a defunctioning ileocolostomy similar to that recommended for regional ileitis is the best course. In necessary cases resection can be undertaken later, for fre-

Ahmed Khan Toufeeq, Contemporary. Professor of Surgery, Sir Ganga Ram Hospital, Lahore, Pakistan.

quently striking improvement occurs after ileocolostomy and subsequent general treatment.

ACTINOMYCOSIS OF THE RIGHT ILIAC FOSSA

Actinomycosis develops in the abdomen when a breach of the intestinal wall caused by other disease or trauma of a foreign body permits the escape of *Actinomyces israeli* into the tissue spaces. The fungus has been found in teeth scrapings, fæces, and even in the saliva of individuals with a healthy mouth. Abdominal actinomycosis, which is comparatively rare, occurs with equal frequency in rural and urban communities. Unlike intestinal tuberculosis, cicatrisation and consequent narrowing of the lumen of the intestine does not occur, neither do the mesenteric lymph nodes become involved. However, suppuration supervenes, and the disease spreads into the retroperitoneal tissues. Unchecked, the abdominal wall becomes the seat of multiple indurated discharging sinuses, and eventually the liver becomes involved by way of the portal vein.

Clinical Features.—The usual history is that appendicectomy has been performed for acute or subacute appendicitis. Possibly had the appendix been subjected to histological scrutiny streptothrix actinomyces would have been found. More usually this examination is omitted or the fungus is not discovered, and about three weeks after the operation a mass forms in the right iliac fossa, and a little later still the wound commences to discharge. At first the purulent discharge is thin and watery ; later, because of secondary infection, it becomes thicker and odorous. Other sinuses form, and fæcal fistulæ are liable to develop. At any stage of the disease, if pus is collected and allowed to trickle down the side of a test-tube, sulphur granules may be discovered. The pus should be kept warm and sent for immediate bacteriological examination. Several examinations are frequently required before the streptothrix is found.

Another clinical type is that of a patient, most usually a young adult male, who presents with vague abdominal pain. On examination a hard, slightly tender mass is found in the right iliac fossa. Extension of the disease to the psoas muscle sometimes causes fixation of the hip in flexion. Such a finding is characteristic, but is only present when the condition is fairly advanced. Little or no help is derived from radiology in distinguishing actinomycosis of the right iliac fossa from hypertrophic tuberculosis or carcinoma of the cæcum ; a deformity of the cæcum is found in all these conditions. Actinomycosis rarely gives rise to obstructive symptoms. When, as is sometimes the case, there is a history of a more or less sudden onset of pain some weeks previously, a subsiding appendix abscess is probably diagnosed, but the mass does not resolve. There is loss of weight, anæmia, and occasional pyrexia. A negative patch test helps to eliminate tuberculosis, but in most instances laparotomy is performed with one of the following findings : (*a*) an abscess is encountered and drained ; (*b*) the mass is found to be densely adherent to the posterior abdominal wall, and irremovable ; (*c*) the mass is sufficiently mobile for right hemi-colectomy to be carried out. Unfortunately,

James Israel, 1848–1926. Chief Surgeon, Jewish Hospital, Berlin.

the last procedure is often followed by actinomycotic fistula formation at the site of the anastomosis, but this can sometimes be prevented by recognition of the condition from the pathological specimen and early intensive general treatment.

Treatment.—Antibiotic treatment, continued for three or four months, is often curative. Penicillin, streptomycin, and aureomycin have all proved effective, depending upon the strain of actinomyces present.

PNEUMATOSIS CYSTOIDES INTESTINALES

Multiple gas cysts of the intestinal wall are pathological curiosities. Translucent, thin-walled cysts ranging from 1 to 2 or more centimetres in diameter, containing gas, mainly nitrogen, and having a lining of flattened cells of doubtful origin, occur in clusters under the serosa or in the mesenteries of the intestines. The condition, which probably is allied to the intestinal emphysema of swine, nearly always affects the small intestine, but occasionally the colon, and even the rectum, are implicated. It is believed that air enters a breach in the mucosa, as would occur in the case, say, of a duodenal ulcer, and the air is driven onwards by peristalsis. The cysts, which are well seen on a plain radiograph, are usually symptomless. Cases have occurred where the cysts so occlude the lumen of the intestine that resection has become necessary.

TUMOURS OF THE SMALL INTESTINE

Compared with the large intestine, the small intestine is rarely the seat of a neoplasm.

Benign.—Adenoma, submucous lipoma and leiomyoma occur from time to time, and sometimes reveal themselves by causing an intussusception. Indeed, a neoplasm of the small intestine is the commonest cause of intussusception in adults. The second most common complication is intestinal bleeding from an adenoma, in which event the diagnosis is frequently long delayed because the tumour is overlooked at a radiological examination by barium meal, and often at operation as well, to be discovered perhaps only after a second or third operation. In only 79 cases among 1,014 was the correct preoperative diagnosis made (L. P. River).

Peutz's syndrome consists of (*a*) familial intestinal adenomatous polyposis affecting mainly the jejunum, where it is a cause of hæmorrhage, and often of intussusception, and (*b*) melanosis of the oral mucous membrane and the lips. The melanosis takes the form of melanin spots which are sometimes present on the digits and the peri-anal skin, but the pigmentation of the lips (fig. 706) is the *sine qua non* of this portion of the syndrome.

FIG. 706.—Melanin spots on the lips of a patient afflicted with Peutz's syndrome.

Treatment.—As a malignant change occurs in at least 25 per cent. of the intestinal adenomatous polyps, elective operation or operations by serial enterotomies or resection of short lengths of affected intestine, is more than justifiable.

Malignant :

Sarcoma (40 per cent.).—Lymphosarcoma and spindle-cell sarcoma—more usually the former—occur in the first five decades, the average age of the patient being thirty-five years. The neoplasm tends to convert the affected intestine into a rigid tube without much interference with the size of its lumen until the disease is advanced. Loss of weight and anæmia are the chief early symptoms. Perforation into the peritoneal cavity occurs more often than is the case with other neoplasms of the small intestine.

Carcinoma (35 per cent.) occurs at the usual carcinomatous age. The jejunum is affected three times more often than the ileum. The most frequent symptoms are those of intestinal obstruction, and in about 40 per cent. of cases a palpable tumour is present. Because the content of the small intestine is fluid, by the time intestinal obstruction has ensued metastasis has occurred. Another train of symptoms is

Louis Philip River, Contemporary. Surgeon, Cook County Hospital, Chicago.
John Law Augustine Peutz, 1886–1957. Chief Specialist for Internal Medicine, St. John's Hospital, The Hague, Holland.

dyspepsia associated with melæna and increasing anæmia, in which case a tumour, often of the papilliferous variety, may be revealed radiologically after a barium meal.

Carcinoid (argentaffin) tumour (25 per cent.). The terminal ileum is the second most frequent site for this rare tumour. Unlike carcinoid tumour of the vermiform appendix (the most common site) the tumour metastasises to the regional lymph nodes and to the liver if it is not excised at a comparatively early stage. What is of considerable interest is that in some instances the metastases, particularly hepatic metastases, are sometimes associated with attacks of flushing (fig. 707), due to high concentrations of 5-hydroxytryptamine in the blood. In some instances, after excision of the primary tumour metastases regress.

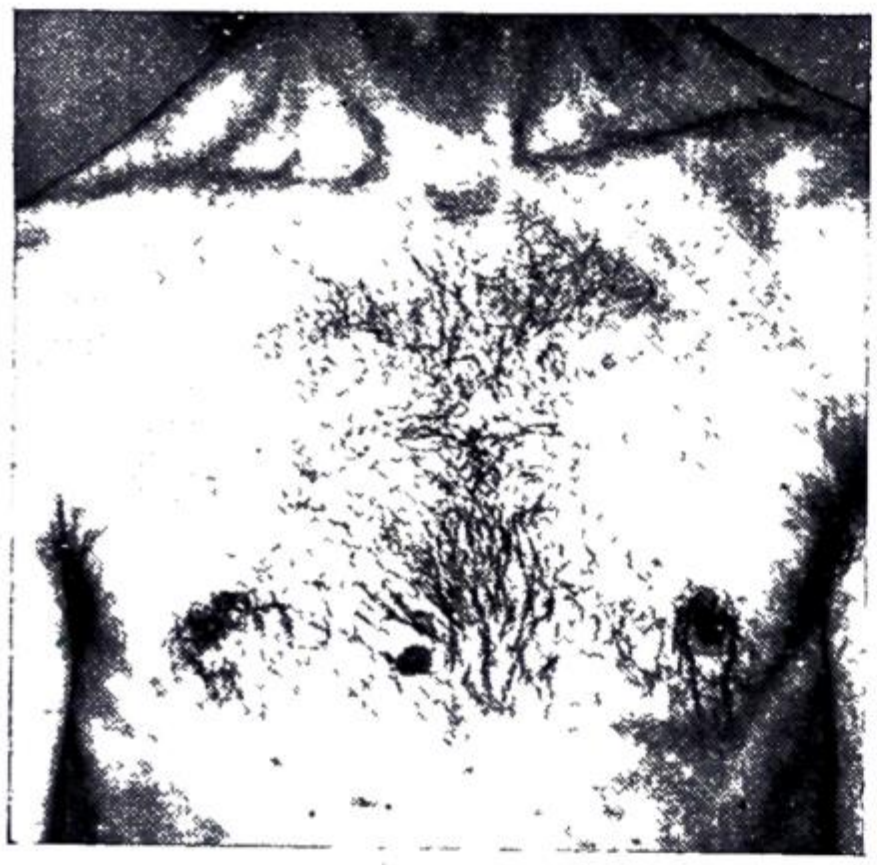

FIG. 707.—Patient with secondary carcinoid tumour of the liver (primary in the terminal ileum) exhibiting flushing after a small dose of alcohol. (*Dr. P. J. D. Snow, Manchester.*) (*The Lancet.*)

Treatment.—In the case of a pedunculated benign tumour, sometimes extirpation can be effected by opening the intestine after applying light clamps above and below the tumour-bearing area. In all other instances of tumours of the small intestine, wide resection of that portion of the intestine bearing the neoplasm, together with its mesentery, followed by end-to-end anastomosis, is indicated.

Prognosis.—Malignant tumours of the small intestine have an evil reputation. However, in the case of carcinoma of the lower ileum, as opposed to the jejunum, with wide resection the prognosis is fair, while if removed reasonably early, the prognosis of carcinoid tumour is frequently excellent: should a solitary metastasis be present in the liver, it also should be resected.

TUMOURS OF THE LARGE INTESTINE

Benign

Adenoma.—Considerable confusion has arisen between the terms adenoma and adenomatous polyp, the more so because often the 'adenomatous' is omitted, and the neoplasm is called a 'polyp', a term which, in point of fact, should be reserved for a chronic inflammatory condition (see p. 530). So-called adenomatous polyps are adenomata with an extremely well-developed central fibrous core (fig. 708), whereas an adenoma of the colon is sessile (fig. 709). A solitary adenoma of the colon is acquired (see below for the multiple familial variety). It occurs in patients over forty years of age, and unless it is pedunculated, it cannot be distinguished macroscopically from a papilliferous carcinoma. At the Christie Hospital, Manchester, 25 per cent. of the adenomas of the colon were found to be cylindromas (H. M. Russell). A cylindroma has a worse prognosis than a carcinoma. In specimens of papilliferous carcinoma of the colon, often tiny adenomata are found adjacent to the parent tumour.

Treatment.—As a rule an adenoma of the colon should be treated by limited resection of that part of the intestine in which it is contained. Only in this way can recurrence and a carcinomatous change be prevented. On the other hand, if the adenoma is situated in a portion of the terminal colon it can be removed by fulguration through a sigmoidoscope. The patient should be kept under intermittent observation for many years to come.

Helen May Russell, Contemporary. Consulting Pathologist, Christie Hospital, Manchester.

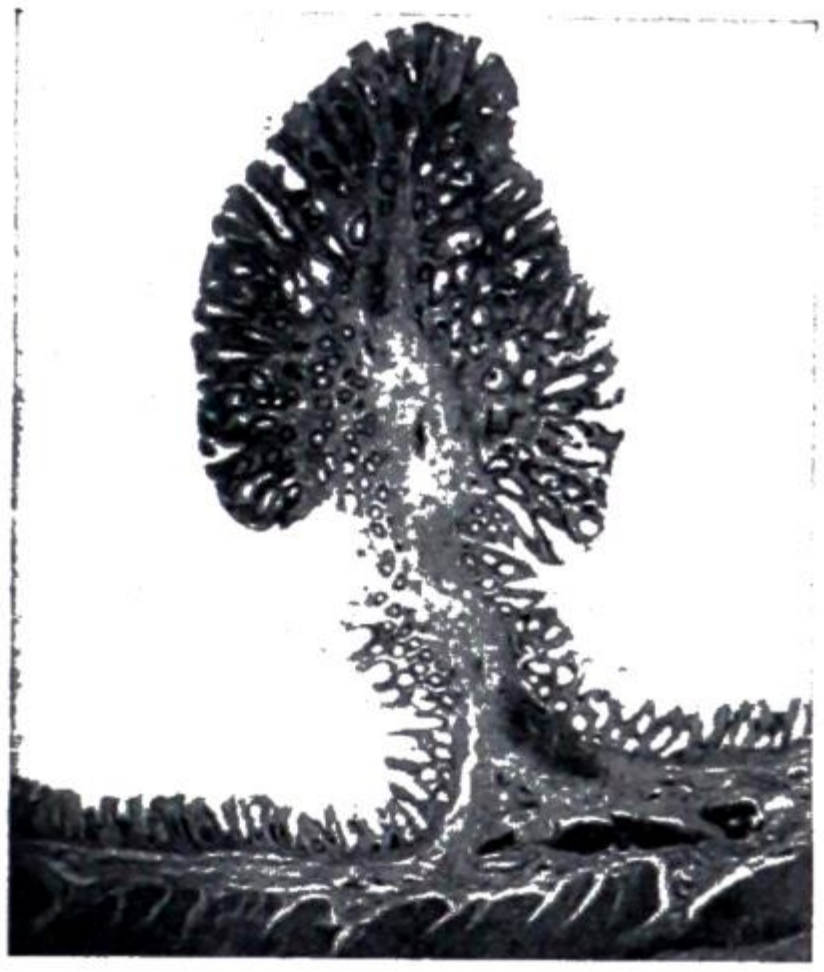

FIG. 708.—Pedunculated adenomatous polyp of the large intestine. Longitudinal section. (*Mr. J. H. Saint, Santa Barbara, California.*)

FIG. 709.—Adenoma of the colon. (*Mr. Max Pemberton, Enfield, Middlesex.*)

Familial Adenomatous Polyposis of the Colon, which must be distinguished from Peutz's syndrome (see p. 543), is transmitted from both sexes to both sexes, though males are affected more frequently than females. H. E. Lockhart-Mummery and Cuthbert Dukes have studied 1069 members of 58 families; of these members 218 have proved to have polyposis, and 154 of them developed colonic carcinoma. The adenomatous polypi are most frequently situated in the sigmoid and the rectum, but they often extend into the descending colon and transverse colon, though rarely into the ascending colon. Often hundreds of tumours are present. The patient complains of attacks of lower abdominal pain associated with loss of weight, diarrhœa, and tenesmus, and the passage of blood and mucus, and sometimes pus—all symptoms very like those of ulcerative colitis. A rectal examination may reveal one or more of these adenomatous polyps. Sigmoidoscopy shows a variety of neoplasms ranging from small sessile pink elevations to pedunculated tumours. A barium enema, especially a contrast barium enema (see p. 549), outlines the larger adenomatous polypi. Whenever the disease is diagnosed all members of the family[1] should be examined and continue to be examined at six-monthly intervals.

Treatment.—In early cases when the polypi are limited to the recto-sigmoid, fulguration through a sigmoidoscope, followed by sigmoidoscopic examination at regular intervals, may prove curative. Much more often a malignant change takes place in one or several of the tumours.

If a decision to operate is reached, complete colectomy is advisable. A stump of rectum can remain, the reasons being (*a*) the risk of carcinoma in the rectal stump is small; (*b*) an artificial anus is avoided; (*c*) other members of the family can be persuaded more easily to undergo the operation before late symptoms develop.

Hæmangioma.—A localised submucous telangiectasis is the cause of bleeding, which is often profuse. When the lesion is beyond the sigmoidoscopy field often the only method of detecting it is to operate while the bleeding is in progress. The distribution of blood within the intestine is noted; scrutiny of the blood-containing portion of the intestine usually reveals a dilated leash of veins in one portion of the mesocolon. Here the intestine is opened and the lesion, which it often small, is found. Resection of the area is the best course.

Lipoma is less frequently encountered in the large than in the small intestine, or in the rectum. Lipomata of the large intestine are almost confined to the cæcum. The tumour is submucous and in more than half the cases it is the cause of an intussusception. Other symptoms to which it gives rise are almost impossible to distinguish from those of a carcinoma of this region; even macroscopical or micro-

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[1] A small family affected by multiple sebaceous cysts as well as familial polyposis of the colon has been described by M. C. Oldfield.

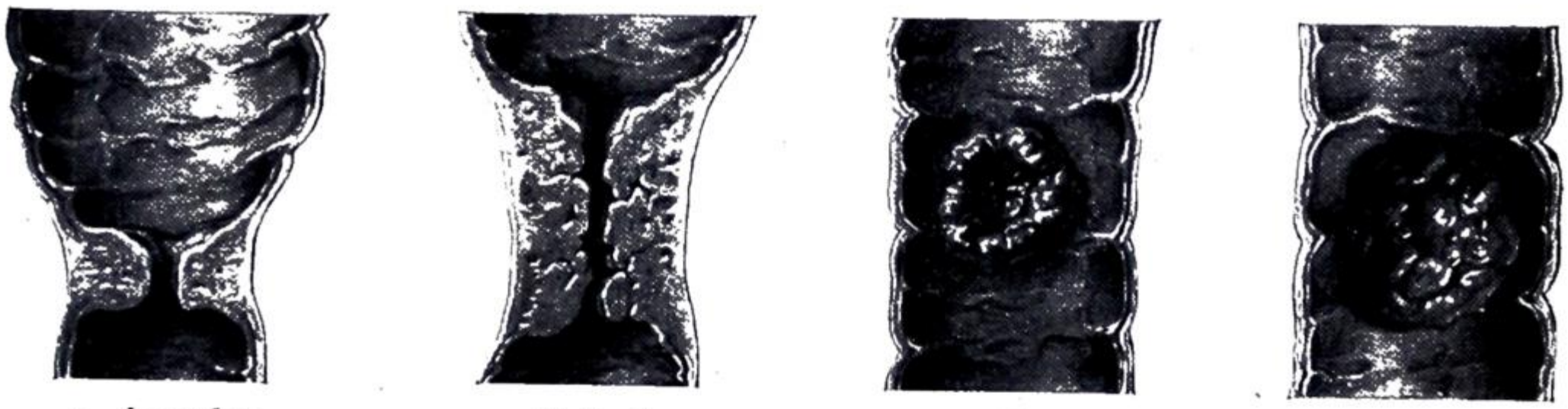
Michael Whitaker Carlton Oldfield, Contemporary. Surgeon, The General Infirmary, Leeds.

scopical blood is found in the stools. A barium meal may suggest a lipoma because of its smooth contour.

Malignant

CARCINOMA OF THE COLON

Pathology.—Microscopically, the neoplasm is a columnar-celled carcinoma originating in the epithelial cells that line the large intestine, or in the crypts of Lieberkühn. Macroscopically the growth takes one of 4 forms :



1. Annular. 2. Tubular. 3. Ulcer. 4. Cauliflower.

FIG. 710.—The 4 macroscopical varieties of carcinoma of the colon.

Type 4 is the least malignant form, and it is now certain that this papilliferous carcinoma commences as a benign adenoma. The annular variety carries a relatively good prognosis, not because the growth is of low-grade malignancy, but because it gives rise to early obstructive symptoms, and therefore is often extirpated before metastases have occurred (G. Grey Turner).

Site.—The most frequent site is toward the termination of the colon, viz. the pelvic colon and the rectosigmoid junction (fig. 711).

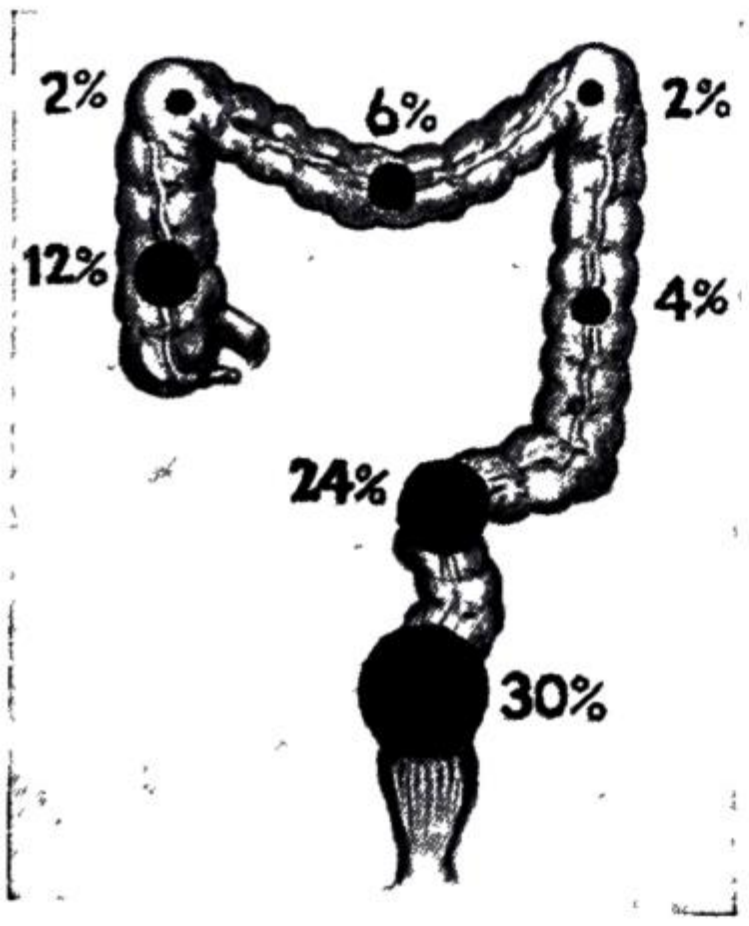


FIG. 711.—Relative frequency of carcinoma in various portions of the large intestine omitting the rectum proper. (*Mayo Clinic statistics.*)

The Spread of Carcinoma of the Colon.—Generally speaking, carcinoma coli is a comparatively slowly growing neoplasm, and if extirpated thoroughly at a reasonably early period a cure can be hopefully anticipated.

Local Spread.—The growth is limited to the bowel for a considerable time. It spreads round the intestinal wall, and to a certain extent longitudinally, but usually it causes intestinal obstruction before it has penetrated adjacent structures. Particularly in the ulcerative variety, penetration of the serous coat is apt to occur and, according to the segment involved, the abdominal wall, stomach, small intestine, spleen, the bladder, or uterus then becomes invaded by the growth. When a hollow viscus is thus implicated an internal fistula results ; in other instances the perforation may lead to the formation of a local abscess and an external fæcal fistula.

Lymphatic Spread.—The lymph nodes draining the colon are grouped as follows :

1. *The epicolic lymph nodes*, situated in the immediate vicinity of the bowel wall.
2. *The paracolic lymph nodes*, lying in relationship to the leash of blood-vessels proceeding to the colonic walls.
3. *The intermediate lymph nodes*, arranged along the ileo-colic, right colic, mid-

George Grey Turner, 1877-1951. Professor of Surgery, Post-Graduate Medical School, London.

colic, left colic, and the sigmoid arteries. In the last instance the paracolic nodes are often absent.

4. *The main lymph nodes*, aggregated around the superior and inferior mesenteric vessels, where they take origin from the abdominal aorta.

Spread by the blood-stream occurs late. Metastases are carried to the liver.

Clinical Features.—Carcinoma of the colon usually occurs in patients over fifty years of age, but it is not rare earlier in adult life. Exceptionally it appears in childhood, when on account of delayed diagnosis the prognosis is very poor.

Taking all sites into consideration, men are attacked more frequently than women (3 : 2), although carcinoma of the ascending colon is encountered more often in women.

While certain outstanding symptoms frequently prevail in all types (see fig. 710) and at all sites (see fig. 711), it is interesting and instructive to contrast and to compare the symptoms produced by a carcinoma of the left side of the colon with that of the right, and to refer to the symptomatology of carcinoma of the sigmoid and that of the transverse colon.

Symptoms	*Right Colon*	*Left Colon*	*Sigmoid*
Abdominal pain	78	68	51
Alteration of bowel habit	30	58	70
Loss of weight	50	15	20
Vomiting (frequently with colic)	32	15	0
Anorexia	18	9	0
Faintness, breathlessness	20	9	6
Bleeding per rectum	8	9	29
Lump present	67	46	39
Indigestion	8	0	0
Acute-on-chronic obstruction	8	21	29

The duration of symptoms is extremely variable; 5·5 months is the average.

(*E. G. Muir's statistics.*)

Carcinoma of the Left Side of the Colon.—As will be seen on reference to fig. 711, in about 75 per cent. of cases the neoplasm is situated on the left side of the colon. Neoplasms in this situation usually are of the stenosing variety (fig. 710, 1 and 2) and as here the fæcal content is relatively solid, and the lumen of the bowel relatively narrow, the main symptoms are those of increasing intestinal obstruction in about 25 per cent. of cases.

Pain is often a dull ache situated across the lower abdomen; later, intestinal colic supervenes.

Alteration of Bowel Habit.—An adult who has had regular bowel movement all his life, in a short space of time develops irregularity. The patient often states that he has *increasing* difficulty in getting the bowels to move, and that he has to take *increasing* doses of purgatives. Because of the drastic purgation, or on account of irritation by the scybala above the constricting neoplasm, attacks of constipation are followed by diarrhœa.

Palpable Lump.—Very often the lump that is felt on abdominal, rectal, or abdomino-rectal examination, is not the growth itself, but impacted fæces above it (J. B. Oldham). When the carcinoma is situated in a pendulous

Edward Grainger Muir, Contemporary. Surgeon, King's College Hospital, London.
James Bagot Oldham, Contemporary. Surgeon, Royal Liverpool United Hospitals, Liverpool.

pelvic colon a hard movable swelling is likely to be felt in the rectovesical pouch.

Distension.—Lower abdominal distension is not uncommon, and, like the pain, is relieved by passing flatus.

Carcinoma of the sigmoid follows the general pattern of the above, but there are differences.

Pain, when it occurs, is usually colicky from the commencement.

Tenesmus.—Growths of papilliferous type situated low in the colon are inclined to give rise to a feeling of the need for evacuation, which may result in tenesmus accompanied by the passage of mucus and blood, especially in the early morning.

Bladder symptoms are not unusual, and in some instances they herald colo-vesical fistula (see Chapter XXXIII).

Carcinoma of the transverse colon is frequently mistaken for a carcinoma of the stomach because of the position of the tumour and anæmia and lassitude that it sometimes engenders.

Carcinoma of the cæcum and ascending colon present in several guises:

(*a*) Anæmia, severe and unyielding to treatment, is a frequent predominating feature. Should a palpable tumour be present, the diagnosis is, to some extent, simplified.

(*b*) The presence of a mass in the right iliac fossa often proves a diagnostic conundrum (see fig. 704, p. 541).

(*c*) Cæcal carcinoma sometimes is discovered unexpectedly at operation for acute appendicitis or for an appendix abscess that 'fails to resolve.' On rare occasions the appendix *is* inflamed, or even gangrenous, from obstruction to the mouth of its lumen by the carcinoma.

(*d*) Less commonly a papilliferous growth is the apex of an intussusception. A lump present at one time and not at another (owing to partial reduction), associated with attacks of acute abdominal pain, is characteristic of this complication.

FIG. 712.—An example of the inestimable value of sigmoidoscopy. The patient had been diagnosed and treated for some weeks for 'ulcerative colitis.' A barium enema was negative. Sigmoidoscopy showed a small bun-shaped carcinoma giving rise to an intussusception.

FIG. 713.—Barium enema showing a carcinoma of the descending colon.

METHODS OF INVESTIGATION

Sigmoidoscopy should be performed in cases where blood and mucus have been passed (fig. 712) and also in suspected cases when a barium enema is negative, because early growths in the lower part of the pelvic colon are not always visualised by radiography.

Radiography after a barium enema often shows a carcinoma of the colon

as a constant, irregular filling defect (fig. 713). On the other hand, it should be more widely known that radiography in comparatively early cases of carcinoma of the colon is not by any means conclusive evidence of the absence of a growth. In 75 cases of carcinoma of the colon examined radiologically, no abnormality was detected in 8 per cent. This entailed considerable delay in diagnosis in many of them (G. S. Ramsay).

More refined methods in radiological technique are however often rewarding, viz :

Contrast Enema.—In cases of a neoplasm involving only the mucous membrane, a contrast enema is very valuable. The barium emulsion is partly evacuated and air is injected into the colon. By this means the walls of the colon become delineated and a neoplasm that fails to alter the contour of the barium-filled colon may be demonstrated.

A tumour of the cæcum is more likely to be discovered by a barium meal than a barium enema. As a rule, in suspected cases of carcinoma of the colon, a barium meal is inadvisable because inspissated barium can precipitate intestinal obstruction if the lumen of the bowel is narrowed.

Exfoliate cytology.—In experienced hands colonic exfoliative cytology is a valuable adjunct in the diagnosis of obscure cases of carcinoma of the colon. Enemas are given until the return fluid is clear. The presence of malignant cells in smears of the washings is conclusive evidence of a malignant lesion of the colon. To obtain satisfactory results, the patient must be prepared carefully by a somewhat laborious procedure. After an interval of five to ten minutes, the returned fluid is collected and centrifuged. Films from the sediment are prepared and stained. Successful diagnoses have been made by this method in growths situated in all parts of the colon.

TREATMENT

Pre-operative Treatment.—When there is no intestinal obstruction, blood transfusion to correct anæmia, if present, enemata to cleanse the bowel, a high caloric and low residue diet, together with succinylsulphathiazole, 2 G. (30 grains) four-hourly for five days, are required. This sulphonamide has a slightly aperient action and it effectively reduces the number of virulent organisms ordinarily present in the colon. Oral antibiotics also can be given 6-hourly for 48 hours prior to operation. Neomycin, bacitracin, and polymyxin all are bactericidal and are virtually not absorbed from the alimentary tract. Neomycin frequently is employed for this purpose ; a combination of bacitracin and neomycin is particularly effective (F. L. Meleney). These pre-operative bactericides have rendered resection and anastomosis of the colon less dangerous than formerly. When intestinal obstruction is present, preliminary drainage of the intestine proximal to the obstruction must be performed (see p. 566).

Operation.—*The Test of Operability.*—The abdomen having been opened : (1) the liver is palpated for secondary deposits ; (2) the peritoneum, particularly the pelvic peritoneum, is inspected, if possible, and palpated for neoplastic implantations ; (3) the various groups of lymphatic nodes that drain the involved segment are palpated. Their enlargement does not necessarily mean that they are invaded by metastases, for the enlargement may be inflammatory, as has been pointed out on p. 524 in the discussion on colonic diverticulitis ; (4) the neoplasm is examined with a

Gordon Stuart Ramsay, Contemporary. Surgical First Assistant, St. George's Hospital, London.
Frank L. Meleney, Contemporary. Professor Emeritus of Clinical Surgery, Columbia University, New York.

view to ascertaining if it is fixed or free. So long as it can be mobilised it is operable. " The whole colon above the last 3 inches of the pelvic portion is either mobile or can be mobilised " (Sir Heneage Ogilvie).

Carcinoma of the cæcum or the ascending colon is treated, when operable, by right hemi-colectomy (fig. 714).

The abdomen is opened through a long right paramedian incision. The technique of right colectomy should include at the outset ligatures placed around the bowel, to prevent intraluminar spread. The peritoneum an inch (2·5 cm.) or more lateral to the ascending colon is incised and the incision is carried around the hepatic flexure. The right colon is elevated, with the leaf of peritoneum containing its vessels and lymph nodes, from the posterior abdominal wall, care being taken not to injure the ureter or the duodenum. The process of elevating the peritoneum is continued medially to near the origin of the ileo-colic artery, which is divided between ligatures, as also is the right colic artery when that vessel has a separate origin from the superior mesenteric. The mesentery of the last foot (30 cm.) of ileum, and the leaf of raised peritoneum attached to the cæcum, ascending colon and hepatic flexure, after ligation of the blood-vessels contained therein, is divided as far as the proximal third of the transverse colon (fig. 714). Many surgeons, having verified that the blood supply to the proposed intestinal stumps is excellent, forthwith clamp and divide the ileum and the transverse colon at the level of their respective severed mesenteries, and excise the freed intestine. The divided ends of the large and small intestines can be closed, and a lateral anastomosis between the ileum and the transverse colon performed. Alternatively, an end-to-end ileo-transverse colostomy may be preferred, in which case it is necessary to divide the intestine obliquely as depicted in all the illustrations relating to colonic resection (figs. 714-718). In this way the blood supply of the extremities of the divided ends is not jeopardised. The anastomosis having been completed, the defect in the posterior parietal peritoneum is repaired and the abdomen is closed.

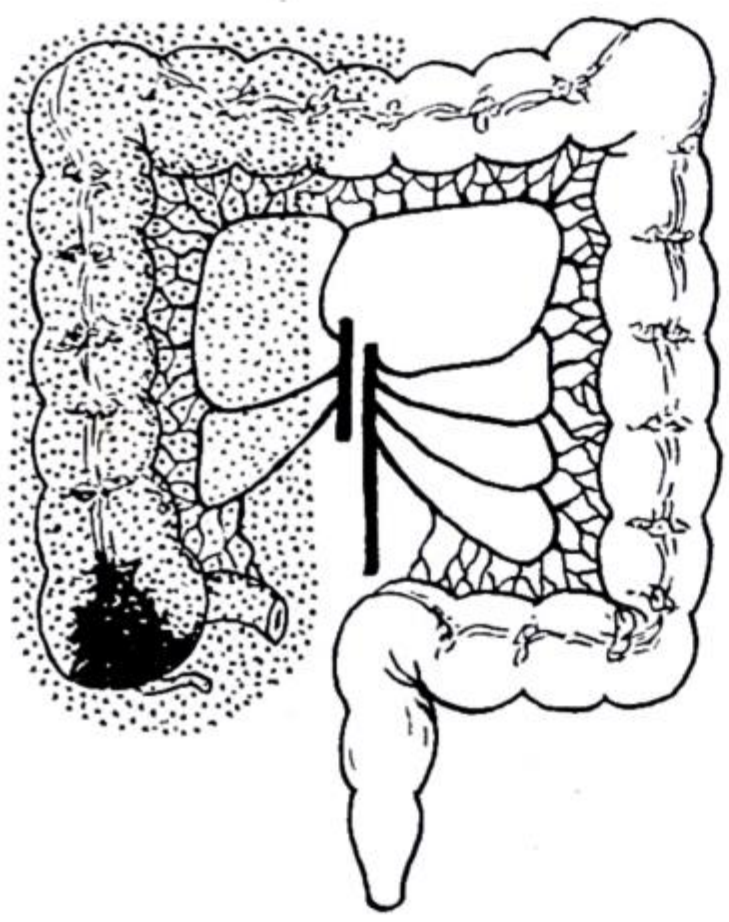


FIG. 714.—Area to be removed when the growth is situated in the cæcum.

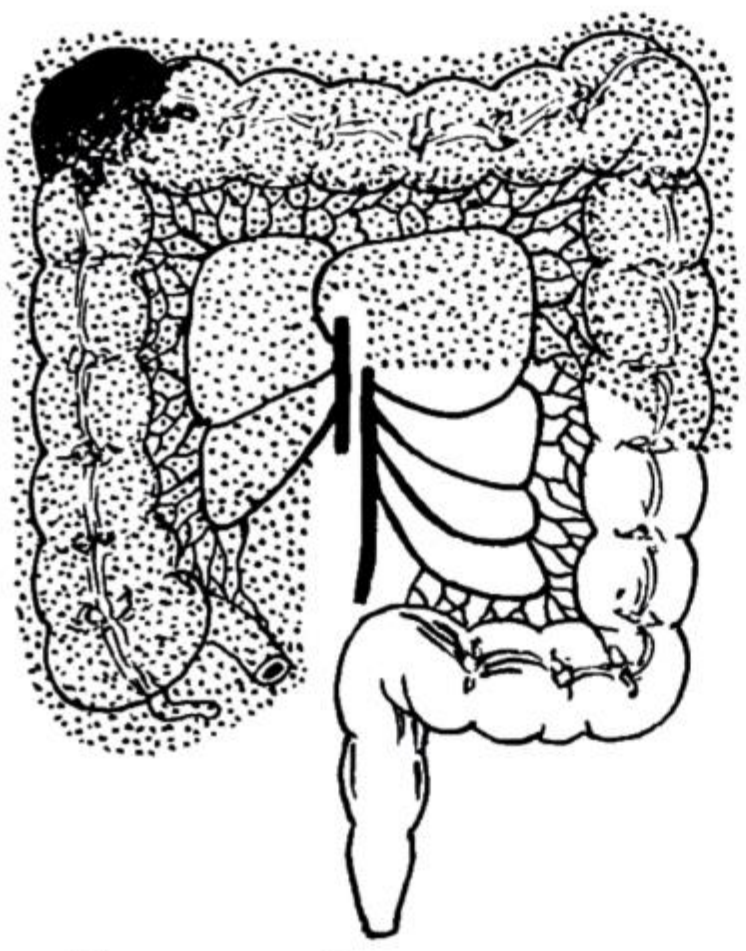


FIG. 715.—Area to be removed when the growth is situated at the hepatic flexure.

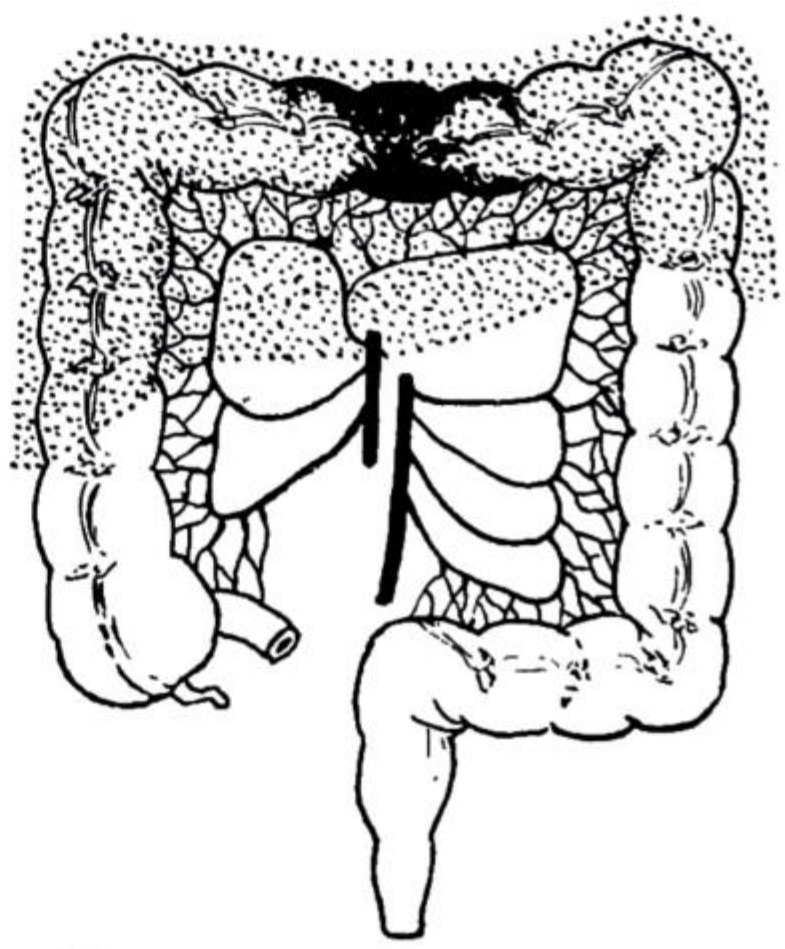


FIG. 716.—Area to be resected when the neoplasm is situated in the transverse colon.

Carcinoma of the Hepatic Flexure.—When the hepatic flexure is the seat of the neoplasm the resection must be extended correspondingly (fig. 715).

Sir Heneage Ogilvie, Contemporary. Consulting Surgeon, Guy's Hospital, London.

Carcinoma of the Transverse Colon.—When there is no obstruction, excision of the transverse colon (fig. 716), together with the transverse mesocolon and the greater omentum, followed by end-to-end anastomosis, is a satisfactory procedure.

If some degree of obstruction exists, after ligation and division of the blood supply to the mesocolon, exteriorisation by the Paul-Mikulicz operation, gives good results in this situation. Alternatively, preliminary cæcostomy can be performed.

Carcinoma of the Splenic Flexure or descending colon. The extent of the resection is shown in fig. 717.

The phrenico-colic ligament is divided, and after incising the parietal peritoneum from the splenic flexure to the pelvic colon, the splenic flexure, descending colon, and pelvic colon are raised from the posterior abdominal wall in the same way as the ascending colon is raised on the right side. When this has been achieved, the left branch of the middle colic and the left colic arteries are ligated near their origin, and the peritoneal leaf with its contained lymph nodes is divided. Excision with end-to-end anastomosis is then carried out.

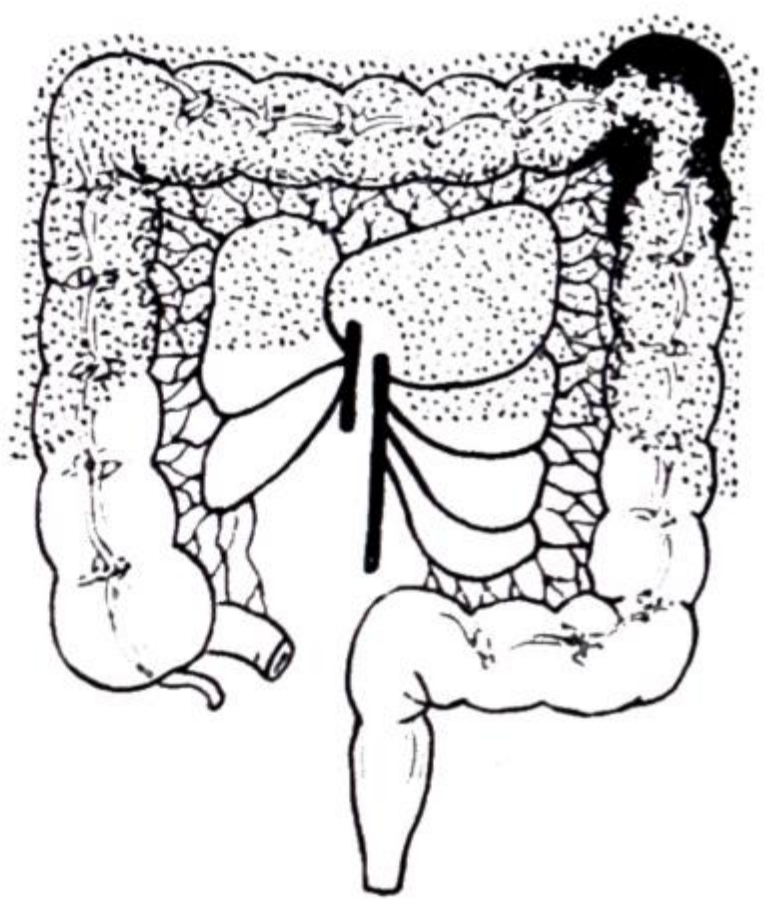


FIG. 717.—Area to be resected when the neoplasm is situated at the splenic flexure, or the proximal part of the descending colon.

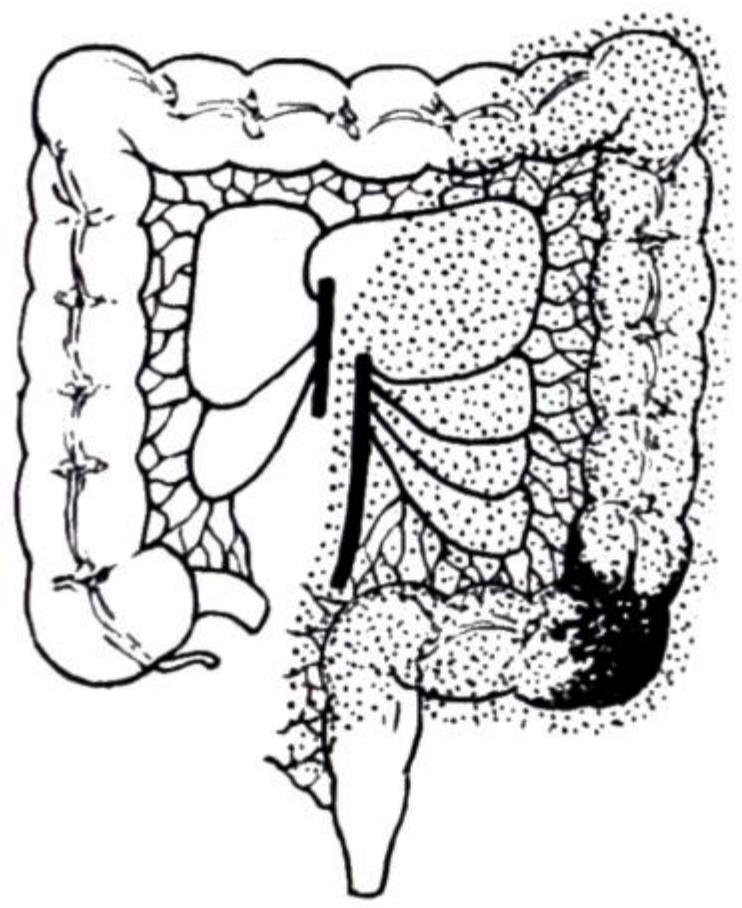


FIG. 718.—Area to be resected in the case of a carcinoma of the pelvic colon.

Carcinoma of the Pelvic Colon.—An oblique muscle-cutting incision gives a good approach. The left half of the colon is mobilised completely (fig. 718).

In order that the operation be rendered radical, the inferior mesenteric artery below its left colic branch, together with the related paracolic lymph nodes, must be included in the resection. This entails carrying the dissection as far as the upper third of the rectum, and accordingly the incision in the lateral leaf of peritoneum is extended downward to an appropriate level.

In every case of resection of the colon with anastomosis, drainage down to the site of anastomosis should be provided.

The Paul-Mikulicz Operation.—It is often contended that the Paul-Mikulicz operation is insufficiently radical, and in some quarters the operation is frowned upon. In the case of the pelvic colon this criticism is justifiable,

Frank T. Paul, 1851–1941. Surgeon, Royal Infirmary, Liverpool.
Johannes von Mikulicz-Radecki, 1850–1905. Professor of Surgery, Breslau.

but in other situations the method has much to recommend it in frail subjects and in those where there is great disparity in size between the intestine proximal and distal to the neoplasm. From the points of view of freedom from shock and from the dangers of leakage from the site of anastomosis, the operation is unique.

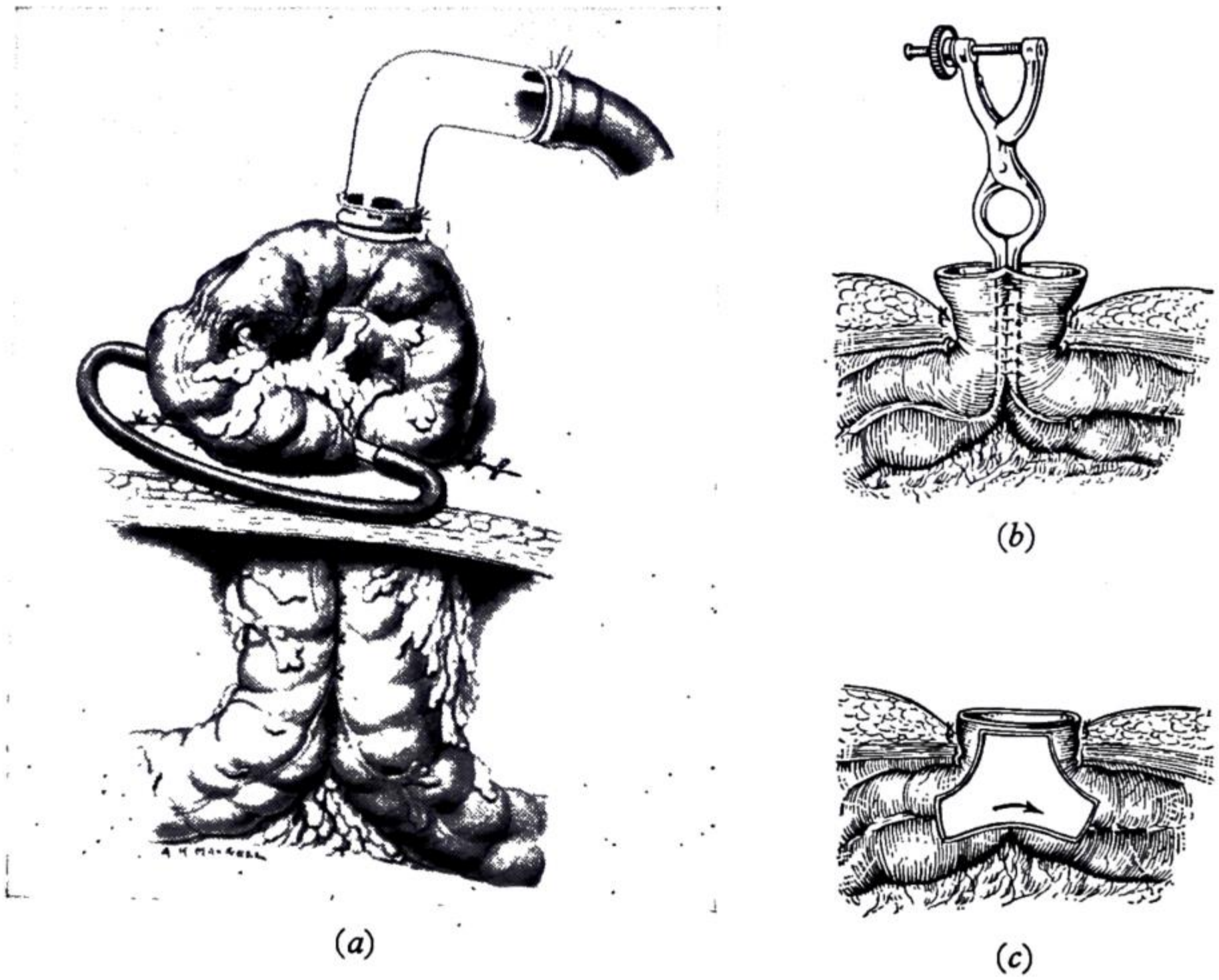


FIG. 719.—Stages in the Paul-Mikulicz operation.

The segment of colon bearing the neoplasm is mobilised fully in a manner appropriate to the site (see above). Using interrupted sutures, side-to-side approximation of the peritoneo-muscular coats of the base of the loop for a distance of 3 in. (7·5 cm.) having been effected (producing a double-barrelled gun effect), that portion of the freed segment lying above the approximation is brought out above skin level. The abdominal wall is closed around the ' barrels,' affixing them to the deeper layers of the wound. The loop bearing the tumour is excised and an enterotome is placed on the intervening spur (fig. 719 (*a*)). If intestinal obstruction is present, excision is deferred and a Paul's tube is tied into the proximal limb of the colon (fig. 719). In these circumstances the excision with a diathermy knife and the application of the enterotome (fig. 719 (*b*)) is postponed until the Paul's tube becomes loose and is removed—a matter of four or five days. An enterotome applied to the spur brings about restoration of the continuity of the colon (fig. 719 (*c*)) by pressure necrosis, and the enterotome falls out in five or six days. Finally, the colostomy is closed.

Post-operative Care following Colonic Resection.—Post-operative treatment includes the administration of antibiotics to guard against possible infection of the anastomotic area by *Cl. welchii*. Phthalylsulphathiazole is usually given by mouth after anastomotic operations, 1·5 G. (23 grains) four-hourly. Its action is slightly constipating, and in this respect it is superior to succinyl-sulphathiazole, the purgative action of which, although slight, might endanger the suture line.

When the Growth is found to be Inoperable.—If the growth is in the upper part of the left colon, transverse colostomy is performed. If it is in the pelvic colon, left iliac colostomy is preferable. When there is an inoperable growth in the ascending colon, ileo-colostomy is the best procedure. Provided the local condition permits mobilisation, metastases in the liver should not be a deterrent to a resection of that portion of the colon containing a neoplasm, perhaps in this instance by the Paul-Mikulicz method.

FÆCAL FISTULÆ

An external fistula communicating with the cæcum sometimes follows an operation for gangrenous appendicitis (fig. 720) or the opening of an appendix abscess. A fæcal fistula can occur from necrosis of a gangrenous patch of intestine after the relief of a strangulated hernia, or from a leak after an intestinal anastomosis. The opening of an abscess connected with chronic diverticulitis or carcinoma of the colon frequently results in a fæcal fistula. Tuberculous peritonitis, ileo-cæcal actinomycosis, and regional ileitis (in this instance nearly always following operation) are also causes of fæcal fistulæ, which may be multiple.

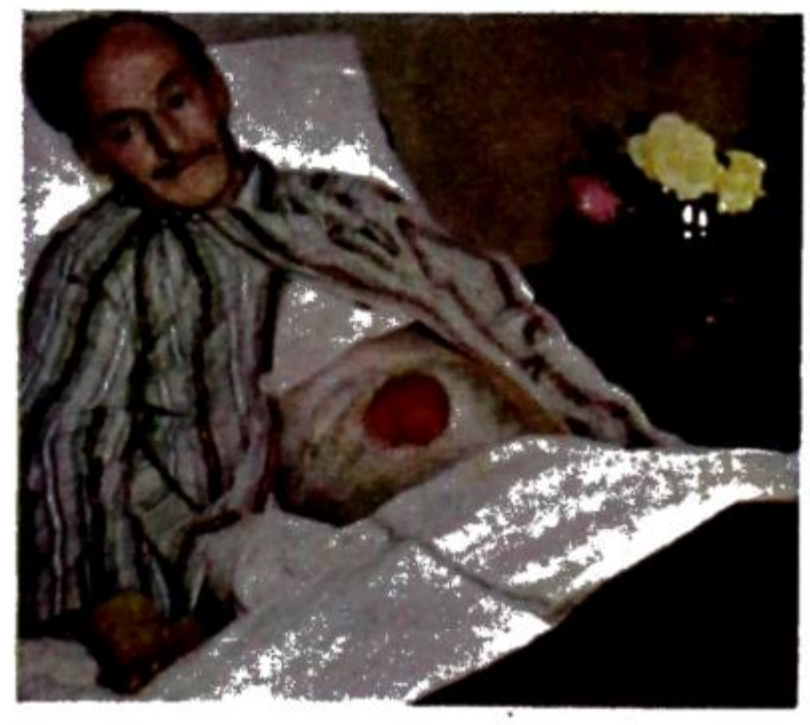


FIG. 720.—Fæcal fistula with prolapse of mucous membrane following acute appendicitis with gangrene of the cæcum. Aluminium paint is protecting the skin from excoriation.

External fæcal fistulæ can be divided into three varieties :

1. A track lined by mucous membrane which protrudes above skin level.

2. A direct track lined by granulation tissue communicating with the exterior.

3. A long, tortuous track lined by fibrous tissue and partly epithelialised.

A fistula connected with the duodenum or jejunum is bile-stained and contains undigested food. When the ileum or cæcum are concerned, the discharge is fluid fæcal matter ; when the distal colon is involved, it is solid or semi-solid fæcal matter. In some cases, when the leak from the small intestine or cæcum is small, it may be difficult to distinguish a fæcal discharge from fæculent pus. If charcoal is administered by mouth and a fæcal leak is present, the black granules will be distinguished easily in the discharge a few hours later. Often the site of the leak and the length of the fistula can be determined by radiography after a barium meal or barium enema. Should this fail to demonstrate the internal orifice, injection of lipiodol into the external opening will usually give the desired information.

Treatment.—Fæcal fistulæ, especially those in connection with the small intestine, tend to heal spontaneously, provided there is no obstruction beyond the fistulous opening. The abdominal wall must be protected from erosion by escaping intestinal juices as has been described in the section dealing with ileostomy (see p. 533).

The higher the fistula in the intestinal canal the more skin excoriation must be expected. This reaches its zenith in the case of a duodenal fistula (see p. 283). Some form of suction apparatus to remove the enzyme-laden discharge is a fundamental procedure.

Ferrum redactum, grains 5 (0·3 G.) in capsules taken orally three times a day renders the intestinal contents less irritating to the skin. Phthalylsulphathiazole, by reducing the bacterial content of the discharge, assists in controlling the concomitant wound infection and favours healing. Pulv. cretæ aromat. ʒi (4 G.) q.i.d., by thickening discharge, is extremely helpful in aiding spontaneous closure. Alternatively isogel can be tried. Fistulæ high in the alimentary tract can result in dehydration and hypoproteinæmia. Intravenous dextrose-saline, together with plasma infusions, may be required. In the case of a fistula without protrusion of mucous membrane which fails to heal in three weeks, closure may be expedited by inserting an obturator in the following way :

A disc of rubber, cut from the inner tube of a motor-car, of suitable size, e.g. about 1 inch (2·5 cm.) in diameter, is transfixed by a mattress suture. The disc is rolled up and the resulting scroll is grasped with dressing forceps and inserted through the fistula into the intestine. When the forceps are removed, the scroll opens in the lumen of the intestine. Traction on the suture approximates the disc to the wall of the intestine and occludes the internal opening of the fistula. The ends of the suture can be tied around a roll of gauze to keep it in place. When the healing of the fistula is almost complete, one end of the suture is cut so that the remainder can be withdrawn, and the rubber disc passes along the bowel and is evacuated.

A fistula with mucosa visibly continuous with the skin edge will never close spontaneously. In some of these cases, where the opening is a large one, the intestine tends to prolapse upon the surface (fig. 720).

The operative treatment for closure of a fæcal fistula consists in making an incision as shown in fig. 721, and dissecting up the tract through the abdominal wall and the peritoneum. The base of the fistula, now free, is crushed, ligated, and oversewn. The abdominal wall is then closed in layers. In the case of a colo-cutaneous fistula connected with colonic diverticulitis, if the fistula fails to heal after a few weeks, resection of the affected segment is usually advisable (see p. 526). Should the mass from which the fistula arises prove to be an inoperable carcinoma, a defunctioning colostomy should be performed at a higher level. When there is no obvious cause for the fistula, the discharge must be examined on several occasions for the *actinomyces* or *mycobacterium tuberculosis*. The demonstration in a plain X-ray of many calcified tuberculous mesenteric lymph nodes favours tuberculosis as the cause. In complicated fistulæ, an operation to close the leak often proves difficult, but in the case of the cæcum it is usually straightforward (fig. 721).

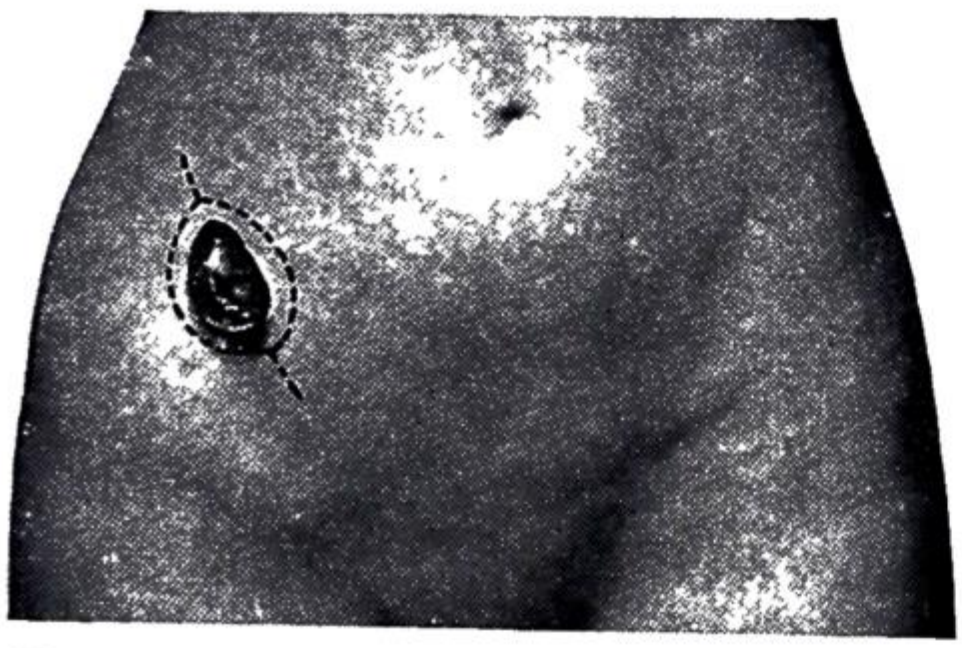


FIG. 721.—Incision for closure of a fæcal fistula of the cæcum.

CHAPTER XXVI

INTESTINAL OBSTRUCTION

HAMILTON BAILEY

THE causes of intestinal obstruction, which may be acute, chronic, or acute-on-chronic, are very numerous. In most instances the obstructing agent falls into one of four categories :

1. **Obturation.**—The lumen of the intestine becomes blocked by a foreign body, a gall-stone, a bolus of incompletely digested material, a fæcolith, or inspissation of fæces or meconium.

2. **Intramural.**—Typically, by cicatrisation of its walls the lumen is rendered so narrow that the intestinal contents cannot pass : this occurs in inflammatory and malignant strictures. Although not due to cicatrisation, narrowing or complete interruption of the intestinal lumen due to intussusception or volvulus is included in this category.

3. **Extramural.**—The intestine is compressed from without. Herniæ, congenital bands, inflammatory bands, and adhesions are the conditions that cause this type of obstruction.

4. **Loss of Propulsive Power.**—One segment fails to transmit peristaltic waves. Paralytic ileus and mesenteric vascular occlusion both come under this category.

The most common causes of intestinal obstruction are external hernia, post-operative adhesions, and primary and recurrent carcinoma of the colon, in that order.

PATHOLOGY

There are four varieties of intestinal obstruction :

1. Non-strangulating obstruction, in which the lumen of the intestine is occluded.

2. Strangulating obstruction, where the blood supply to a segment of intestine is seriously impaired, or cut off completely.

3. Closed-loop obstruction, in which a segment of intestine is shut off both from above and below.

4. Neurogenic obstruction, that arises from paralysis of the nervous mechanism controlling peristalsis.

Frequently two or more of these varieties are present concurrently.

1. **Non-strangulating Obstruction.**—At the outset, the intestine *above* the point of obstruction endeavours to overcome the obstruction by vigorous peristalsis. Increased peristalsis continues for a period of from forty-eight hours to several days ; the more distal the point of obstruction, the longer does it remain vigorous. If the obstruction is not relieved, a time is reached

when increasing distension causes peristalsis to become less and less ; finally, peristalsis ceases, and the obstructed intestine becomes flaccid and paralysed.

For two or three hours following the obstruction, the intestine *below* the point of obstruction exhibits normal peristalsis, and absorption from it continues until the residue of its contents has been passed onwards. Then the empty intestine becomes immotile, contracted, and pale, and so it remains until the obstruction has been overcome, or death ensues.

Distension.—The intestine above the obstruction commences to distend almost as soon as the obstruction occurs, but before the distension can be recognised clinically it must be considerable. Intestinal distension is partly gaseous and partly fluid :

Gas.—At first the distension is mainly gaseous. In non-strangulating obstruction 68 per cent. of the gas is due to swallowed air, 22 per cent. to diffusion into the bowel lumen of gases from the blood-stream, and 10 per cent. to bacterial decomposition of the intestinal contents (J. S. Hibbard). In closed-loop obstructions the gas must be derived entirely from the latter two sources. Whatever its original composition, the oxygen is quickly absorbed into the blood-stream and the gas becomes composed of about 90 per cent. nitrogen, the remainder being carbon dioxide and hydrogen sulphide.

Fluid is composed mainly of digestive juices. Each twenty-four hours there are secreted no less than 7,500 ml. (13 pints) of these juices :

Saliva	1,500 ml. (3 pints)
Gastric juice	2,500 ml. (4 pints)
Bile and pancreatic juice . .	500 ml. (1 pint)
Succus entericus . . .	3,000 ml. (5 pints)

In obstruction of the ileum, these secretions are at first absorbed, but as the distension progresses the veins at the mesenteric border of the intestine become increasingly compressed, viz. ——→ consequently absorption of fluid by them becomes correspondingly diminished. Thus it will be appreciated that the patient with intestinal obstruction is subjected to a dual deprivation of water and salts (*a*) by reason of vomiting and (*b*) by defective intestinal absorption.

Loss of Fluid and Electrolytes.—The seriousness and rapidity of the depletion are dependent upon, and proportional to, the level of the obstruction. Obstruction high in the small intestine, by causing early and profuse vomiting and preventing absorption from the ileum, produces dehydration in a matter of twelve hours. The urine becomes scanty and chlorides disappear from it, and the plasma chlorides, normally 550 to 620 mg. per cent. as NaCl (95 to 105 mEq per litre), become progressively reduced. In the case of the lower ileum—a frequent site of intestinal obstruction—the loss is much slower, because absorption by the ileal mucous membrane above the obstruction continues for upwards of forty-eight hours. Nevertheless, as time goes on, for the reason described and illustrated above, the absorptive power of the ileum decreases. In colonic obstruction the amount of fluid and electrolytic loss is small.

The Rôle of Intestinal Toxins.—Prevailing opinion is that absorption of toxins is not a lethal factor in non-strangulating obstruction. In late cases, owing to breakdown of the protein content of the intestinal juices and increased bacterial activity, toxins *are* present in the obstructed intestine, but, by

James Sutherland Hibbard, Contemporary. Surgeon, Wichita, Kansas, U.S.A.

reason of the diminished absorption consequent upon the distension (see p. 556), the amount of toxins carried by the portal circulation to the liver is not greater than a healthy liver can detoxicate. On the other hand, sudden release of obstruction allows highly toxic substances to pass into the non-obstructed intestine below, where they are absorbed greedily and, perchance, in overwhelming amounts. Pre-operative gastro-intestinal suction helps to moderate this flooding.

2. **Strangulating obstruction** resulting from entrapment of bowel in a hernia, beneath a band, or consequent upon a volvulus or advanced intussusception occurs *pari passu* with non-strangulating obstruction, but the gravity of the non-strangulating moeity pales before that of the strangulating. The dual lesion is the result of compression of the bowel *and its mesentery*. Mesenteric vascular occlusion alone gives rise to gangrene without mechanical obstruction.

The first effect of strangulation is so to compress the veins as to cause the strangulated bowel, and its involved mesentery, to become blue and congested.

Loss of blood volume into the congested segment is proportional to the length of that segment. When, as is often the case in strangulated external hernia, only a few inches is involved, the amount of blood thus imprisoned is inconsequential; on the other hand, when a whole coil of intestine becomes strangulated the loss of blood is sufficient to render the patient oligæmic, while when several metres of small intestine is involved the volume of circulating blood is so reduced as to imperil the patient's life.

Distension.—For a considerable time the strangulated segment (fig. 722 (B)) alone distends, the greatest distension occurring when the venous return is completely obstructed while the arterial supply remains unimpaired. Unlike non-strangulating obstruction, early distension of the proximal intestine (fig. 722 (A)) is absent; indeed, for a time varying from a few minutes to several hours the proximal intestine contracts. After this varying interval vigorous peristalsis occurs in the proximal segment, but is still unaccompanied by distension. By the time gangrene of the strangulated segment is imminent, retrograde thrombosis is proceeding along the related tributaries of the mesenteric vein. Distension then appears both on the proximal and distal sides of the strangulation (fig. 722 (A) and (C)) (J. T. Chesterman).

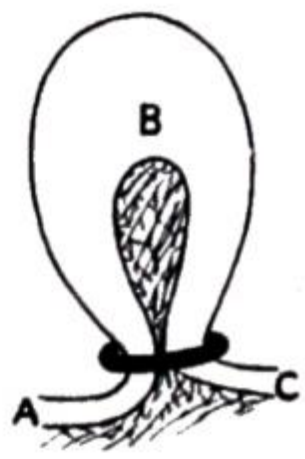


FIG. 722

The Onset of Gangrene.—Much depends on the tightness of the constricting agent. When the venous return is completely occluded, the colour of the intestine turns from purple to black. About this time, in many instances, owing to increased œdema at the point of obstruction, the arterial supply is jeopardised. Then the peritoneal coat loses its glistening appearance, the mucous membrane becomes ulcerated, and finally moist gangrene sets in.

Transmigration of Bacteria and Toxins.—When the wall of the intestine becomes partly devitalised, both bacterial toxins and the products of tissue autolysis pass into the peritoneal cavity, there to be absorbed into the circula-

Judson T. Chesterman, Contemporary. Surgeon, City General Hospital, Sheffield.

tion. This is followed by the migration of bacteria, and peritonitis sets in. So it comes about that strangulation in an *external* hernia is far less dangerous than intraperitoneal strangulation, for in the former the transudate containing lethal toxins and bacteria is confined to the small absorptive area of the hernial sac.

FIG. 723.—Carcinomatous stricture of the hepatic flexure : closed-loop obstruction.

3. **Closed-loop obstruction** is present in the majority of cases of intestinal strangulation. In its pure form it is seen in carcinomatous stricture of the colon (fig. 723). Distally the colon is occluded by the neoplasm, while in one-third of cases the ileo-cæcal valve prevents regurgitation of the contents of the large intestine into the ileum, and consequently that part of the colon proximal to the neoplasm is closed at both ends (fig. 723). As a result of anti-peristalsis the pressure within the cæcum becomes so high as to compress the blood-vessels within its walls. Stercoral ulceration, gangrene, and perforation of the cæcum sometimes occurs from this cause. In cases where the ileo-caecal valve permits regurgitation readily, the obstruction is of the non-strangulating variety.

4. **Neurogenic obstruction** (paralytic ileus) is discussed under Special Forms of Intestinal Obstruction (p. 582).

CLINICAL FEATURES OF ACUTE INTESTINAL OBSTRUCTION

The symptoms and signs vary with the nature and site of the obstruction, so that often it is possible to differentiate obstruction of the small intestine from obstruction of the large intestine.

When the Obstruction occurs in the Small Intestine:

Abdominal pain is the first symptom : it commences suddenly, and often without warning. It becomes increasingly severe, then passes off gradually, only to return at intervals of a few minutes to a quarter of an hour. These attacks of intestinal colic, which last from three to five minutes, spread all over the abdomen, but are localised mainly at the umbilicus. In between attacks the patient is often quite free from pain. Recurring attacks of severe abdominal pain are a leading feature of all varieties of acute intestinal obstruction, with the sole exception of paralytic ileus, in which condition pain is singularly absent, unless the meteorism is extreme, when abdominal discomfort may be experienced.

Vomiting.—When the jejunum is mechanically obstructed, vomiting occurs with the first and each succeeding attack of pain. In the much more common obstruction of the ileum the patient may vomit once, following which there is an interval of several hours during which time the attacks of pain occur without vomiting. Ultimately copious, forcible, oft-repeated vomiting sets in. As acute intestinal obstruction progresses, the character of the vomitus alters. Initially it contains partly digested food ; next it consists entirely of mucoid fluid ; thereafter the vomitus becomes yellow or green from regurgitation of bile ; finally, it is fæculent.

That the vomitus commences to assume a fæculent character only after three and a half days of complete intestinal obstruction should be remembered by the reader when seeking this sign.

Pulse-rate and temperature are normal in early cases, but in advanced intestinal obstruction the temperature becomes subnormal and the pulse-rate increases steadily.

Dehydration.—Repeated vomiting and also loss of absorptive power by the distended intestine leads to dehydration, and when the patient is first examined signs of dehydration—a dry skin, a dry tongue, and sunken eyes—may be present. The output of urine is small ; it is concentrated, and contains little or no chlorides.

Distension.—In early cases of obstruction of the small intestine abdominal distension is often slight, or absent. Centrally placed distension is present in fully established cases of obstruction to the ileum.

In view of the fact that external hernia is the commonest cause of intestinal obstruction, *an examination of the common hernial sites* must be undertaken early in the course of the clinical examination. In the absence of an irreducible hernia the abdomen is again inspected.

Visible peristalsis may be present (fig. 724). In order to observe it the abdomen must be watched for several minutes. If waves of peristalsis are seen and their appearance synchronises with an attack of intestinal colic, the information gained clinches a diagnosis of intestinal obstruction. In very thin subjects normal peristalsis is sometimes visible, but in this instance the undulations are gentle and more constant ; they do not come in waves and then depart, and their appearance does not coincide with an attack of pain. When the hand is laid lightly upon the abdomen of a patient with obstruction of the ileum, an obstructed coil may be felt to harden and soften intermittently.

FIG. 724.—Visible peristalsis. Intestinal obstruction due to the strangulated right femoral hernia, which can be seen.

Borborygmi are sometimes loud enough to be heard by the unaided ear. More often auscultation is necessary. The sound of turbulent peristalsis coinciding with an attack of colic is valuable evidence of intestinal obstruction.

Bowels.—In complete intestinal obstruction, after the contents of the bowel below the obstruction has been evacuated, there is constipation, and usually neither fæces nor flatus are passed. It should be noted that there may be a natural action of the bowels after the onset of the attack, and that it is commonplace for an enema to yield a small fæcal result. Of more diagnostic assistance is a second enema given half an hour later. If the obstruction is

Large intestine (the cæcum excepted) shows haustral folds. Haustral folds, unlike the valvulæ conniventes, are spaced irregularly and the indentations are not placed opposite one another (fig. 727 (*c*)).

(*a*) Jejunum (*b*) Ileum (*c*) Colon

FIG. 727.—Gas shadows characteristic of various parts of the intestine.

Fluid Levels.—In infants under the age of two years a few fluid levels in the small intestine are a normal occurrence. In adults, two inconstant fluid levels must be regarded as physiological. One is at the duodenal cap ; the other, which is more infrequent, is within the terminal ileum. In intestinal obstruction it takes a little time for the gas to separate from the fluid ; consequently fluid levels appear later than gas shadows. When paralysis of the intestine has occurred, fluid levels become more conspicuous and more numerous. By the time fluid levels are pronounced, obstruction is advanced. The number of fluid levels is proportionate to the degree of obstruction and to its site in the small intestine ; the nearer the obstruction is to the ileo-cæcal valve, the larger the number of fluid levels (fig. 728). Obstruction *low* in the colon does not commonly give rise to fluid levels in the small intestine, but in the case of obstruction *high* in the large intestine, this phenomenon is not unusual, because in many individuals the ileo-cæcal valve is incompetent. The commonest cause of false fluid levels is an incompletely evacuated enema.

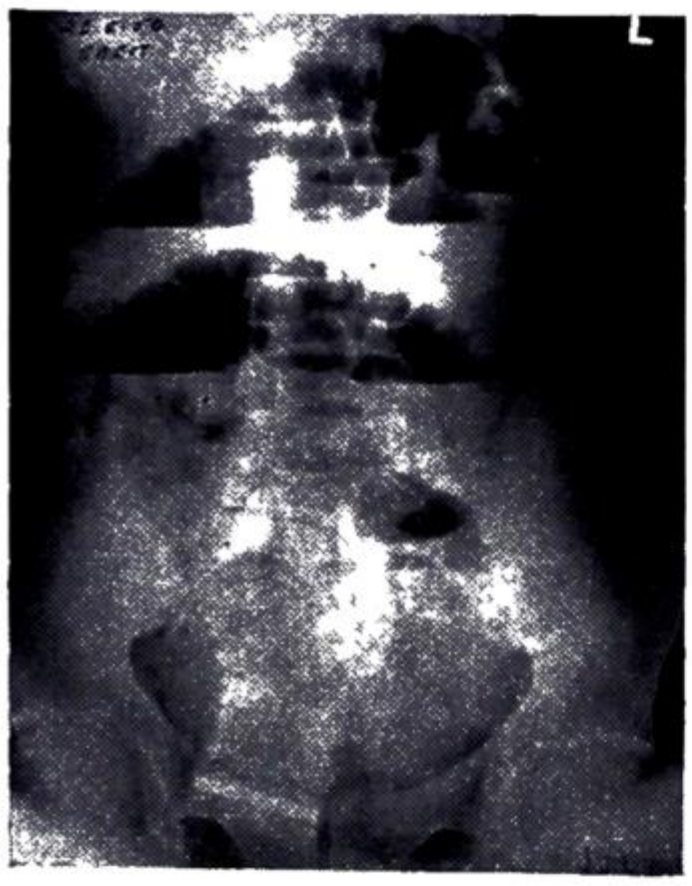


FIG. 728.—Fluid levels ; subacute intestinal obstruction by bands.

In obstruction of the large intestine, a plain radiograph always shows a large amount of gas in the cæcum. In most cases a barium enema gives concrete evidence of the presence and site of colonic obstruction.

TREATMENT OF ACUTE INTESTINAL OBSTRUCTION

There are three measures for combating and overcoming the effects of intestinal obstruction. They are : (1) gastro-duodenal or, when possible, gastro-intestinal suction drainage ; (2) replacement and maintenance of fluid and electrolytic balance ; (3) relief of the obstruction by operation.

The first two are always necessary preliminaries to the relief of obstruction by operation, and they are the mainstays of post-operative treatment. In some cases, as will be shown, they are used exclusively.

In every case of acute intestinal obstruction the first step is to empty the stomach by a transnasal aspirating tube and to keep the stomach empty by withdrawing the contents with a syringe or by continuous suction. The second step is to administer normal saline solution intravenously in proportion to the patient's needs. If signs of dehydration are present, the rate of flow should be 100 drops a minute until chlorides appear in the urine. The rate is then reduced to 60 drops a minute and dextrose-saline solution substituted for the normal saline. The amount of saline solution and dextrose-saline solution given intravenously, and the amount of fluid aspirated, is recorded, as also is the amount of urine passed. This enables an accurate fluid intake and output chart to be compiled. When, on clinical examination, the cause of the obstruction is not obvious, radiographs of the abdomen are taken and the films examined. The clinical and radiographic data are correlated. The main indications for early operation, i.e. as soon as the fluid and electrolytic depletion has been corrected, are :

1. Obstructed or strangulated external hernia (Chapter xxx).
2. Internal intestinal strangulation.
3. Acute or acute-on-chronic obstruction of the large intestine.

The most urgent of these is intestinal strangulation. Gastro-duodenal aspiration should be continued throughout the operation, and also in most instances the intravenous infusion, which, in cases of strangulation, should be supplemented by blood transfusion.

Relief of Obstruction by Operation.—When the cause of the obstruction lies within the abdomen but its site is doubtful, a right lower paramedian incision is employed.

When the Obstruction lies in the Small Intestine.—The hand is passed to the cæcum. In obstruction of the small intestine the cæcum is collapsed. The site of obstruction may be obscured by dilated coils of intestine, in which event an unobstructed contracted coil of ileum is sought (fig. 729) and followed upwards. This will guide the fingers to the site of obstruction which, if deeply placed, is exposed by displacing distended coils away from the site with warm, moist abdominal packs. Occasionally it is necessary to withdraw several coils of distended intestine before the site of obstruction can be displayed satisfactorily. Eviscerated coils must be kept covered by abdominal packs or, better, by placing them in a special rubber or polythene bag made with a double draw-string (F. Lahey). The obstruction is relieved or short-circuited by one of the various methods described under special forms of intestinal obstruction.

FIG. 729.—Acute intestinal obstruction : tracing a collapsed coil to the site of obstruction.

Frank Lahey, 1880–1953. Founder and Director of the Lahey Clinic, Boston, Mass.

Measures to be taken when the Intestine is Strangulated.—If, as is frequently the case in intra-abdominal strangulation, blood-stained fluid is present in the peritoneal cavity, the fluid should be removed by suction or mopped up as completely as possible, for it is toxic and liable to be infected. After the relief of strangulation a decision must be reached as to whether the segment that was strangulated is viable. When it is black and the peritoneal coat has lost its sheen, when the mesentery shows a lack of arterial pulsation, or thrombosis of its veins, it is non-viable, if not already gangrenous, and resection followed by anastomosis must be carried out. In doubtful cases when the intestine is blue, purple, or dark red, the effect of wrapping it in a warm moist abdominal pack is tried. At the same time the anæsthetist administers pure oxygen for three minutes. By these means viable is differentiated from non-viable intestine thus :

Intestine	*Viable*	*Non-viable*
Circulation	Dark colour becomes lighter; mesentery bleeds, if pricked.	Dark or black colour does not lighten; no bleeding if mesentery is pricked.
Peritoneum	Shiny.	Dull and lustreless.
Intestinal musculature	Firm. Pressure rings may or may not disappear. Peristalsis may be observed.	Flabby, thin, and friable. Pressure rings persist. No peristalsis.
Test	10 ml. of 1 per cent. procaine injected into the mesentery with a very fine needle improves the appearance.	Injection of procaine—no effect.

Special attention should always be paid to the sites of previous constriction (' pressure rings ') at each end of the segment. The proximal site of constriction, that has borne the brunt of the obstruction, sometimes alone is of doubtful viability, in which case it should be enfolded by passing sutures through the sero-muscular coats and covering the area with a patch of greater omentum.

Pressure rings having received attention, viable intestine is returned to the abdominal cavity and the laparotomy incision is closed. When the strangulated intestine is deemed non-viable, it must be resected and the continuity of the alimentary canal restored by anastomosis. When the patient's general condition is too poor to withstand resection, doubtfully viable intestine should be returned to the peritoneal cavity, but obviously gangrenous intestine must be exteriorised with about 5 inches (12·5 cm.) of healthy intestine at either end. After closing the abdominal wall around the exteriorised intestine, the

gangrenous portion must be excised forthwith. If it is not excised, thrombophlebitis spreads from the veins of the extraperitoneal mesentery into the intraperitoneal mesentery, and so previously viable intraperitoneal intestine becomes gangrenous. Should the gangrene not extend far into the mesentery, after resecting the gangrenous portion, extra-abdominal anastomosis can be performed as shown in fig. 730. This spares the patient the misery and dangers of a total fæcal fistula and, if all goes well, in four or five days the enterostomy can be closed and the anastomosis returned to the abdominal cavity.

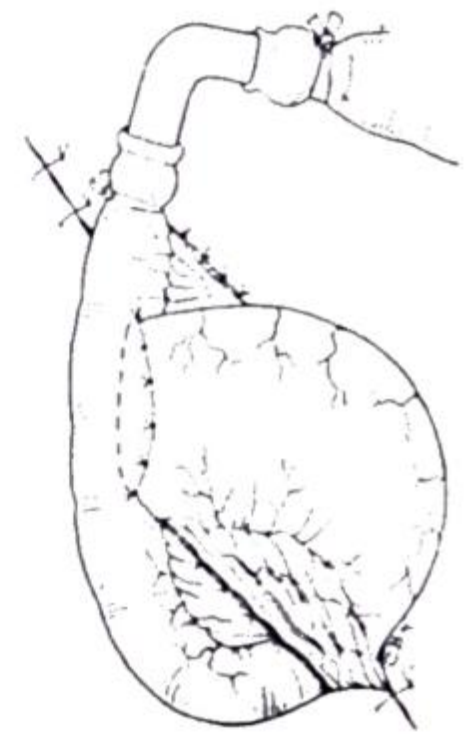


FIG. 730.—Extra-abdominal resection with anastomosis of the proximal end into the side of the distal end with enterostomy.

When the obstruction occurs in the large intestine it is usually due to a carcinoma, or occasionally, in the case of the pelvic colon, to its imitator, diverticulitis, and the obstruction is of the acute-on-chronic variety. Acute-on-chronic obstruction of the large intestine should always be treated by early operation. If the patient's condition is good, laparotomy is performed through a right or left paramedian incision, according to the site of the obstruction. If the site is unknown, a right lower paramedian incision is employed. Distension of the cæcum at once confirms that the obstruction lies in the large intestine. Palpation of the pelvic colon and, if that be collapsed, the transverse colon, will readily lead to the obstruction. When removable obstruction is present in the ascending colon, at the hepatic flexure, or in the transverse colon, *cæcostomy* through a separate grid-iron incision is the best means of relieving the obstruction. For irremovable obstruction in the right colon, *ileo-transverse colostomy* is indicated. If the obstruction lies at the splenic flexure, in the descending colon, the pelvic colon, or in the rectum, *transverse colostomy* is performed (fig. 731).

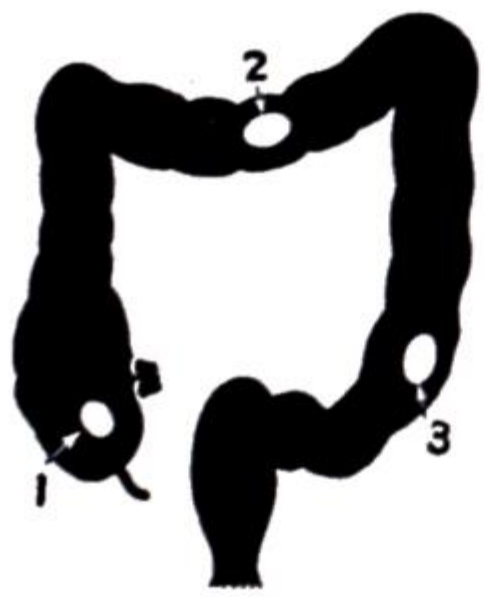


FIG. 731.—The sites for an artificial anus in acute-on-chronic intestinal obstruction. 1. Cæcostomy. 2. Transverse colostomy. 3. Left iliac colostomy.

In very old or enfeebled patients when a carcinoma of the rectum is fixed and probably irremovable, left iliac colostomy is the best site for a permanent artificial anus.

When the distension is great and the condition of the patient is poor, blind cæcostomy is sometimes the best procedure.

Blind Cæcostomy.—Under local anæsthesia a grid-iron incision is made in the right iliac fossa. If possible, the cæcum is brought to the surface. In a proportion of cases it is necessary to enlarge the wound a little, and incise the peritoneum to the outer side of the cæcum, and with the index finger free the cæcum from its loose peritoneal attachments before the organ can be withdrawn satisfactorily. A length of $\frac{1}{2}$-inch (1·3-cm.) rubber tubing is passed under the upper part of the cæcum through an incision made in an avascular portion of the true or artificially made mesocæcum. In order to prevent the tube slipping out, each end is doubled over, and maintained in that position by a strong ligature. The peritoneum is closed above and below the protruding cæcum, but no attempt is made to suture the cæcum to the peritoneum. Dry

abdominal packs are placed around the protruding viscus so as to isolate it. Three or four aspirating needles are passed through the cæcal wall, and allowed so to remain. Through their lumen much gas and a little fæcal matter escape. The cæcum and its contained needles is covered with gauze, and is re-examined six-hourly, when the directions of the needles can be readjusted, if necessary, and should one or more appear blocked, it can be cleared by injecting a little water through it. During the whole of this period the patient's fluid and electrolytic balance is restored, and antibiotic therapy is given. After forty-eight hours the hollow needles are removed, a purse-string suture is inserted, a small stab incision is made, a rubber tube is passed into the cæcum and fastened to the cæcal wall by a stitch, and the purse-string suture tied to close the cæcum about the tube. The tube is then connected to a bedside bottle by a glass connection, and a further length of tubing. Performed in this way, cæcostomy is effective and carries a low mortality.

When the obstruction has been proved radiologically to be on the left side of the colon, or if it is palpable per rectum, blind transverse colostomy is to be preferred.

Blind Transverse Colostomy.—The abdomen is opened through a short transverse incision over the right rectus muscle 2 inches (5 cm.) above the umbilicus. A portion of the transverse colon is delivered. After detaching the greater omentum from the loop, a glass rod is passed through the transverse mesocolon and the peritoneum is closed on either side, incorporating an appendix epiploica in each stitch. Tulle gras is applied to the skin about the exteriorised loop, which is emptied by a hollow needle attached to a suction apparatus. The apex of the loop is then opened and a Paul's tube tied into it.

GASTRO-INTESTINAL SUCTION DRAINAGE[1]

When intestinal strangulation can be ruled out and the obstruction lies in the small intestine, a period of several hours' gastro-intestinal suction drainage is the best form of preliminary treatment. Suction drainage should also be employed almost exclusively in paralytic ileus. In mechanical obstruction the combined effects of relieving distension by suction and the administration of fluid intravenously greatly improve the general condition of the patient for operation. Locally, diminution in size of the distended coils of intestine facilitates the operation and closure of the incision. Another most important consideration is that if highly toxic intestinal contents are aspirated before operation, it spares the patient the danger of absorbing this material after the obstruction has been relieved.

The balloon of a gastro-intestinal tube (fig. 732) is propelled by peristalsis, thus enabling coil after coil of distended intestine to be decompressed, the

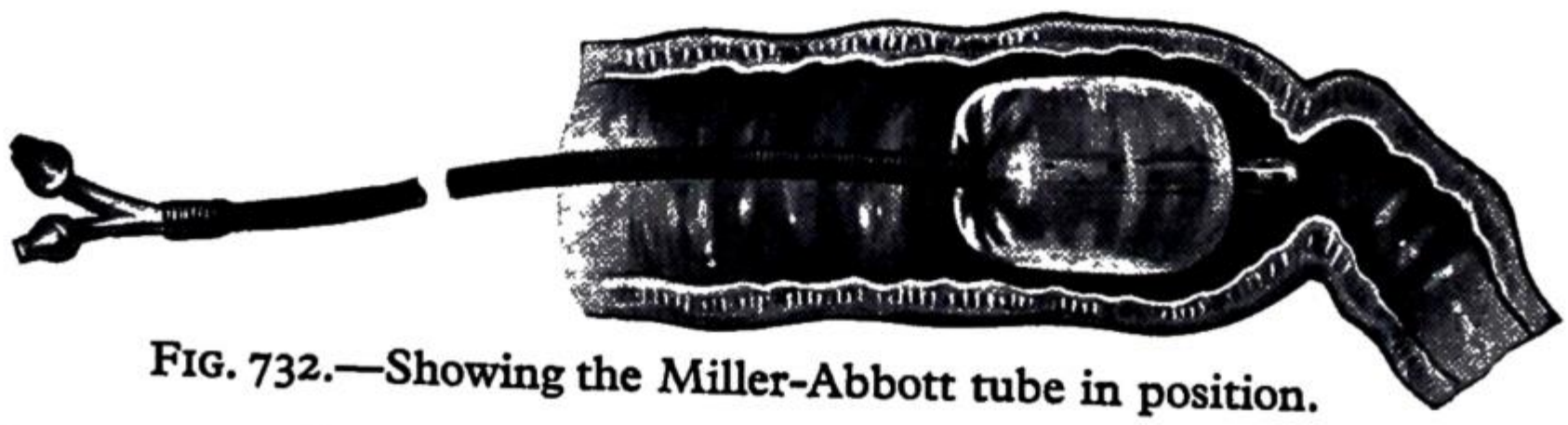


FIG. 732.—Showing the Miller-Abbott tube in position.

onward progress of the tube being arrested only at the site of considerable mechanical obstruction.

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[1] Even when strangulation can be ruled out, not more than six or eight hours should be expended in this form of treatment. If operation is delayed over twenty-four hours, in spite of suction drainage, the mortality is nearly doubled.

Frank T. Paul, 1851–1941. Surgeon, Royal Infirmary, Liverpool.

There are two main types of gastro-intestinal tube :

The Miller-Abbott Double Lumen Tube.—One-third of the lumen is used for inflation of the balloon which is attached to the distal extremity of the tube ; two-thirds of the lumen are devoted to suction drainage, viz.——————→ 

The tube is 10 feet 8 inches (3·2 metres) in length, and is calibrated in centimetres.

The Cantor gastro-intestinal tube is a $\frac{3}{16}$-inch (0·5 cm.) single lumen tube to the end of which is attached a balloon containing 5 ml. (1¼ drachms) of mercury. The distal 20 inches (50 cm.) of the tube above the balloon is provided with multiple side holes set 1 inch apart. These holes, which maintain very free drainage, also serve to calibrate the tube. Advantages of the tube are the weight of mercury enables the pylorus to be negotiated with less difficulty, the comparatively wide lumen, and the multiple side holes minimise blockage.

The Passage of a Gastro-intestinal Tube.—After preliminary cocainisation of the wider naris, the bag, folded round the end of the tube and well lubricated with liquid paraffin, is passed into the naso-pharynx and thence, while the patient sips water, into the stomach. When 2 feet (60 cm.) has been passed, the contents of the stomach are aspirated by a syringe. The tube is supported by allowing it to hang in a loop of tape fastened to the cheek with adhesive strapping. After the contents of the stomach have been aspirated the tube is advanced inch by inch at intervals of five minutes, while the patient sips water, until 2 feet 6 inches (75 cm.) has been paid out. The patient then lies on his right side for half an hour, continuous suction drainage being maintained. A radiograph is taken and this should show the tube lying along the greater curvature with its tip directed to the pylorus, in which case the patient sips more water and the tube is advanced another 2 inches (5 cm.). In favourable cases the tip of the tube should now have passed beyond the pylorus. If the tube is shown to be curled up in the stomach, it is withdrawn to the 60-cm. (2-feet) mark, and the process repeated. It may take many hours to negotiate the pylorus ; in some instances the method fails. Once the pylorus has been passed the balloon is inflated with 30 ml. of air and about 6 inches (15 cm.) of freshly lubricated tube is inserted every twenty minutes. Sometimes the tube is carried onwards spontaneously, therefore a loop is always left in readiness between the nostril and the tape. Cantor's tube is advanced in much the same way, the mercury-filled bag being the impetus for propulsion along the intestine once the pylorus has been negotiated.

Suction.—O. H. Wangensteen showed that in order to remove gas as well as fluid from the intestine a sub-atmospheric pressure of 75 cm. (30 inches) of water applied to the tube is the optimum negative pressure. Continuous suction is effected by an electric pump or by water-operated suction. More commonly, intermittent aspiration by a syringe is employed.

Maintenance of Fluid and Electrolytic Balance.—Once the fluid and the salt loss have been restored—and commonly as much as 7 pints (3·5 litres) of normal saline solution is required if the patient shows signs of dehydration, but considerably less if such signs are absent—a balance chart must be compiled, and the daily needs of the patient adjusted accurately as described in Chapter v.

ACUTE INTESTINAL OBSTRUCTION OF THE NEWBORN

Congenital atresia[1] **and stenosis**[2] are the most common causes of intestinal obstruction in the newborn. The site of the obstruction is as follows :

Duodenum	33 per cent.
Jejunum	15 „
Ileum	25 „
Ascending colon . . .	10 „
Multiple sites . . .	17 „

[1] Atresia = imperforation.
[2] Stenosis = narrowing.

Thomas Grier Miller, Contemporary. Emeritus Professor of Medicine, University of Pennsylvania, Philadelphia, Pa.
William Osler Abbott, 1902–1943. Assistant Professor of Medicine, University of Pennsylvania, Philadelphia Pa. He died of leukæmia.
Meyer O. Cantor, Contemporary. Surgeon, Sinai Hospital, Detroit.

mixture becomes progressively inspissated (fig. 740). The infant is born with intestinal obstruction. At times the coil filled with inspissated meconium can be felt as a rubbery mass. A typical radiograph shows distended small intestine, some of which is mottled. Unlike ileal atresia, there is no abrupt termination of the gas-filled intestine.

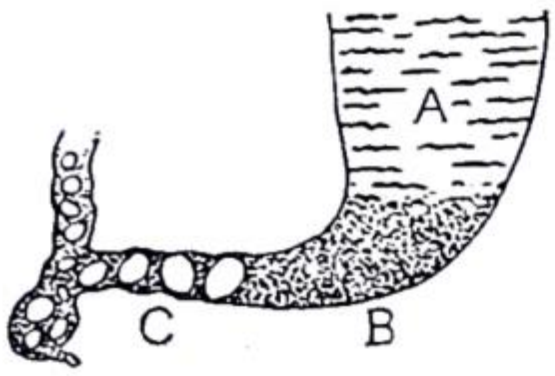

FIG. 740.—Operative findings in meconium ileus : (*a*) Treacly fluid ; (*b*) Putty-like material ; (*c*) Meconium pellets.

Pathognomonic Test.—Into a bowl of vomitus is placed a piece of exposed X-ray film, and there it is left for half an hour. If trypsin is present, the gelatine that constitutes the sensitised coat of the film will be digested off. In meconium ileus nothing happens, except that the film is rendered a little soft.

Treatment.—Laparotomy with the usual pre-operative preparation is most necessary. The only condition with which meconium ileus can be confounded is Hirschsprung's disease affecting the whole colon, but in the latter condition (*b*) and (*c*) of fig. 740 are lacking. A freshly made solution of 3 per cent. hydrogen peroxide 1 part, in water 3 parts, should be available. The coil containing the putty-like material is isolated with abdominal packs, and an incision $\frac{1}{2}$ inch (1·3 cm.) is made into the antimesenteric border of the intestine. Through the opening a catheter is inserted, at first in a proximal direction, and 10 ml. of hydrogen peroxide is injected. After waiting for several minutes, an endeavour is made to milk the viscid contents through the opening. By repeating this process a number of times the obstructing material can be expressed both from above and below the opening. This accomplished, fluid meconium is aspirated with a sucker. No solution other than hydrogen peroxide has the property of loosening this extremely viscid material, which adheres to everything with which it comes in contact even more tenaciously than glue. When the operator is satisfied that the obstruction has been relieved, the opening in the intestine is sutured securely. The coil, mechanically cleansed, is replaced, after which the abdomen is closed.

Post-operative Complications.—In the section dealing with fibrocystic disease of the pancreas, attention has been directed to the susceptibility of infants with mucoviscidosis to pulmonary complications. Oxygen and antibiotic therapy, in addition to the usual post-operative régime for intestinal obstruction, are therefore mandatory.

ACUTE INTUSSUSCEPTION

One portion of the gut becomes invaginated into another immediately adjacent ; almost always it is the proximal into the distal. Very rarely indeed is an intussusception retrograde.

Ætiology.—In a few cases there is some understandable cause, for, at the apex of the intussusception, a polyp, a papilliferous carcinoma, a submucous lipoma, or an inverted Meckel's diverticulum is found protruding. Obviously such a protrusion invites intussusception (fig. 741). In intussusception of infants it is generally agreed that :

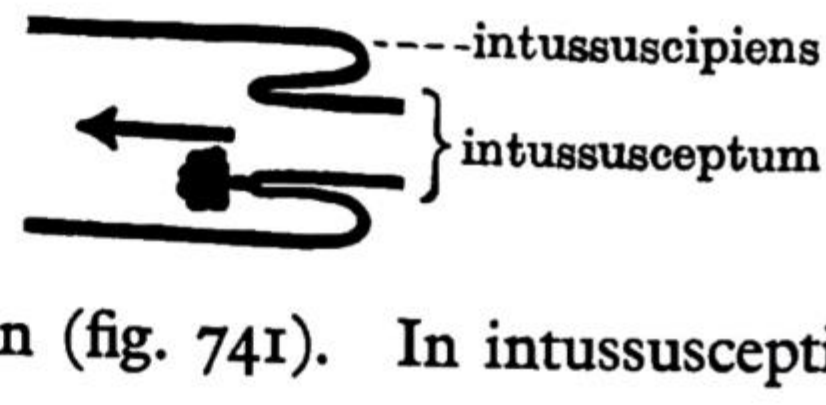

FIG. 741.—The mechanism of the production of an intussusception.

1. Idiopathic intussusception occurs most often between the sixth and ninth months.
2. Between the sixth and ninth months there is a change in the infant's diet—it is weaned.
3. An idiopathic intussusception usually commences in some part of the last 2 feet (60 cm.) of the small intestine.
4. The maximum aggregation of Peyer's patches is in the lower ileum.

Johann Peyer, 1653–1712, Successively. Professor of Logic, Rhetoric, and Medicine, Schaffhausen, Switzerland.

in forty-eight hours of birth, a satisfactory procedure, is a Paul-Mikulicz anastomosis (fig. 736). After the abdomen has been closed, and the wound protected with a dressing, a small Paul's tube is tied into the proximal and a small catheter into the distal limb of the spur. Four days later the tubes are removed and a crushing clamp is applied to the septum. When the stenosis lies in the jejunum, so devastating are the effects of even a temporary jejunal fistula that primary end-to-back anastomosis (fig. 737), although a more difficult operation, is the one that must be recommended.

FIG. 737.—End-to-back anastomosis. (*After Denis Browne.*)

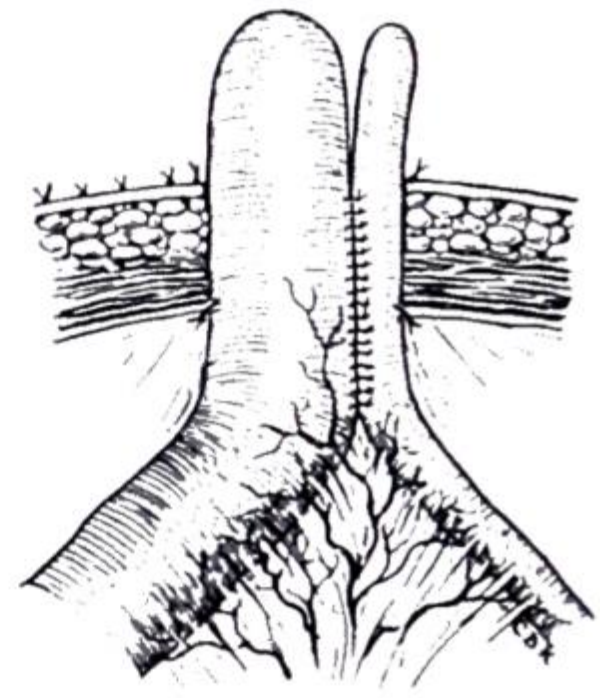

FIG. 736.—Spur enterostomy for ileal atresia. (*After G. H. McNab.*)

Arrested Rotation.—The cæcum remains in the right hypochondrium, and a peritoneal band is found running from the cæcum to the right side of the abdomen, and then across the second part of the duodenum. This is the transduodenal band of Ladd (fig. 738). The symptoms (repeated vomiting), due to pressure on the duodenum, and the radiographic appearances are identical with those of duodenal stenosis.

Treatment.—Early laparotomy is called for. The pressure on the duodenum can be relieved immediately by dividing the attachment of the band near the parietal peritoneum. Often there is a second peritoneal band, extending from the middle line to the commencement of the jejunum ; this also must be divided. The cæcum, now freed, is placed on the left side of the abdomen, and the abdomen is closed. The results of timely operation in these cases are excellent.

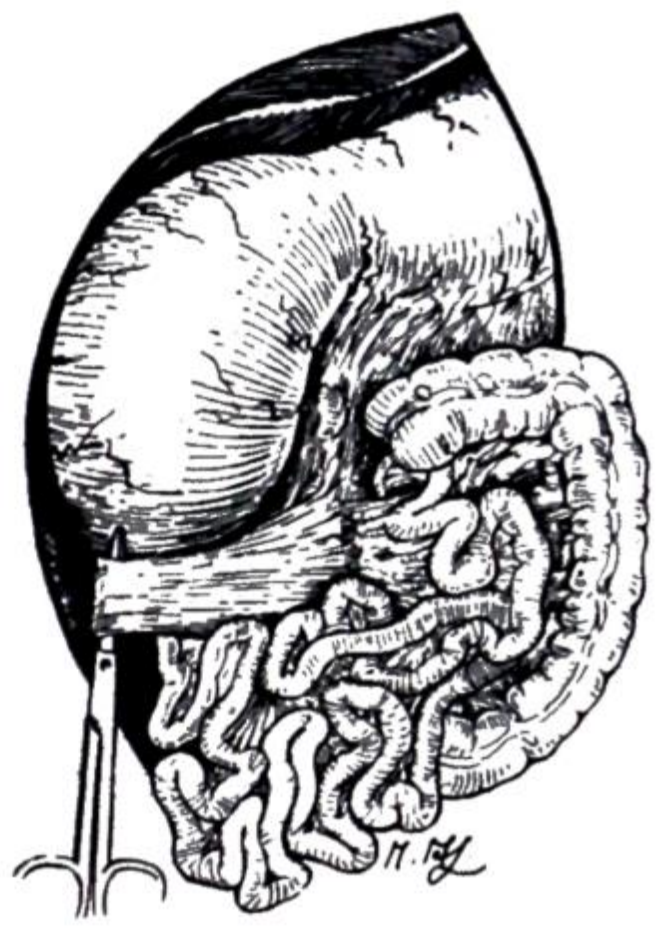

FIG. 738.—Incomplete rotation of the cæcum and Ladd's transduodenal band compressing the duodenum. (*Mr. V. Swain, London.*)

Volvulus of the Midgut (*syn.* Volvulus Neonatorum).—Arrested rotation described above predisposes to volvulus of the midgut. The floating cæcum, together with the whole of the small intestine, which has a narrow attachment, revolves. Broadly speaking, clinical features are similar to, and the radiological findings are identical with, those of arrested rotation which, indeed, is present. The onset, however, is more catastrophic, and dehydration occurs more rapidly than in arrested rotation *per se*. In addition, abdominal distension is often evident.

FIG. 739.—Volvulus of the midgut.

Treatment.—When the abdomen is opened, only distended coils of small intestine (which may or may not be cyanotic) and the stomach are seen. The whole of the midgut must be delivered on to the surface, where the intestine is protected with warm, moist abdominal packs. Only after this step has been taken is it possible to see the volvulus (fig. 739) which usually takes place in a clockwise direction. Untwisting is only half the operation ; of equal importance is to divide the second obstructive lesion—the transduodenal band of Ladd—which is often present.

Meconium ileus is the neonatal manifestation of mucoviscidosis (fibrocystic disease of the pancreas, see p. 461). The terminal ileum becomes filled with meconium admixed with viscid mucus, notably from the pancreas, and during the latter months of fœtal life this

Johann von Mikulicz-Radecki, 1850–1905. Professor of Surgery, Breslau.
William E. Ladd, Contemporary. Emeritus Professor of Child Surgery, University of Harvard, Boston, Mass.

Theory

1. Change of diet brings about a change of intestinal flora.
2. This predisposes to inflammation of the intestinal tract.
3. Which in turn causes inflammation of Peyer's patches.
4. As a result Peyer's patches swell.
5. A swollen Peyer's patch produces an elevation protruding into the lumen of the gut comparable to one of the known causes of intussusception.

Another theory

When the terminal ileum is contracted by a spasm of intestinal colic, it is guided through the ileo-cæcal valve by the 'bloodless' fold of Treves (see p. 579), which is well developed in infancy.

Intussusception occurs commonly at holiday time when children are taken out and given unsuitable food.

Pathology.—An intussusception is made up of three parts :

1. The entering, or inner, tube.
2. The returning, or middle, tube.
3. The sheath, or outer tube.

The outer tube is called the *intussuscipiens*. The inner and middle tubes together form the *intussusceptum*. The neck is the junction of the entering layer with the mass. That part which advances is the apex, and the mass which constitutes the intussusception (fig. 742) increases as it advances.

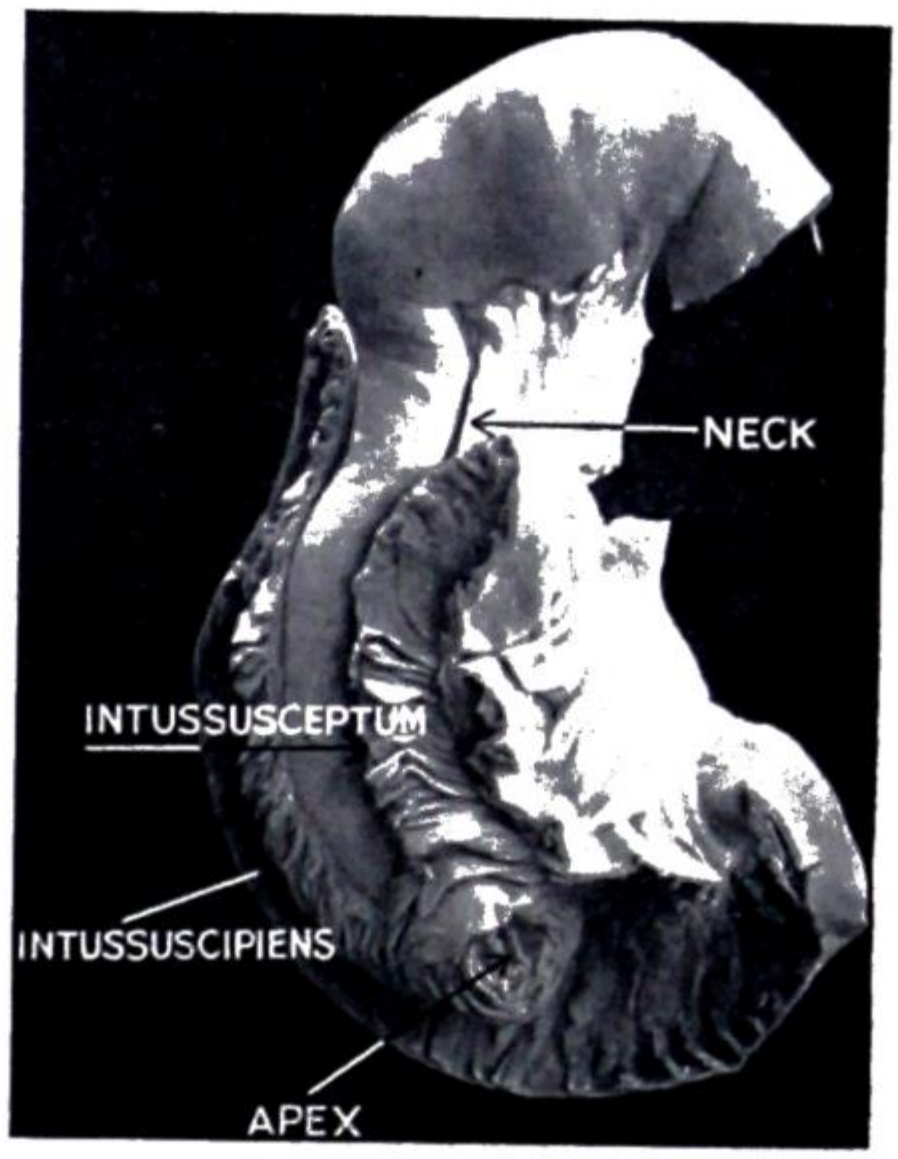

FIG. 742. — An intussusception dissected to show its constituent parts.

The blood supply of the inner layers of the intussusception is liable to be cut off. It will be appreciated that the onset of early gangrene is dependent upon the tightness of the invagination. Because of the great pressure exerted upon it by passing through the ileo-cæcal valve, an ileo-colic intussusception provides most examples of early gangrene.

Varieties (fig. 743).—The following is a simple classification :

1. *Ileo-ileal.* Ileum is invaginated into ileum . .	Approx.	8	per cent.
2. *Ileo-colic.* An ileo-ileal intussusception which has passed through the ileo-cæcal valve into the colon .	,,	36	,,
3. *Ileo-cæcal.* The ileo-cæcal valve is the apex of the intussusception	,,	46	,,
4. *Cæcal.* The caput cæci becomes invaginated . .	,,	2	,,
5. *Colo-colic.* The colon is invaginated into the colon .	,,	8	,,

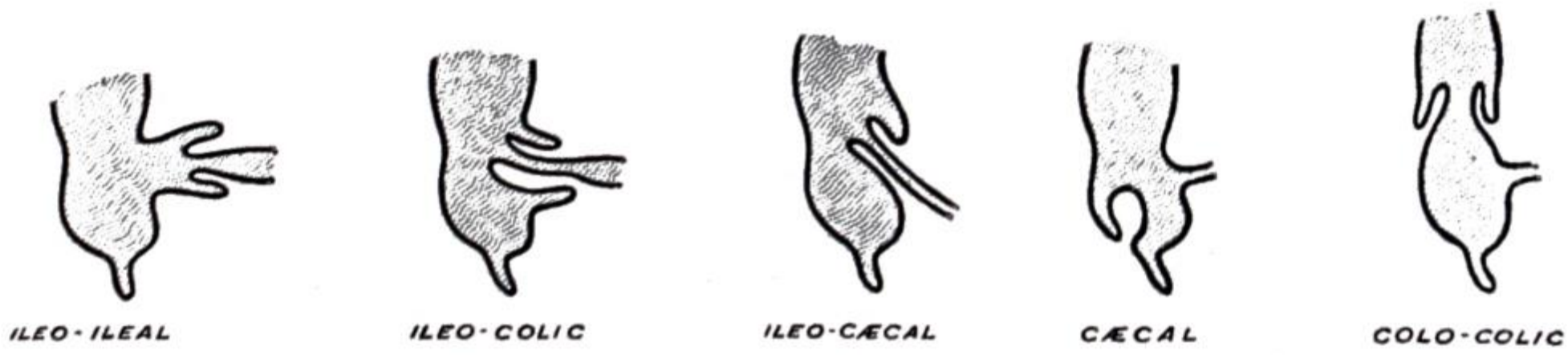

FIG. 743.—Varieties of intussusception.

Clinical Features of Intussusception in Infants.—Usually the patient is a fine, lusty male child between six and twelve months of age and in perfect

Sir Frederick Treves, 1853–1923. Surgeon, The London Hospital.

health when attacked. The onset is sudden. The child has a paroxysm of abdominal pain, draws up his legs, and screams. He may vomit shortly after the onset of the attack, but this is not a constant occurrence ; not until the intussusception has been present for about twenty-four hours is vomiting a conspicuous feature. The attacks, which last a few minutes and recur at about every fifteen minutes, are accompanied by facial pallor. They become progressively more severe. In between the attacks he lies listless and looks somewhat drawn. In the early stages frequently a normal stool is passed ; later blood and mucus are evacuated, which together are very characteristic, and constitute the well-known ' red-currant jelly ' stool.

On Examination.—The abdomen is not distended. It is important to palpate with a warmed hand between the spasms if possible while the baby is asleep. Typically a lump, which hardens on palpation, is felt in some part of the course of the colon (fig. 744), but if the lump is lying under the right

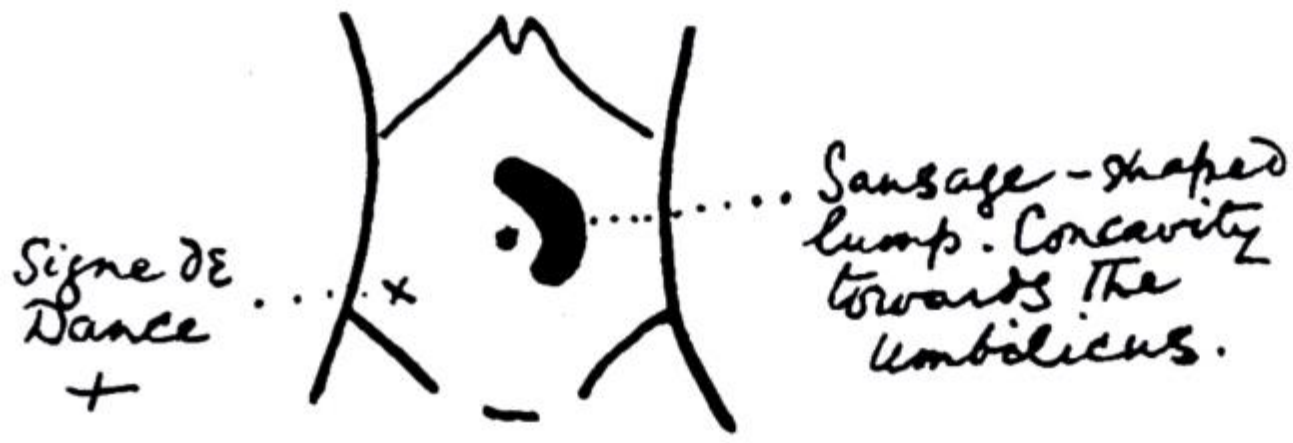

FIG. 744.—The physical signs recorded in a typical case of intussusception in an infant.

or left costal margins, sometimes it is not possible to feel it, even under an anæsthetic. There is said to be a feeling of emptiness in the right iliac fossa (*signe de Dance*). On rectal examination, if the intussusception has travelled far enough, its apex, a conical mass which is aptly likened to the cervix uteri, will be felt, especially on bimanual examination. In the majority of cases the intussusception has not advanced far enough along the colon for the apex to be felt per rectum, but in more than 60 per cent. of cases blood-stained mucus will be found on the examining finger.

In the remaining 40 per cent. the occult blood test often is positive, and the small amount of blood left on the finger-stall, undetectable by the naked eye, is sufficient for the test.

In a few instances the intussusception actually protrudes through the anus. This does not necessarily imply that the intussusception is of long standing, but rather that the patient is possessed of a long mesentery, rendering the small intestine unduly mobile.

Unrelieved, the pain becomes continuous. After twenty-four to thirty-six hours the abdomen commences to distend, and vomiting becomes copious. Absolute intestinal obstruction follows, and death from this cause, or from peritonitis secondary to the gangrene, is the rule. Once in a while a natural cure, due to sloughing of the intussusceptum, has been reported.

Radiography.—Plain films of the abdomen usually reveal increased gas shadows in the small intestine, and at times an absence of the cæcal gas shadow. Radiography following a barium enema gives positive evidence of the presence of an ileo-colic intussusception, but it must be understood that if the intus-

Jean Dance, 1797–1832. Physician, Hôpital Cochin, Paris.

susception is ileo-ileal, and the ileo-cæcal valve is competent, this form of examination is negative.

Differential Diagnosis

1. *From Acute Entero-colitis.*—Unlike intussusception, diarrhœa is a leading symptom. As in intussusception, abdominal pain and vomiting often occur; likewise blood and mucus may be passed, but in entero-colitis fæcal matter or bile is always present in the stools.
2. *From Purpura with Intestinal Symptoms* (*syn.* Henoch's Purpura).—There is likely to be the characteristic rash, which might be mistaken for flea-bites. Intussusception is a not uncommon accompaniment of this form of purpura, consequently the differential diagnosis is not of vital importance, for exploratory laparotomy must be performed in suspicious cases.
3. *From Prolapse of the Rectum.*—This can be eliminated readily. In prolapse the projecting mucosa can be felt continuous with the perianal skin. In intussusception protruding from the anus, the finger passes indefinitely into the depths of a sulcus (see figs. 835 and 836, p. 640).

Intussusception in adolescence is nearly always caused by an inverted Meckel's diverticulum (see p. 521).

Intussusception in adults is most often due to a papilliferous carcinoma; consequently, the colo-colic type is frequent. Rarer causes are a papilloma or a submucous lipoma. Idiopathic intussusception is extremely uncommon except in Egypt immediately after the Mohammedan fasting seasons (A. Mooro).

TREATMENT OF INTUSSUSCEPTION

Preliminary Treatment.—Half an hour should be devoted to improving the general condition of the patient by the administration of dextrose-saline solution. Especially in cases of more than twelve hours' duration, gastric aspiration should be carried out and continued during and after the operation.

Reduction by Hydrostatic Pressure.—In early cases, Australasian surgeons[1] in particular favour reduction by hydrostatic pressure. In the operating theatre an unlubricated catheter is passed into the rectum of the anæsthetised infant. The catheter is connected to the tubing of a reservoir filled with saline solution, and elevated to the height of 3½ feet (1 metre). The solution is allowed to run into the bowel for four minutes, while the buttocks are pressed together to prevent escape of the fluid. The catheter is then removed, and the fluid is allowed to escape into a bowl. Several such injections are made. After the first injection the fluid returned is blood-stained; subsequently, if reduction of the intussusception is effected, flatus and fæcal matter are passed. If there is the *slightest* doubt as to whether the intussusception has been reduced completely, laparotomy is performed but, as a result of the hydrostatic pressure, in most instances the intussusception is so reduced in size, and is so near its place of origin, that a grid-iron incision in the right iliac fossa is all that is necessary. The more usual method of treatment is to proceed with the operation, without resorting to these preliminary measures.

Operative Reduction.—The abdomen is opened through a right lower paramedian incision. The first part of the reduction is accomplished by squeezing the lowest part of the sausage-like mass, and little by little the intussusception is reduced (fig. 745). The last part of the intussusception is the most difficult to reduce and should be withdrawn and gently compressed in a warm, saline-soaked pack, to lessen the œdema. In the majority of instances reduction is completed easily by squeezing the apex of the intus-

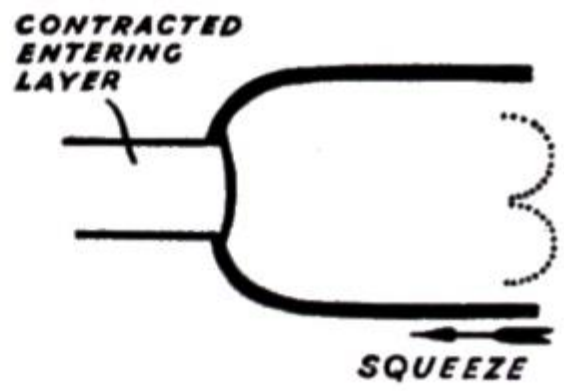

FIG. 745. — Diagram showing the method of reducing an intussusception.

[1] Intussusception is common in the urban districts of Australia.

Edouard Henoch, 1820–1910. Professor of Diseases of Children, Berlin.
Abdel Mooro, Contemporary. Professor of Surgery, Cairo.

FIG. 746.—Reducing the terminal part of the intussusception. (*After R. E. Gross.*)

susception, as shown in fig. 746. After reduction is completed, the terminal portion of small intestine, the caput cæci, and the appendix will be seen reddened and stiffened with œdema.

Unless the diagnosis has been unduly delayed, the intussusception can be reduced. Thanks to earlier diagnosis, the number of irreducible intussusceptions in infants is becoming smaller and the mortality is correspondingly lower.

Methods of effecting Reduction in Difficult Cases.—If the intussusception cannot be reduced in the manner described, the following methods are attempted, in sequence.

1. The little finger is inserted into the neck of the intussusception and an endeavour is made to separate adhesions between the intussuscipiens and intussusceptum, after which reduction is re-attempted (Cope's method).

2. The thumb and forefinger are placed as shown in fig. 747, and gentle pressure is exerted. Gradually the pressure is increased. In this way œdema is squeezed from the region of the ileo-cæcal valve (Sawyer's manœuvre). If, when reduction of the intussusception is attempted once more, it is found still to be irreducible, gangrene is imminent, or has occurred already, and resection must be carried out.

FIG. 747.—Sawyer's manœuvre to aid reduction of an intussusception.

Resection of an Irreducible or Gangrenous Intussusception.—The most expeditious, and probably the best method of carrying out resection and anastomosis is by Woodhall's ileo-transverse colostomy with exteriorisation of the divided ends of the ileum and transverse colon. An ileo-transverse colostomy is performed 2 inches (5 cm.) from the ends of the divided intestine and the abdominal wall is closed, leaving the clamps and about 1 inch (2·5 cm.) of the ends of the intestine protruding through the upper part of the incision (fig. 748). If distension occurs, as it often does during the first three or four days, this procedure permits removal of the clamp on the small intestine for a short time, with escape of gas and fluid fæces. After seven days the anastomosis should be functioning satisfactorily, and the open ends of the bowel can be closed extraperitoneally.

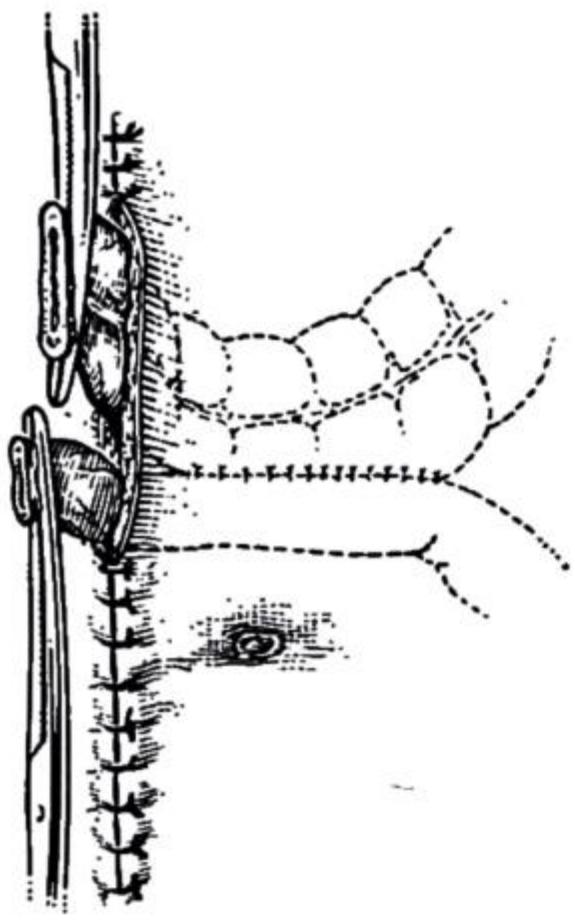

FIG. 748.—Woodhall's operation of ileo-transverse colostomy with exteriorisation of the divided ends of the small and large intestine.

Aiding Spontaneous Passage of a Detached Gangrenous Intussusception per Rectum.—An irreducible intussusception has been treated by inserting sutures so as to unite the intussuscipiens to the bowel entering the neck of the intussusception, special care being taken to close the space where the mesentery passes between the layers. By this measure peritonitis may be avoided, and occasionally the gangrenous detached intussusceptum is passed per rectum. Sloughing of the intussusception occurs and the continuity of the intestine is restored before the serious effects of intestinal obstruction arise (A. H. Montgomery). This method should be employed in desperate cases only.

Sir Zachary Cope, Contemporary. Consulting Surgeon, St. Mary's Hospital, London.
Kenneth C. Sawyer, Contemporary. Surgeon, Presbyterian Hospital, Denver, Colorado, U.S.A.
Barnes Woodhall, Contemporary. Professor of Neurosurgery, Duke University, Durham, North Carolina.
Albert Horr Montgomery, 1882–1948. Chief Surgeon, Children's Memorial Hospital, Chicago.

After-treatment.—When reduction is effected easily, gastric aspiration should be continued for twelve to twenty-four hours and dextrose-saline administered intravenously, or subcutaneously with hyaluronidase. In infants, nepenthe minims 1 (0·06 ml.) is given by mouth every three or four hours. On the second day the gastric tube is removed and sips of water are given. A few hours later feeding is commenced, with the mother's milk if the infant is breast-fed or, if artificially fed, with citrated cow's milk. In cases where resection has been necessary, more vigorous methods of immediate after-treatment are required. Plasma infusions are valuable to combat shock. When the hæmoglobin estimation is reduced, as it often is, blood transfusion is necessary.

Recurrent intussusception occurs in only 2 per cent. of cases of idiopathic intussusception. If a second operation is necessary to reduce an intussusception, in the endeavour to avoid still further recurrence the last few inches of the ileum should be anchored to the ascending colon by sutures.

VOLVULUS

Compared with intussusception, volvulus is rare in this country. A volvulus is caused as a result of axial rotation of a portion of the alimentary tract.

(*a*) **Volvulus Neonatorum** (see p. 569).

(*b*) **Volvulus of the small intestine,** other than the above, usually occurs in the lower ileum, and is favoured by the presence of an adhesion passing from the antimesenteric border of an intestinal loop (fig. 749) to the parietes or to the female pelvic organs. In Africans, volvulus involving many feet of small intestine without causative adhesions occurs rather commonly. The consumption of a large meal of maize and vegetables seems to predispose to the condition.

FIG. 749.

Treatment is to untwist the loop, if possible. A causative adhesion should be divided and the stump of its intestinal attachment buried in the intestinal wall by a purse-string suture. When the intestine is gangrenous, resection followed by anastomosis is required.

(*c*) **Volvulus of the cæcum** occurs occasionally, especially when the right half of the colon is lax and mobile, and again it is favoured by a band of adhesions from the caput cæci to the peritoneum of the right iliac fossa, such as may follow appendicitis. Volvulus of this part of the large intestine occurs nearly always in a clockwise direction. The first twist obstructs the ascending colon; if a second twist occurs, it obstructs the ileum also. The highest incidence is between twenty-five and thirty years of age. The symptoms are those of acute obstruction of the small intestine. In about 25 per cent. of cases there is a palpable tympanitic swelling not, as a rule, in the right iliac fossa, for in process of torsion the mobile cæcum moves out of the right iliac fossa into the mid-abdomen, or even to the left side. A plain radiograph shows loops of gas-filled ileum, and sometimes an especially large gas shadow which can be recognised as the cæcum. At first the obstruction is not absolute; fæces and flatus may be expelled after an enema, but unless spontaneous untwisting occurs, the distension and attacks of intestinal colic continue.

Treatment.—Laparotomy should be performed. In early cases it is usually possible to untwist the organ. Sometimes before untwisting can be accomplished it is necessary to deflate the ballooned cæcum by the insertion of a hollow needle. A purse-string suture is inserted and tied before the hollow needle is withdrawn. Untwisting should be followed by cæcostomy, which serves two purposes—it relieves distension and it fixes the organ to the abdominal wall, thereby preventing a recurrence. If the cæcum is gangrenous or its viability is not assured, Woodhall's method of excision with ileo-transverse colostomy (see fig. 748) is to be recommended.

(*d*) **Volvulus of the pelvic colon** is common in Eastern Europe, India, Scandinavia, and Peru. The predisposing causes are indicated in fig. 750. The loop

may rotate half a turn, in which event spontaneous rectification sometimes occurs. After the loop has rotated 1½ turns the veins involved in the torsion are compressed, and the loop becomes greatly congested. If, as is sometimes the case, it rotates more than 1½ turns, the blood supply is cut off entirely and the loop becomes gangrenous. The rotation nearly always occurs in an *anti-clockwise* direction.

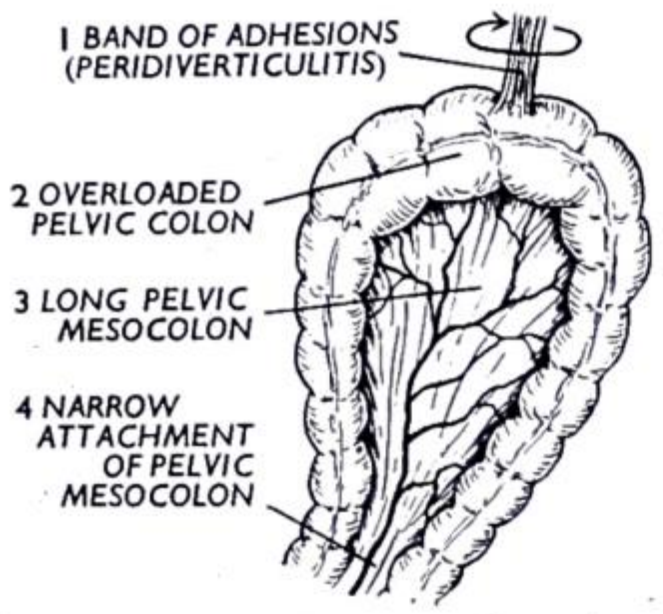

FIG. 750.—The predisposing causes of volvulus of the sigmoid colon.

Clinical Features.—Males are more commonly affected than females, and the sufferers are usually middle-aged or elderly. There is often a history of chronic constipation, and sometimes a history of acute attacks of left-sided abdominal pain, probably due to a partial volvulus, that untwists itself and is followed by the passage of large quantities of flatus and fæces. As a rule the onset of volvulus of the pelvic colon is sudden and is characterised by severe abdominal pain, usually coming on while the patient is straining at stool. Abdominal distension soon follows; indeed, in no other condition does extreme abdominal distension occur so quickly. If the patient is examined two or three hours after the commencement of the attack, the distension is mainly left-sided. In a matter of six hours the whole abdomen becomes distended. Hiccough and retching occur early; vomiting is late. Constipation is absolute, but an enema may be returned blood-stained. Occasionally the rectal wall is felt or seen by sigmoidoscopy to be œdematous; this is due to venous engorgement consequent upon the superior hæmorrhoidal vein becoming occluded by the torsion.

Treatment.—In early cases an attempt should be made to pass a rectal tube with the patient in the knee-elbow position. Sometimes the tube can be passed with the aid of a sigmoidoscope when blind insertion fails. If this does not succeed, laparotomy must be performed.

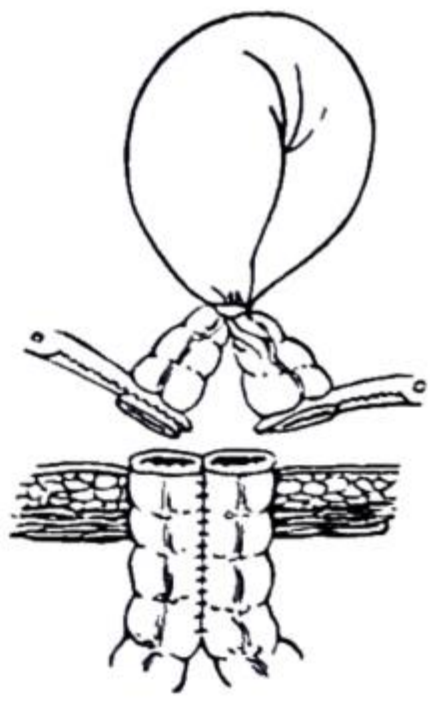

FIG. 751.—The Paul-Mikulicz operation applied to volvulus of the pelvic colon.

While an attempt should be made to untwist the gas-filled viscus, often this step is difficult or impossible because of adhesions. The safest method of procedure is to exteriorise and resect a volvulus of the sigmoid in order to prevent recurrence or subsequent mesenteric thrombosis, even if the involved colon appears viable (John Bruce).

Redundancy makes the performance of this procedure (fig. 751) simple.

OBSTRUCTION BY ADHESIONS AND BANDS

By Adhesions

Type 1.—Post-operative *fibrinous* ('bread and butter') adhesions are unlikely to produce complete intestinal obstruction unless an element of functional paresis (paralytic ileus) is present also—a not infrequent occurrence. This type of obstruction commences between the third and sixth post-operative days.

Type 2.—Post-operative *fibrous* adhesions can give rise to intestinal obstruction at any time from months to many years after an abdominal operation.

Type 3.—Adherence of a loop or loops of intestine to an inflamed intraperitoneal structure, e.g. a tuberculous mesenteric lymph node (see fig. 664, p. 507), is a fairly common cause of intestinal obstruction.

Type 4 is a complication of the plastic form of tuberculous peritonitis.

Type 5 follows chemical irritation—(*a*) iodine or other skin antiseptics, (*b*) talc glove powder entering the peritoneal cavity by accident, or (*c*) sulphonamide or penicillin placed there by design.

John Bruce, Contemporary. Regius Professor of Surgery, Edinburgh.

Post-operative adhesions giving rise to intestinal obstruction usually involve the lower ileum. Operations for acute appendicitis necessitating drainage of the peritoneal cavity or drainage of an appendix abscess are the most common precursors of this condition, while gynæcological operations follow closely as the second most common cause of obstructing adhesions.

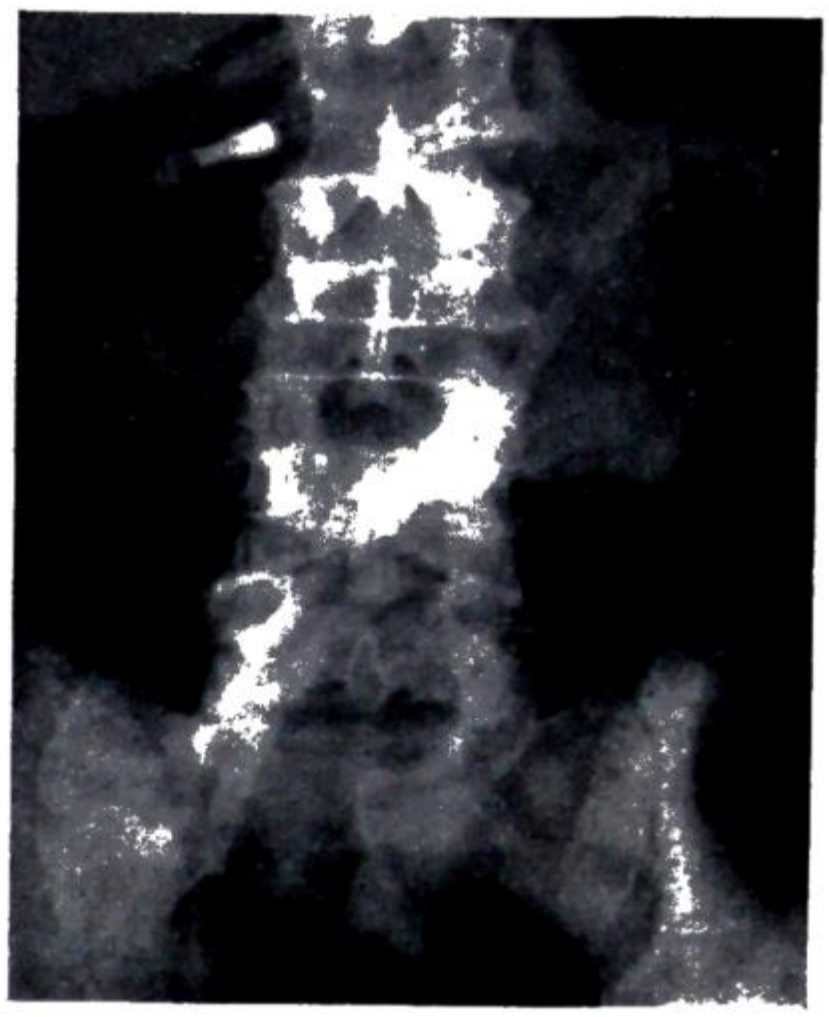

FIG. 752.—Tip of a Miller-Abbott tube arrested in the ileum. Case of obstruction to the small intestine due to adherence of the ileum to the transverse colon after resection of a carcinoma of the transverse colon. The condition of the patient was much improved by suction drainage, after which the coil was freed by operation.

Treatment.—Gastro-intestinal suction drainage (fig. 752), combined with intravenous fluid therapy, is extremely beneficial; occasionally it is curative, but only in *Type* 1. On this account especial vigilance is necessary, for strangulation, if not present initially, is liable to develop during the course of such treatment. When, as is usually the case, operation is required, although many adhesions are often present, frequently only one of them is found to be the cause of the obstruction, in which case the condition can be remedied easily by dividing this adhesion. At other times the intestine is angulated by adherence to the parietes, to the mesentery, to another coil of intestine (fig. 753) or, in the female, to the uterus or adnexa. In these circumstances it is sometimes possible to free the obstructed intestine by dissection. In order to prevent recurrence the bare areas should be covered with omental grafts.

FIG. 753.—Adhesions causing intestinal obstruction by angulation.

In *Type* 1 the adhesions (which are the only ones that are sometimes tided over their obstructive phase by suction drainage) can usually be broken with a finger, thereby releasing the obstruction. When adhesions are widespread and the small intestine so matted that a definite point of obstruction cannot be found, anastomosis of an obviously distended loop to a collapsed loop has often resulted in relief of the obstruction. When preliminary gastro-intestinal suction drainage has been effective, the obstructed coils will be empty, in which case anastomosis should be performed between the collapsed coil containing the extremity of the tube and the most proximal collapsed coil not containing the tube.

By a Band

A band (usually one band only is culpable) is occasionally the cause of acute obstruction to the small intestine. Such a band may be :

(*a*) Congenital : most often an obliterated vitello-intestinal duct (see p. 670).

(*b*) A string-like, frequently thin and fragile, band (fig. 754) following previous bacterial peritonitis.

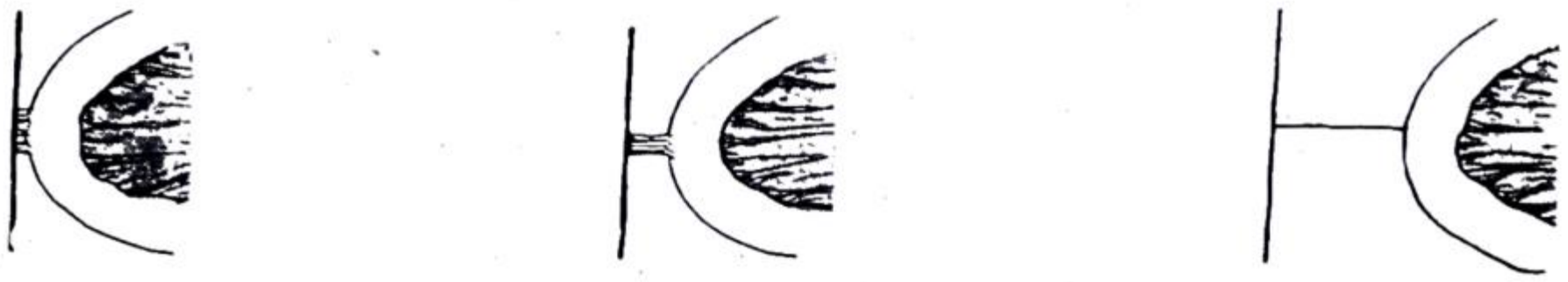

FIG. 754.—Metamorphosis of an inflammatory peritoneal band from an adhesion.

(*c*) A portion of great omentum, adherent usually to the parietes, constitutes an obstructing band of relative stoutness (fig. 755).

FIG. 755.—Intestinal obstruction occurring under a portion of great omentum adherent to the parietal peritoneum.

Treatment.—Provided the involved intestine is viable, division of the band and release of the obstructed loop is the simplest and one of the most satisfactory operations in surgery. Entrapment of a coil of small intestine beneath a string-like band is notorious for producing gangrene (fig. 756) early. Even when the imprisoned intestine, after release, regains its pristine hue, if the narrow areas that have suffered direct compression by the band show any colour changes, they should be invaginated. Gangrenous intestine must be resected.

FIG. 756.—Strangulation by a band from Meckel's diverticulum, leading to gangrene. (*Dr. L. C. Hermitte, Sheffield.*)

OBSTRUCTION DUE TO AN INTERNAL HERNIA

A portion of the small intestine passes into one of the retroperitoneal fossæ or into a congenital defect of one of the mesenteries, there to be imprisoned. The potential internal herniæ are as follows :

1. * **The Foramen of Winslow.**—The portal vein, common bile duct, and hepatic artery lie in its free border.
2. * **A Hole in the Mesentery.**
3. **A Hole in the Transverse Mesocolon.**
4. **A Defect in the Broad Ligament.**
5. **Congenital or Acquired Diaphragmatic Hernia** (see Chapter xliii).
6. **One of the retroperitoneal fossæ,** of which the following are most important :

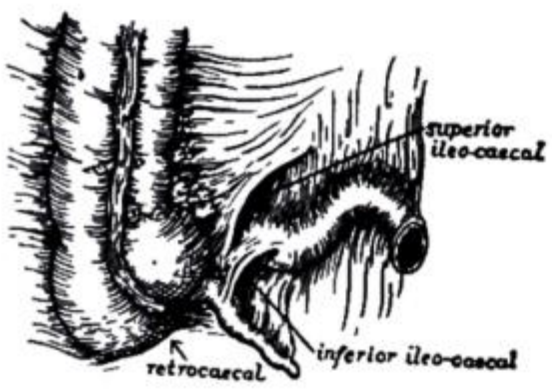

FIG. 757.—Peritoneal fossæ around the cæcum and appendix.

Fossæ about the Duodenum

(*a*) * *The Left Paraduodenal Fossa.*—The inferior mesenteric vein lies in its free border.

(*b*) * *The Right Duodeno-jejunal Fossa.*—The superior mesenteric artery runs in its free border.

Fossæ about the Cæcum and Appendix (fig. 757).

(*a*) *Superior Ileo-cæcal Fossa.*—Between the general mesentery and a fold of peritoneum raised by the anterior cæcal branch of the ileo-colic artery.

(*b*) *Inferior Ileo-cæcal Fossa.*—Between the 'bloodless' fold of Treves and the mesentery of the appendix.

(*c*) *The retro-cæcal fossa* behind the cæcum.

The Intersigmoid fossa is situated in the base of the pelvic mesocolon. Its mouth looks downwards.

The supravesical fossa is bounded by the median and lateral umbilical folds (obliterated urachus and umbilical arteries). A loss of weight and a coincident loss of prevesical fat deepens the fossa.

An internal hernia is an uncommon cause of intestinal obstruction; a pre-operative diagnosis that such obstruction is due to an internal hernia can only be a guess.

Treatment.—In those internal herniæ marked with a * a vital blood-vessel runs in the edge of the hernial orifice, and to divide the constricting agent (which is the correct treatment for the non-* varieties) would result in disaster. When confronted with the difficult problem of how to release imprisoned intestine without dividing the constricting agent, the reader is referred to the principle invoked in fig. 758. In order to reach the obstructed coil, the peritoneum over the fundus of the sac must be incised when such is present. In the case of the foramen of Winslow, the lesser sac is opened between the stomach and the colon to give access to the imprisoned loop.

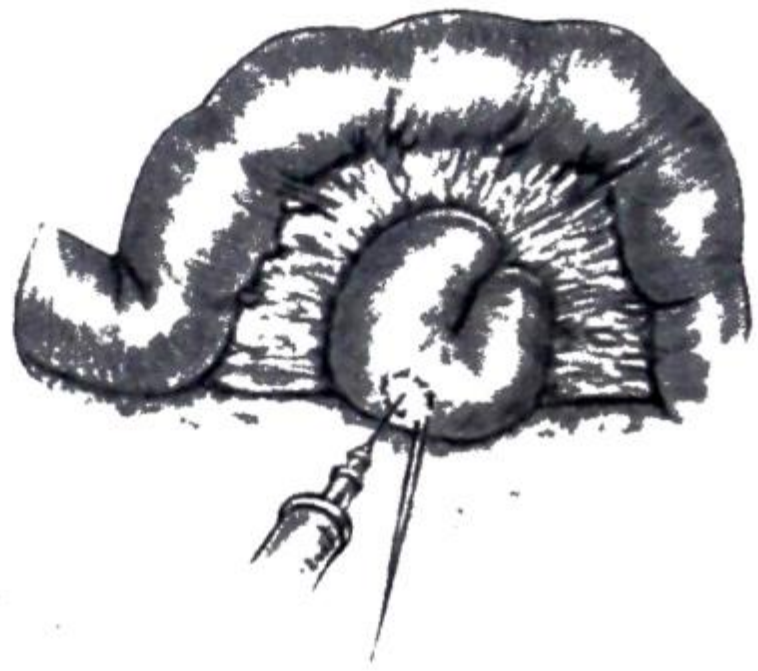

FIG. 758.—Strangulation through a hole in the mesentery. Emptying the obstructed coil before attempting reduction.

OBSTRUCTION FROM STRICTURE OF THE SMALL INTESTINE

Cicatricial contracture is usually an aftermath of tuberculous ulceration. Multiple strictures are usually present, and the one with the smallest lumen becomes blocked with food residue or such object as a fruit stone. Malignant stricture is rare, although both carcinoma and sarcoma occur from time to time (p. 543).

Treatment.—Usually a simple stricture can be circumvented by lateral anastomosis. A malignant stricture of the small intestine should be resected widely and the continuity of the intestine restored by end-to-end anastomosis.

OBSTRUCTION BY OBTURATION OF THE SMALL INTESTINE

Obstruction by gall-stone (*syn.* gall-stone ileus) usually occurs in old women. The gall-stone, which is 1 inch (2·5 cm.) or more in diameter, slowly ulcerates through the neck of the gall-bladder into the duodenum. It passes down the small intestine, and becomes impacted about 2 feet (60 cm.) from the ileo-cæcal valve, because this is the narrowest part of the small gut. The symptoms are elusive. The patient experiences colic, accompanied by copious vomiting. As the obstruction is incomplete, there is often a fair result from an enema, and remissions of symptoms are frequent, but the vomiting returns, and by this time it is bilious; as time goes on it becomes fæculent. Occasionally a radiograph demonstrates the calculus

Jacob Winslow, 1669–1760. A Dane who migrated to Paris, and there established a School of Anatomy.

within the small intestine. In any event, the small intestine will be seen to be distended with gas. There is no abdominal distension until late, and late intestinal obstruction in an old person is an almost hopeless condition. On rare occasions a large gall-stone ulcerates through the gall-bladder into the transverse colon, and from time to time it remains in the large intestine long enough so to increase in size, by the deposition of fæcal matter upon it, that eventually it becomes impacted in the rectum.

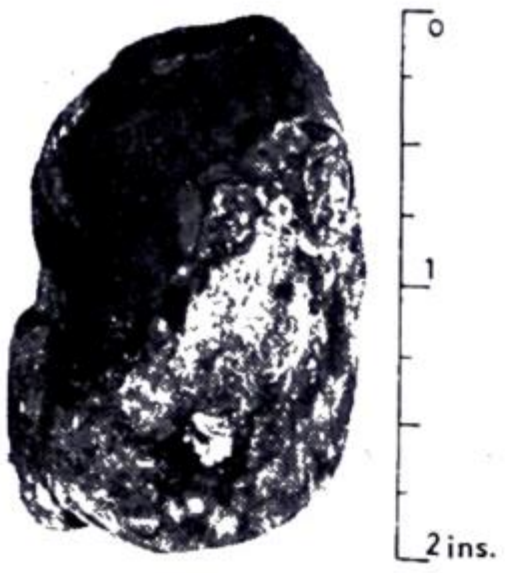

FIG. 759.—Gall-stone removed from the lower end of the ileum.

Treatment.—A lower right paramedian incision is made. The loop containing the stone is delivered and packed off most carefully. After the application of rubber-covered clamps, the intestine is opened and the stone removed. It is an advantage to sew up the incision in the intestine transversely, to avoid constriction at this point. A gall-stone that causes intestinal obstruction is nearly always barrel-shaped and unfaceted (fig. 759). If there is a facet, it is advisable to palpate the intestine above the obstruction for a possible fellow-calculus.

Bolus obstruction is particularly liable to occur after partial gastrectomy. It will be appreciated readily that after partial gastrectomy insufficiently masticated articles of food will be hurried into the small intestine, perhaps there to become impacted, whereas normally they would be retained in the stomach until they had become partially digested. Brussels sprouts, green figs, and, particularly, unmasticated orange pulp have caused intestinal obstruction in these circumstances. Especially foodstuffs that swell, e.g. dried fruits swallowed in large lumps, as is likely in edentulous patients, and also articles that are poorly digested in the stomach, e.g. the sections of an orange, can cause obstruction of the small intestine after escaping through a normal pylorus.

Treatment.—Timely laparotomy is required. Often there is a considerable quantity of clear free fluid in the peritoneal cavity. An attempt should be made gently to squeeze the bolus onward into the cæcum, and there to break it up by kneading. Should the bolus be impacted so firmly that this manœuvre proves impracticable—and such is likely only in cases of obstruction by dried fruit or when a stricture of the small intestine is present—after isolating the coil with abdominal packs, enterotomy must be performed. After the obstructing material has been removed the opening in the intestine is sutured, the coil mechanically cleansed before returning it to the abdomen, which is then closed.

Obstruction by stercolith gives rise to symptoms identical with those of obstruction by a gall-stone, for a stercolith, contrary to what might be expected, is usually formed and found in the small intestine, particularly in cases where a diverticulum or a stricture is present in the ileum. On careful dissection of a stercolith it is not unusual to find a nucleus of recognisable material, e.g. tomato skins.

Obstruction due to Worms.—An aggregation of *ascaris lumbricoides* is sometimes the cause of low small intestinal obstruction in children, usually under ten years of age, and especially those living in or near the tropics. There is debility out of proportion to that produced by the obstruction. The obturation is inclined to follow the ingestion of an anti-helminthic. If it is not known that the patient is suffering from ascaris infestation, a worm in the vomitus or the presence of eosinophilia may be the means of making a correct pre-operative diagnosis. In this form of intestinal obstruction laparotomy must be performed, but if possible the tangled mass should be kneaded along the ileum into the cæcum. Although opening the intestine and removing worms has been followed by success, it has also been followed by fatal post-operative peritonitis from a worm insinuating itself through the suture-line.

EMBOLISM AND THROMBOSIS OF THE MESENTERIC VESSELS

Arterial embolism is more common than spontaneous thrombosis, and the superior mesenteric vessels are implicated far more frequently than the inferior; probably this is due to the better collateral circulation of the latter.

Embolism of the Superior Mesenteric Artery.—The embolus is derived from a vegetation on the mitral valve, the left auricular appendage, an atheromatous plaque arising from the aorta, or from a pyæmic infarct of the lung which has led to thrombosis of the pulmonary vein.

Primary thrombosis of the superior mesenteric artery is the result either of arteriosclerosis or thrombo-angiitis obliterans.

Primary thrombosis of the superior mesenteric veins occurs occasionally in portal hypertension and in pylephlebitis.

No matter whether the occlusion is arterial or venous, hæmorrhagic infarction occurs although, in the case of embolism, a short-lived pallor has been observed at early laparotomy. The intestine and its mesentery become swollen and œdematous, demarcation between infarcted and healthy intestine being gradual. Blood-stained fluid is exuded into the peritoneal cavity and the lumen of the infarcted intestine becomes filled with blood. When the main trunk of the superior mesenteric artery becomes occluded, infarction of nearly the whole of the small intestine, the cæcum, and part of the ascending colon occurs (fig. 760). More frequently a branch of the main vessel alone is implicated and the area of infarction is considerably less.

Clinical Features.—An embolus lodging in the superior mesenteric artery or thrombosis occurring in the corresponding vein gives rise to the same train of symptoms, but the former is likely to be more precipitous.

Repeated vomiting ushers in this catastrophe.

Facial pallor and a fall in systolic pressure closely resembles an internal hæmorrhage; indeed, the volume of blood lost to the circulation by imprisonment in the infarcted area, for practical purposes *is* a serious internal hæmorrhage.

Abdominal rigidity is usually present, and is widespread in embolic cases.

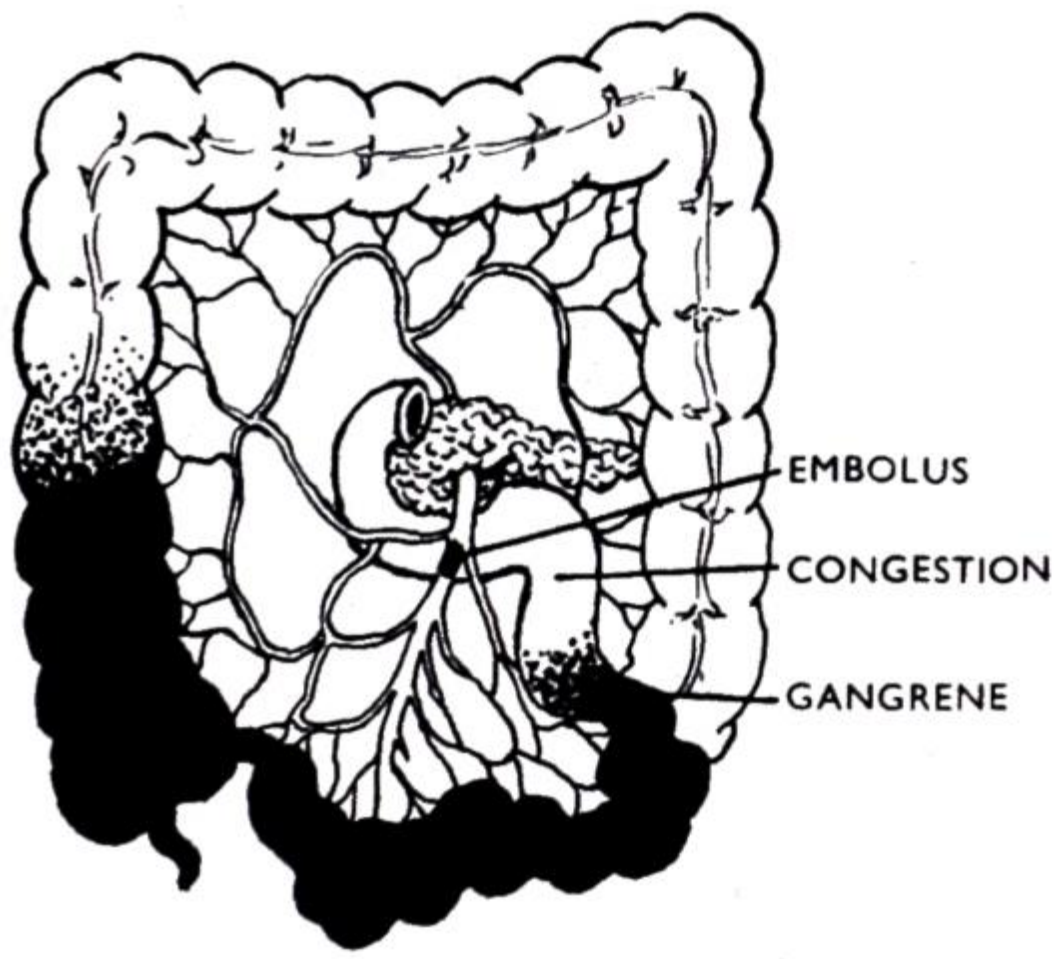

FIG. 760.—Embolus lodged in the main trunk of the superior mesenteric artery. Showing the widespread gangrene that results.

Shifting dullness can be demonstrated readily; at least some free (blood-stained) fluid is present. Infarcted coils also respond to the law of gravity.

Rebound tenderness is a constant sign.

Sometimes an indefinite lump can be felt in thin subjects.

In 20 per cent. of cases the patient passes pure blood per rectum, and in a further proportion blood is revealed in the result of an enema.

When it is known that the patient is suffering from one of the conditions that predispose to mesenteric thrombosis and embolism, the correct diagnosis is not difficult.

Treatment.—While astounding cases of resection of up to 19 feet (5·8 metres) of infarcted small intestine with complete recovery have been reported, when the diagnosis is certain, or has been confirmed at laparotomy, *and over half the small intestine is involved*, a conservative course is more than justified, especially in the frail and elderly. Remarkable recoveries have resulted also solely from the administration of heparin, followed by such products as dindevan (given orally) in their proper sequence, combined with blood transfusion. In a few instances superior mesenteric embolectomy[1] has been performed. In all cases, blood transfusion is

[1] Robert S. Shaw, contemporary, Chief of the Vascular Clinic, Massachusetts General Hospital, performed the first successful embolectomy for mesenteric arterial occlusion in 1956.

required. In patients who recover from non-operative measures, intestinal obstruction is liable to occur later from fibrosis of an area of small intestine.

A conservative attitude cannot be recommended for cases of infarction of the large intestine. The least uncommon of these rare cases is lodgment of an embolus in the middle colic artery. Here resection of the transverse colon with closure of both ends and a temporary cæcostomy is indicated. When the walls of the large intestine are lifeless, so swift is the onset of peritonitis that resection of any portion of infarcted colon, with a terminal colostomy, offers the patient the only chance, slender as it may be.

PARALYTIC ILEUS (*syn.* ADYNAMIC ILEUS)

Neurogenic obstruction is due to failure of a segment of intestine to transmit peristaltic waves. In the majority of instances it is the ileum which is first and chiefly affected—hence the term paralytic ileus. The fault lies in the local neuromuscular mechanism, probably in the nerve plexuses of Auerbach, situated between the circular and longitudinal muscle fibres of the intestine, and those of Meissner, in the submucous layer. As a result of the paralysis, a large amount of fluid and gas collects in the intestines, the consequent distension interfering with the absorption of both gas and fluid (see p. 556). This leads to a further increase in the distension. The local effect of this is to cause partial obstruction by kinking of the apices of the coils (fig. 761).

FIG. 761.—Partial obstruction occurring at each end of greatly distended coils by reason of the distension. (*After J. Devine.*)

Following every abdominal operation, peristalsis ceases for about twenty-four hours. Often it returns in one segment and is delayed in another. This is the cause of the common 'gas pains' in the immediate post-operative period. Paralytic ileus often follows extensive abdominal operations. It is also frequently encountered after operations for diffuse or pelvic peritonitis. In the latter instance the primary condition is believed to be due to bacterial toxins diffusing from the peritoneum and causing direct damage to the intramural nerve plexuses. Plastic adhesions between loops of the ileum also play a part. These adhesions do not constrict the intestine or angulate it sufficiently to cause intestinal obstruction when peristalsis is normally vigorous, but when peristaltic waves become enfeebled, such 'bread and butter' adhesions become formidable obstacles. Ileus following peritonitis is therefore mainly paralytic but partially mechanical.

Clinical Features.—Paralytic ileus should be suspected if the patient has neither passed flatus nor is there any result from an enema forty-eight hours after an abdominal operation. This suspicion becomes confirmed if there is no evidence of resumption of peristalsis after sixty hours. At first the distension is most apparent below the level of the umbilicus, but as the condition progresses the whole abdomen becomes involved. If gastric aspiration has not been commenced at an early stage, which is unlikely, the patient vomits 'mouthfuls' repeatedly in an effortless manner. With gastric aspiration large quantities of bile-stained material continue to be withdrawn and the material recovered by gastro-intestinal aspiration be-

Leopold Auerbach, 1828–1897. Professor of Neuropathology, Breslau.
Georg Meissner, 1829–1905. Professor of Physiology, Göttingen.

comes brown and fæculent. There is an absence of intestinal colic ; indeed, there is no abdominal pain and no tenderness, except that to be expected in the region of the incision. On auscultation of the abdomen, in contradistinction to mechanical obstruction, there is an ominous silence.

In severe cases the abdomen becomes tense and drum-like, and the pulse-rate rises steadily.

Reflex paralytic ileus presumably is due to interference with the extrinsic nervous control of intestinal musculature. It occurs in a number of heterogeneous conditions, which include fracture of the spine or ribs, a retroperitoneal hæmorrhage, or the application of a tight plaster jacket.

Paralytic Ileus in Uræmia.—Abdominal distension, vomiting, and hiccough are frequent accompaniments of advanced uræmia. Abdominal distension following prostatectomy may be uræmic in origin, although in some instances it occurs when the output of urine is satisfactory and the blood urea is normal or but little raised. In such cases the distension is probably reflex and it appears to affect the large more than the small intestine.

Management and Treatment.—Increase or decrease in the girth of the abdomen at the level of the umbilicus, measured with a tape-measure left in position, should be recorded four-hourly. Radiography shows many coils of small intestine filled with gas and often multiple fluid levels. Radiography, repeated frequently, is valuable in assessing the effect of treatment on the distension. The general consensus of opinion is that all types of peristaltic stimulants have no place in the treatment of paralytic ileus. Too often they activate only the comparatively less affected jejunum and force more gas and some fluid into the paralysed ileum (fig. 762), with the result that the distention increases and a greater length of small intestine becomes totally paralysed.

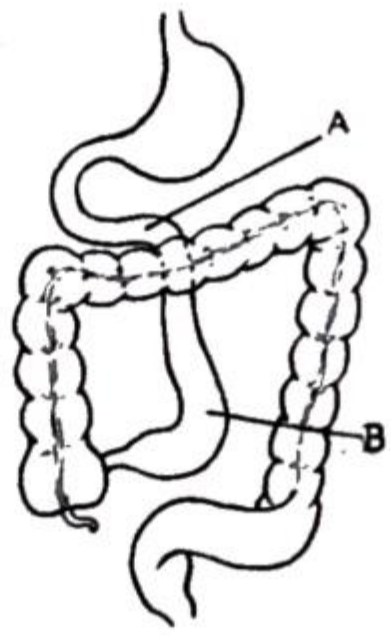

FIG. 762.—Showing how peristaltic stimulation by drugs often not only fails to relieve but increases paralytic ileus.
(*a*) Comparatively healthy small intestine which responds to the stimulation, and thereby forces intestinal contents into (*b*) the paralysed segment which, unable to contract, becomes still further distended.

Morphine and its derivatives, especially if repeated, are likely to inhibit normal intestinal propulsion and may cause segmental tonic contraction of the small intestine. Pethidine or amidone, which are free from these objections, should be substituted. Calcium pantothenate, 50 mg., injected intramuscularly and repeated in six hours, if required, is valuable, especially in early cases. Pantothenic acid is a component of the vitamin B complex, and is concerned in the physiological elaboration of acetylcholine, which is necessary for normal contraction of involuntary muscle. The fluid and electrolytic balance must be adjusted daily by the administration of dextrose-saline intravenously, the amount required being assessed by keeping a fluid intake and output chart. Careful watch must be kept that neither hypochloræmia nor hypokalæmia develop, as both these deficiencies favour

paralytic ileus. As hypoproteinæmia also favours paralytic ileus, on the third or fourth day from the onset 1 pint (568 ml.) of the total fluid administered should be given as a plasma infusion. This can be repeated at intervals of forty-eight hours, as necessary. It is in cases of paralytic ileus that the Miller-Abbott or other type of intestinal tube with continuous suction drainage is especially valuable, but in a number of instances great difficulty or failure is experienced in negotiating the pylorus. In cases of successful intubation of the distended intestine, if the measurements and radiograph show that distension is decreasing, the intestinal tube is clamped for a few hours, and provided there is no vomiting or no increase in the distension, the tube can be removed and an enema given. Should this be followed by even the passage of flatus, the paralysis probably has been overcome. In some instances the distension returns and the gastro-intestinal tube must be replaced. Throughout the treatment a rectal flatus tube is passed every six hours. When paralytic ileus persists and an intestinal tube cannot be passed beyond the pylorus, a lateral procaine block of the nerves of the abdominal wall (fig. 763) sometimes is followed by immediate resumption of peristaltic activity. When this measure fails, jejunostomy for the purpose of inserting a Miller-Abbott or other intestinal tube through a small opening in the jejunum sometimes proves a life-saving measure.

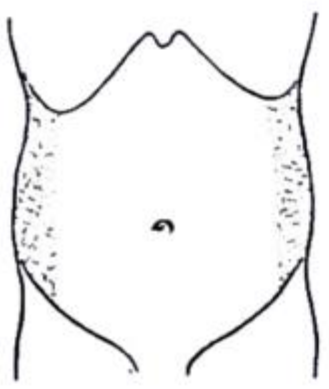

FIG. 763.—Extent of the procaine block of the abdominal wall.

In cases following peritonitis, when the symptoms persist in spite of six or seven days of conservative treatment, especially if cessation of suction drainage is followed by abdominal pain and some intestinal sounds can be heard on abdominal auscultation, operative treatment is indicated, as the condition is partly mechanical. A left upper paramedian incision is made, and the first loop of distended small intestine to be encountered is anastomosed to the transverse colon. After this procedure the patient often passes flatus within twenty-four hours, and in forty-eight hours there is an action of the bowels.

RECURRENT ATTACKS OF ACUTE INTESTINAL OBSTRUCTION

Recurrent attacks of intestinal obstruction due to adhesions usually are the aftermath of peritonitis. The difficult problem of preventing further attacks has, to a large extent, been solved by a plication operation. When a patient has had two or more attacks of intestinal obstruction due to adhesions, necessitating operation for its relief, the arguments in favour of performing a plication operation are weighty.

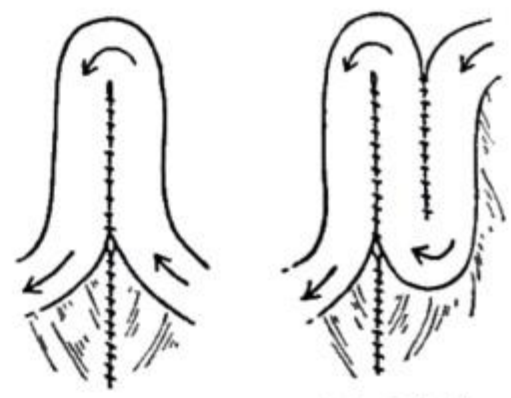

FIG. 764.—Noble's plication operation.

Noble's plication operation (fig. 764) must never be performed until about six weeks to two months after the last urgent operation. After the abdomen has been opened by an adequate incision, all involved intestine is freed by dissection. Adjacent coils (average length 6 to 8 inches (15 to 20 cm.)) are sutured together along their mesenteric borders. The resulting folds of mesentery are also united. This accomplished, the abdomen is closed.

Thomas Benjamin Noble, Contemporary. Surgeon, Indianapolis, Indiana.

CHRONIC INTESTINAL OBSTRUCTION

The various abnormalities and diseases giving rise to chronic intestinal obstruction are described in Chapters xxv and xxviii. There remains one condition to be considered here.

Fæcal impaction occurs principally in elderly, and often bed-ridden, patients. The mass of hardened fæces usually accumulates in the upper part of the rectum.

The symptoms are those of chronic intestinal obstruction, and attacks of spurious diarrhœa are a common accompaniment. A fæcal accumulation may form a palpable abdominal mass which can be indented. When the mass is situated in the rectum, it can be indented by the palpating finger.

Treatment.—Enemata are usually insufficient. The patient must be given an anæsthetic, and after the anal sphincter has been dilated the mass is removed digitally or with a spoon. In some instances fæcal impaction occurs above an innocent or malignant stricture of the rectum. In such cases, it may be necessary to resort to left iliac colostomy before the mass can be disimpacted.