ABDOMINAL OPERATIONS

Fifth Edition

RODNEY MAINGOT, F.R.C.S.

volume 2

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48 PORTAL HYPERTENSION

Alan H. Hunt

Obstruction to the flow of portal blood leads to portal stagnation, a rise of portal venous pressure, and congestion of the organs which drain into the portal tree, especially the spleen. The blood is diverted through naturally occurring communicating veins into the systemic circulation, especially at the cardiac end of the stomach and lower oesophagus. It is this condition that is now referred to as portal hypertension, although the elevated portal pressure is but one of its aspects.

PORTAL VENOUS PRESSURE

The normal portal pressure, as taken directly from a radicle of the portal vein, is about 100 mm. water, with limits of 50 to 200. It depends on the systemic arterial pressure and the vena caval pressure and fluctuates slightly with respiratory movements. These same factors influence the exaggerated pressure which exists in cases of portal obstruction, when it is usually about 300 mm. of water. Occasionally it may be as high as 600 mm. and yet sometimes pressures of no more than 150 mm. are encountered in cases of obvious portal stasis.

METHOD OF MEASURING

The simplest method of measuring the pressure is by means of a cerebrospinal fluid manometer to which is attached a 300-mm. length of rubber tubing carrying a No. 2 "serum" needle (Fig. 1). The whole, including the bulb of the manometer, is filled with 3.8 percent sodium citrate solution and the tube is clamped. Air locks must be avoided. The needle is inserted into a convenient vein (usually an upper jejunal vein), while the manometer is held vertically well up above the needle. The clamp is then removed. The citrate will run down the manometer, which is lowered and balanced off quickly against the portal venous pressure. The reading is obtained by adding the vertical distance between the point of the needle and the zero mark on the manometer (measured with a stainless steel foot rule calibrated in millimetres) to the height of the meniscus up the manometer. Care must be taken to ensure that there is no interference whatsoever in the free communication between the main portal venous trunk, the radicle from which the pressure is being obtained, and the citrate in the manometer. Slight fluctuations in the meniscus synchronous with respiratory movements give adequate proof of the freedom of the flow and therefore of the accuracy of the reading. Many other methods have been devised, examples being those described by Taylor and Egbert (*Surg Gynec Obstet 92*:64, 1951), Gray (*Ann Roy Coll Surg 8*:354, 1951), by Sherlock and her colleagues (*Lancet 1*:918, 1953; *1*:1325, 1954) in which the portal pressure is estimated by occlusive catheterisation of the hepatic vein or at the time of splenic puncture.

A single reading may suffice, but sometimes it may be necessary to take pressures from different parts of the portal venous tree to determine the site of the obstruction.

PORTAL OBSTRUCTION

CAUSES

These are outlined by Whipple (*Ann Surg 122*:449, 1945), Rousselot (*Surgery 8*:34, 1940), and Hunt (*Portal Hypertension*, 1958).

1. *Extrahepatic (prehepatic)*

(a) A primary obliteration or constriction of the portal vein is encountered in children and adolescents. It is presumed to be brought about in a number of these cases, when no other cause can be elucidated, by an extension of the obliterative process affecting the ductus venosus and the umbilical vein at birth (Thompson, *Ann Intern Med 14*:255, 1940). Neonatal umbilical sepsis is an accepted aetiological factor, and exchange transfusions through the umbilical vein a possible additional factor. The portal vein is usually replaced by a number of tortuous varicosities, the so-called cavernoma. The

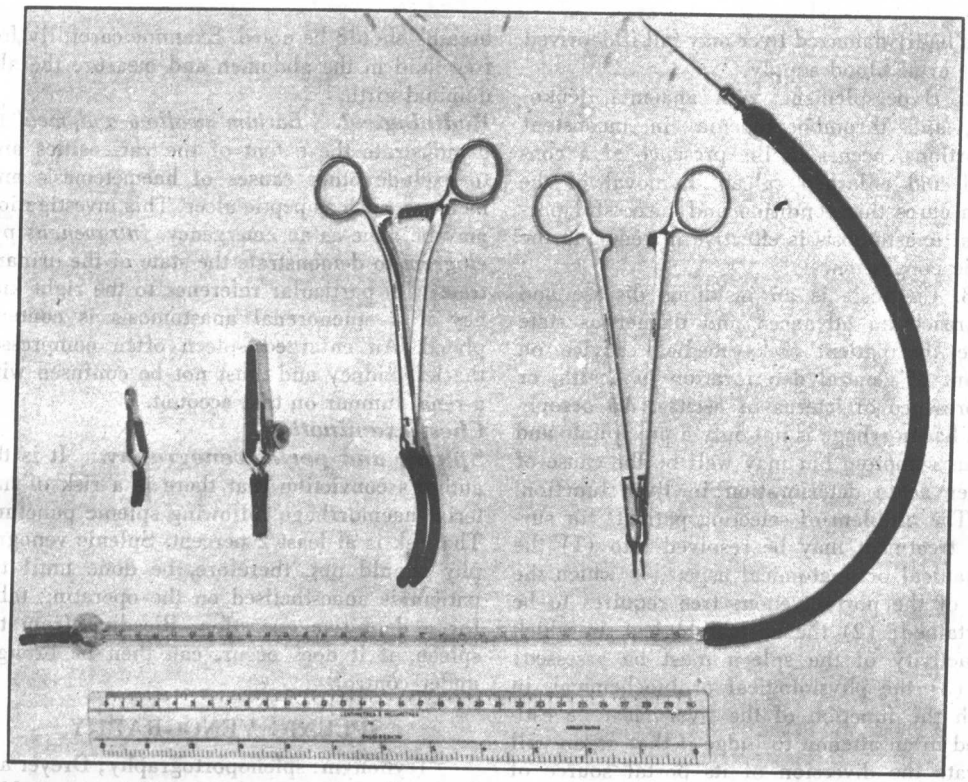

Fig. 1. To illustrate special types of instruments: pressure manometer; Blalock, Blakemore and Tom Smith's clamps; long needle-holder with jaws 1.5 mm wide. (The two latter instruments were supplied by Down Bros. and Mayer and Phelps, Ltd.)

extent of the obstruction and the nature of these cavernomatous channels can be demonstrated by venography.

(b) Acquired thrombosis of the portal vein, e.g., following portal pylephlebitis.

(c) Compression or obliteration of the portal, splenic, or mesenteric veins may be caused by many conditions such as carcinoma of the stomach or pancreas, or growths arising outside the portal system, such as hypernephroma. Acquired conditions are less common than are the congenital type of obliteration and adults are usually affected.

2. *Posthepatic.*

Conditions such as constrictive pericarditis and thrombotic obliteration of the hepatic veins (Chiari's disease) cause portal hypertension as a result of posthepatic obstruction. The effects on the portal system are incidental.

3. *Intrahepatic.*

Cirrhosis hepatis of all types accounts for the great majority of cases of portal obstruction (87 percent). The height of the pressure is not proportional to the extent of the cirrhotic process (Hunt, *Brit Med J 2*:4, 1952).

Occasionally cirrhosis is associated with significant thrombosis of the portal vein, the obstruction being thus both intra- and extrahepatic.

CLINICAL ASPECTS

The presenting symptom complex associated with portal hypertension (Banti's syndrome) may be considered under three headings: (1) gastro-oesophageal haemorrhages; (2) hypersplenism; and (3) liver dysfunction.

1. Severe haemorrhages from the gastro-oesophageal varices are very dangerous and usually require to be treated surgically. The present-day operation of portal-systemic venous anastomosis was devised to lower the portal pressure (Blakemore and Lord, Jr., *Ann Surg 122*:476, 1945) in order to relieve the dangerously exposed gastro-oesophageal varices of their unwelcome burden of blood and thus permanently prevent further haemorrhage. How-

ever, a badly damaged liver may fail if deprived of its portal blood supply.

2. Hypersplenism, with anaemia, leukopenia, and thrombocytopenia, in inconstant proportions, occurs in the presence of a congested and enlarged spleen. Removal of the spleen cures this condition and successful portacaval anastomosis is effective in reducing the splenic congestion.

3. Cirrhosis is an insidious disease and may reach an advanced and dangerous state before the patient seeks medical advice on account of general deterioration in health, or the presence of icterus or ascites. An oesophageal haemorrhage is not only a precipitate and serious symptom but may well be the cause of further acute deterioration in liver function.

The problem of selecting patients for surgical treatment may be resolved into (1) the mechanical or anatomical aspect, in which the state of the portal venous tree requires to be ascertained; (2) the haematological, in which the activity of the spleen must be assessed; and (3) the physiological or biochemical, in which the function of the liver must be estimated in an attempt to judge if that organ will tolerate the diversion of its portal source of blood.

Careful study and investigation of all patients is, therefore, imperative. The following abbreviated review of certain necessary investigations makes no attempt to be comprehensive.

THE INVESTIGATION OF PATIENTS

Clinical. Enquire into accidents, operations or infections which might have caused damage to the portal venous tree, and into the past history with reference to the causes of cirrhosis: e.g., *infective hepatitis; dietary deficiencies;* habitual consumption of alcohol or the taking of medicine containing such hepatotoxic drugs as arsenic; syphilis and antisyphilitic treatment; tropical diseases such as schistosomiasis; any untoward results of pregnancy; evidence of chronic biliary obstruction.

Palmar blush and spider naevi may be the only clinical evidence of liver derangement. The liver and spleen need to be carefully defined, by palpation and percussion. (The upper level of the spleen can be ascertained by heavy percussion from above downward in the left midaxillary line.) The degree of icterus or jaundice and the presence or absence of "liver breath" should be noted. Examine carefully for free fluid in the abdomen and measure the abdominal girth.

Radiological. *Barium swallow and meal* to demonstrate the extent of the varicosities and to exclude other causes of haemetemesis and melaena, such as peptic ulcer. This investigation may be done as an emergency. *Intravenous pyelogram* to demonstrate the state of the urinary tract with particular reference to the right kidney if a splenorenal anastomosis is contemplated. An enlarged spleen often compresses the left kidney and must not be confused with a renal tumour on that account.

Chest examination

Splenic and portal venography. It is the author's conviction that there is a risk of material haemorrhage following splenic puncture. The risk is at least 2 percent. Splenic venography should not, therefore, be done until the patient is anaesthetised on the operating table for a definitive operation. Bleeding from the spleen, if it does occur, can then be brought under control.

SPLENIC VENOGRAPHY

(synonym: splenoportography; Dreyer and Budtz-Olsen, *Lancet 1*:530, 1952; Leger, *Mem Acad Chir 77*:586, 1951; *Splenoportography*, Springfield, Ill., Thomas, 1966; Abeatici and Campi, *Acta Radiol 36*:383, 1951; Walker *et al., Brit J Surg 40*:392, 1953; Atkinson *et al., Quart J Med 24*:77, 1955).

The patient is placed in the dorsal position. A standard fine lumbar puncture needle is inserted in the midaxillary line over the ninth or tenth interspace within 1 or 2 in. (2.5 to 5 cm.) of the left costal margin and directed horizontally inward through intercostal muscles and diaphragm. The point is then directed 10° or 15° headwards and slightly backwards and as the needle is further inserted it will be felt to enter the substance of the spleen. It should be advanced another ½ to 1 in. (1.3 to 2.5 cm.) into the substance of the spleen and its position confirmed by the backflow or aspiration of blood. Then 20 to 30 ml. of 45 percent Hypaque is injected as forcibly as possible and serial x-rays taken. The important films are those taken precisely at the end of the injection and 2 sec. later. A successful venogram will demonstrate splenic and portal vein and backflow up oesophageal varices and out into the systemic circulation (see Fig. 2).

If done under general anaesthesia, absolute apnoea is maintained during the 15 to 30 sec. of the investigation. If done under local anaesthesia, the needle, once inserted, must be free to move with respiration and the injection must be done through a flexible connecting tubing between syringe and needle. Some workers prefer to use a power-assisted device for the injection.

In the case of a very large spleen, the needle may be inserted over the abdomen.

Pain is experienced during the injection when the portal pressure is high and after the injection if there is backflow of Hypaque or blood along the needle track.

Splenic venography should not be done under local anaesthesia if the platelets are reduced to less than 100,000 or the prothrombin index is less than 50 percent.

PORTAL VENOGRAPHY

(Fig. 3), introduced originally by Blakemore, is done after the abdomen has been opened. Recently a method has been worked out of introducing the contrast medium into the portal vein by opening up the obliterated umbilical vein (Bayly, *Amer J Gastroenterol 41*: 235, 1964; Lavoie *et al., Canad J Surg 9*:338, 1966). Thirty millilitres of 45 percent Hypaque is injected rapidly into a radicle of the portal vein, usually a jejunal vein, and serial radiographs are obtained, the operator's fingers being protected by a lead shield during the exposures.

The two methods are often complementary to one another, in that neither can be depended upon to demonstrate more than half the portal tree. The Hypaque injected into the spleen, for example, may be diverted before it reaches the portal vein, giving an impression that the vein is absent. Since the most important purpose of these venographic methods is to obtain an exact definition of the portal vein, splenic venography alone may be very misleading.

Some workers, notably Sherlock and Leger (*Presse Med 66*:1673, 1958) use the occasion of the splenic puncture to estimate the portal venous pressure, but this is considered unwise for three reasons; it adds to the risks of splenic tear, it is not a method of proved or constant accuracy and a single pressure reading may be misleading.

Instrumental *Oesophagoscopy* should be used to demonstrate the presence of oesophageal varices if the barium swallow is inconclusive; *Gastroscopy*, only if oesophagoscopy is negative. The liver may usefully be examined by peritoneoscopy in a few doubtful cases, a method which may also be used to demonstrate peritoneal malignancy.

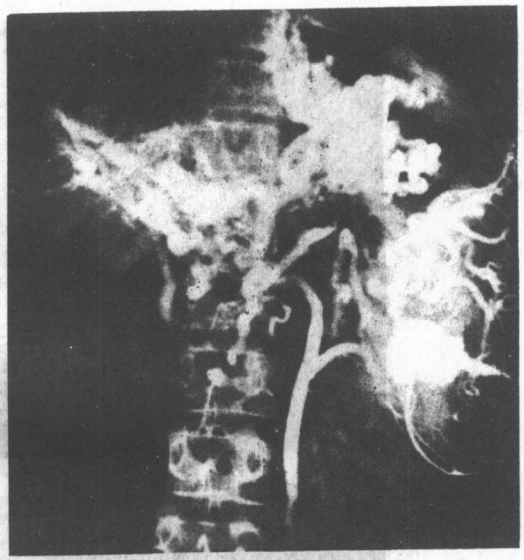

Fig. 2. Splenic venogram showing: 1, absence of splenic and portal veins; 2, replacement of these channels by collateral vessels, constituting the cavernoma; 3, diversion of portal blood up gastric and oesophageal varices, down the inferior mesenteric vein and out along many perisplenic veins. Splenectomy only possible and a bloody operation at that.

Pathological *Blood:* complete count and differential, with platelets and reticulocytes; bleeding, clotting, and prothrombin times; group and serum for cross grouping. Sternal puncture if any suggestion of failure of haemopoiesis. Serological tests for syphilis.

Liver-function tests The most valuable are those that estimate the capacity of the liver to manufacture blood proteins and eliminate abnormal constituents. The blood protein estimation and the bromsulphthalein elimination test, therefore, should be done in every case. "If the serum albumen stabilizes around 3.3 to 3.5 grams percent, other liver function values being equally favorable, the risk of shunting is not excessive" (Blakemore, *Surg Gynec Obstet 94*:443, 1952). The serum globulin level is not of such vital importance, nor the albumen-globulin ratio per se. Evidence produced by Blakemore (1952) "would seem to indicate that bromsulphthalein retention up to between

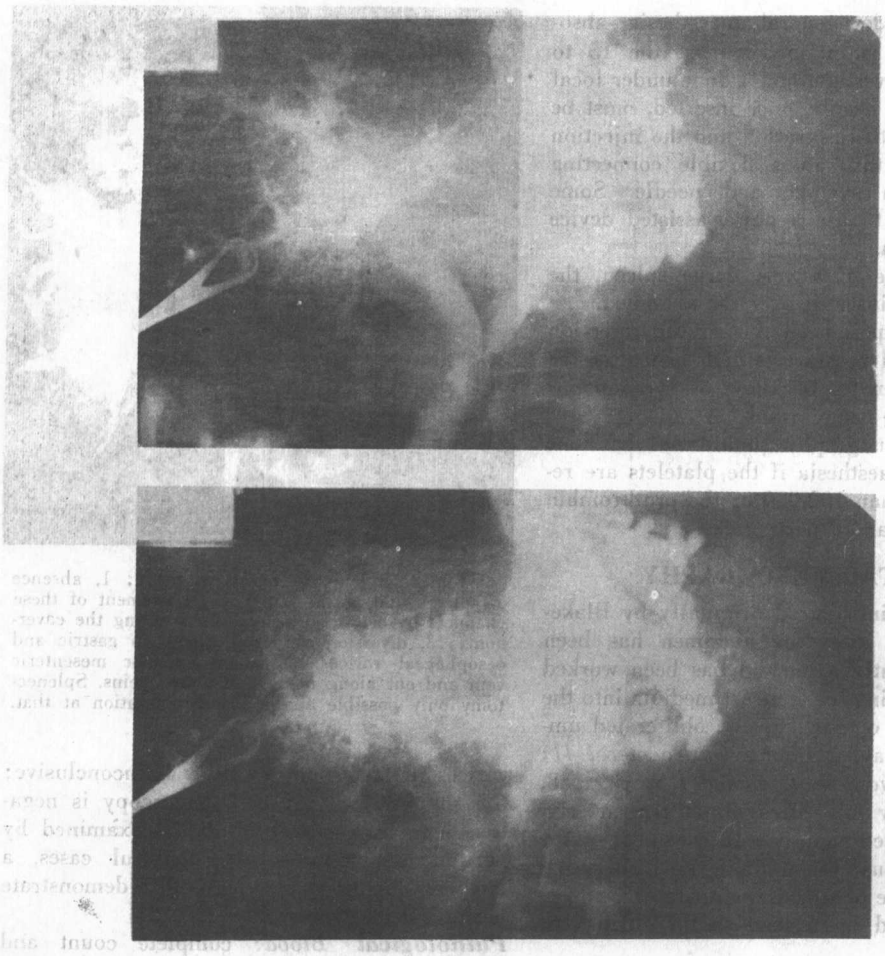

Fig. 3. Portal venogram showing a portal vein satisfactory for portacaval anastomosis. Much diversion up enlarged left gastric vein to gastric and oesophageal varices and peri-oesophageal venous plexus. Some blood is seen traveling to the liver in biliary collateral channels, the hepatopetal veins of Chiari.

20 and 25 percent (half an hour after injection) has little effect upon operative risk."

Tests indicating the amount of gamma globulins in the blood are uncertain in the presence of a hyperactive spleen. As a routine, however, the electrophoretic pattern of the serum proteins may be of diagnostic assistance.

Failure of the liver to restore the blood prothrombin to normal after intensive administration of vitamin K gives further evidence of liver failure. Indeed, many of the so-called *function* tests should more correctly be termed tests of *failure.*

The serum bilirubin and alkaline phosphatase estimations will indicate the presence or absence of biliary obstruction. The transaminases will give considerable assistance in evaluating the activities of parenchymatous disease within the liver.

Many other liver tests may be of assistance from time to time, but it is the author's intention to limit the "hepatogram" to those investigations which are essential in order to decide whether or not a cirrhotic patient is a reasonable operative risk. Tests of the liver's enzymatic function (the serum pseudocholinesterase) may prove to be of considerable value.

Puncture biopsy of liver (Menghini) This promising test is of great value in demonstrating the state of the liver parenchyma. A nega-

tive finding may be misleading because the fibrotic strands of cirrhosis tend to be missed by the needle.

Liver scan (McAffee, Ause, and Wagner, *Arch Intern Med 116*:95, 1965). Radioactive scan may show the presence of a tumour within the liver.

Paracentesis abdominis is essential in all cases of ascites. Infection and malignancy should be excluded. The protein content of the fluid should be estimated on each occasion.

OPERATIONS AND TECHNIQUES

Indications

The systematic study of patients will usually enable them to be placed in one of four main categories: (1) prehepatic portal obstruction with normal liver; (2) early cirrhosis; (3) advanced cirrhosis with good prognosis; or (4) advanced cirrhosis with poor prognosis.

When to operate and what to do for each individual patient are questions which only the surgeon can answer. It is not possible to be dogmatic about the indications for the different operations. There are certain matters, however, on which opinion is becoming crystallized.

1. The most satisfactory way of mitigating haemorrhages in patients with portal hypertension is to reduce the portal pressure by the construction of a large, clean, direct portal-systemic venous anastomosis.

2. Patients with normal or only mildly cirrhotic livers (which give a virtually normal "hepatogram") are excellent operative risks. They should be operated upon as soon as blood loss has been restored and their general condition made satisfactory. Preparation may take up to three weeks after a severe haemorrhage. Only in children whose veins are small should operation be deferred.

3. If the liver is normal in structure, the probability is that the portal vein will be useless for a shunt. Splenectomy and splenorenal anastomosis is then the method of choice. Splenectomy alone should be done only after certain proof has been obtained (by venography) that the splenic vein is unsuitable for a shunt.

4. The best type of shunt mechanically is portacaval anastomosis, of the largest possible size and constructed with a fine everting suture. Such an anastomosis very rarely thromboses. Splenorenal anastomoses, on the other hand, become obliterated by clot in about 30 percent of instances, even in cirrhosis, and smaller veins, e.g., collateral channels and inferior mesenteric, more often still. Most workers are now agreed that makeshift anastomoses using these smaller veins (Linton, *Ann Intern Med 31*:794, 1949) are of little or no value. The superior mesenteric vein may be anastomosed side-to-end to the inferior vena cava or common iliac vein with reasonable prospect of success (Clatworthy *et al.*, *Ann Surg 150*:371, 1959; Marion *et al.*, *Ann Chir Paris 14*:581, 1960; Voorhees *et al.*, *Surgery 54*:559, 1963).

5. A portal-systemic venous anastomosis for ascites in a patient with severe advanced cirrhosis is a hazardous procedure and should never be done until after three months of intensive and carefully supervised medical therapy.

Broadly speaking, therefore, it may be stated that splenorenal anastomosis is the method of choice in cases of primary extrahepatic portal obliteration and portacaval anastomosis in cirrhotic patients who will tolerate diversion of their portal blood. Some other operation is necessary for the cirrhotic patients whose disease is so advanced that a shunt operation would kill them—and then only after failure of medical therapy or on account of persistent haemorrhage.

It has been pointed out by Linton *et al.* (*Surg Gynec Obstet 87*:129, 1948) that a mistake in technique during a portacaval anastomosis may be a matter of life or death to the patient, whereas a comparable error in the course of a splenorenal shunt operation is of far less moment. Furthermore excellent results have been obtained in cases of early cirrhosis by means of a splenorenal anastomosis (Linton *et al.*; Welch, *New Eng J Med 243*:598, 1950; Julian *et al.*, *Arch Surg 63*:373, 1951). A very large and overactive spleen should probably be removed. It is, therefore, a widely held opinion that an end-to-side splenorenal shunt is the best operation for cirrhotic patients who have gross hypersplenism as shown by a white blood cell count which remains persistently less than 2,500 and a platelet count of less than 75,000 per cu. mm. Lesser degrees of hypersplenism resolve after a portacaval shunt. When there is no hypersplenism, the spleen should be preserved. Portacaval anastomosis is then without doubt the operation of choice. Whether splenic arterial ligation should be combined with portacaval anastomosis and whether there is a place for removing the spleen before proceeding

to a portacaval shunt (Rousselot, *JAMA 140*: 282, 1949; Stock, *Ann Roy Coll Surg Eng 10*: 187, 1952) are questions which remain a matter of opinion. That portal vein thrombosis occurs relatively commonly suggests that the preliminary removal of the spleen is not generally to be recommended.

Gastro-Oesophageal Haemorrhage

The seriousness of such haemorrhage in the cirrhotic patient cannot be exaggerated. It is probable that 60 percent of such patients die within a year of their first haemorrhage. Fortunately, however, portal hypertension accounts for only about 3 to 7 percent of all cases of haemorrhage from the upper alimentary canal. (This is true in the British Isles, although there are parts of the world where the disorder is commoner.) The rapidity of decline of the cirrhotic patient is out of proportion to the blood loss; thus effective methods for the control of haemorrhage are essential.

As soon as *sedation and transfusion* is shown to be ineffective, one should proceed to *tamponade* with a Sengstaken-Blakemore tube (Sengstaken and Blakemore, *Ann Surg 131*: 781, 1950). Deflate the balloons after 36 hr. and have all preparations made for operative treatment. Remove the tube after a further 24 hr. if no bleeding occurs. If the bleeding recurs, reinsert the tube, blow up the balloons, and prepare to operate. In most cases the safest and quickest operation to stop haemorrhage will be to under-run the varices, approaching the gullet either (a) through a thoracotomy incision (Boerema, *Arch Chir Neerl 1*:253, 1949; Crile, *Surg Gynec Obstet 96*:573, 1953) or (b) through an abdominal incision (Welch, *New Eng J Med 256*:677, 1956) if there is associated pleural or pulmonary disease or if it is essential to exclude some other cause for haemorrhage (such as a gastric ulcer). The usual reason for the abdominal approach is that the abdomen has already been opened in the erroneous belief that the cause of bleeding was a peptic ulcer.

In the Boerema-Crile operation (Fig. 4), the approach is through the bed of the eighth rib. The lower oesophagus is isolated from the posterior mediastinum (which may be excessively vascular) and opened longitudinally down to the level of the diaphragm. The three main columns of varices are then under-run with a continuous locking stitch of No. 0 or 1 chromic catgut on an atraumatic needle. As the lower end of the oesophagus is approached it will be found that the projecting mucous membrane can be pulled up with each stitch until gastric mucosa appears in the oesophagotomy incision and can itself be under-run for nearly an inch (2.5 cm.). Other lesser veins may then need to be dealt with. It is often disappointing that no specific bleeding point can be demonstrated. Nevertheless, the operation is successful in stopping haemorrhage unless the source of the haemorrhage is within the stomach. To complete the operation, a Ryle's tube is passed and the oesophagus closed in two layers. The mediastinal pleura is sutured and the chest closed with a water-sealed drainage.

In the Welch operation, the approach is through a high midline incision, with splitting or excision of the xiphisternum. The stomach is opened just below the cardia, care being taken to ligature the subserous veins before dividing them, and the incision extended up into the abdominal oesophagus. The columns of varices will be seen running up to the cardia and may be dealt with as in the previous operation. A Ryle's tube is passed before the stomach is closed.

In favour of the thoracic approach is that it is the more certain operation and leaves the abdominal field free for subsequent definitive operative treatment. It is much the easier and is the only procedure possible in the presence of a large hard liver. If it is found that the bleeding is coming from a gastric varix, the diaphragm can be divided and the stomach opened.

Bleeding is suppressed only for a limited time by under-running the varices. The patient should be built up for a shunt operation as soon as possible, probably within six weeks.

Occasionally the only possible method of stopping the haemorrhage is to do a more extensive operation, such as proximal gastric *transection* (Tanner, *Ann Roy Coll Surg Engl 22*:30 1958) or *resection* (Phemister and Humphreys, *Ann Surg 126*:397, 1947). These extensive operations are not advocated in cases of moderate or advanced cirrhosis. The damaged livers will not stand up to the strain. If bleeding is noticed to be coming from the stomach during the course of a Boerema-Crile operation done through a transthoracic approach, the diaphragm and stomach should be incised

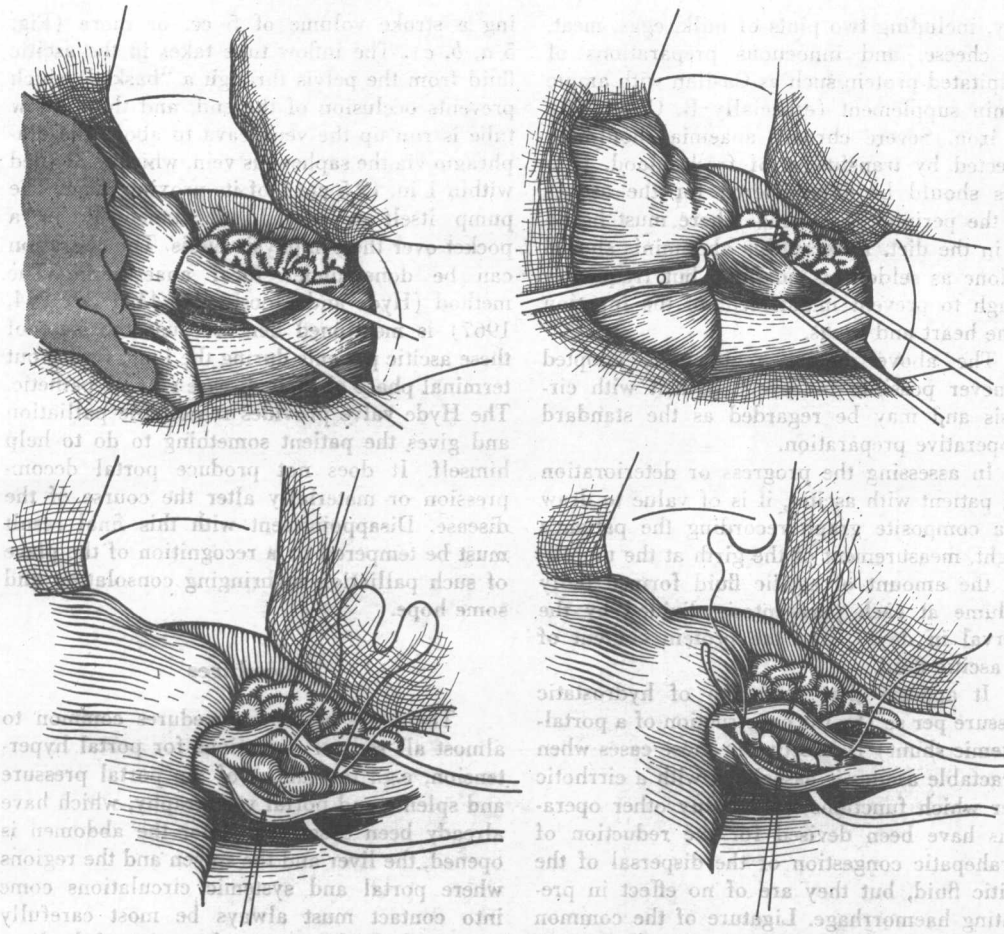

Fig. 4. Boerema-Crile operation. Transthoracic oesophagotomy and the underrunning of oesophageal varices. In B a Tom Smith's clamp has been applied to the lower end of the isolated oesophagus to prevent undue haemorrhage. (From Hunt, A. H. *Portal Hypertension*, 1958. Courtesy of Livingstone.)

and the bleeding point under-run or the stomach transected or resected.

Emergency portacaval anastomosis may be done when the patient's condition is good and liver function reasonable, and when the haemorrhage has been brought rapidly under control by tamponade.

Injection of varices with sclerosants through an oesophagoscope (Crafoord and Frenckner, *Acta Otolaryng* 27:422, 1939) requires patience and persistence on the part of both patient and surgeon. It has its place in the treatment of varices, particularly in patients who have no vein suitable for portal-to-systemic venous anastomosis, in whom other operations have failed, or in cirrhotics whose poor liver function or some other serious illness precludes a major operation.

Ascites

Ascites is probably due to a number of factors, increased hydrostatic pressure (portal hypertension), decreased osmotic pressure (reduction of blood albumen), salt retention, and retention of aldosterone undoubtedly playing significant parts. Obstruction to the flow of hepatic lymph is probably contributory.

The treatment is medical; operation is resorted to only when medical treatment has been shown to have failed. The diet should be rich in animal protein, 100 to 120 grams protein

a day, including two pints of milk, eggs, meat, fish, cheese, and innocuous preparations of precipitated protein such as Casilan with ample vitamin supplement (especially B, C, and K) and iron. Severe chronic anaemia should be corrected by transfusion of fresh blood. Diuretics should be administered for the ascites and the peripheral oedema. There must be no salt in the diet. Paracentesis abdominis should be done as seldom as possible, but frequently enough to prevent impairment of the function of the heart and lungs.

The above dietary regimen is adopted whenever possible for every patient with cirrhosis and may be regarded as the standard preoperative preparation.

In assessing the progress or deterioration of a patient with ascites, it is of value to draw up a composite graph recording the patient's weight, measurement of the girth at the umbilicus, the amount of ascitic fluid formed daily (volume at each paracentesis divided by the interval in days), and the protein content of the ascitic fluid.

It appears that reduction of hydrostatic pressure per se (by the construction of a portal-systemic shunt) is effective in those cases when intractable ascites is associated with a cirrhotic liver which functions well. Many other operations have been devised for the reduction of intrahepatic congestion or the dispersal of the ascitic fluid, but they are of no effect in preventing haemorrhage. Ligature of the common hepatic and splenic arteries (Rienhoff, *Bull Johns Hopkins Hosp 88*:368, 1951) may rarely lead to improvement. Palliative operations such as the Talma Morison omentopexy, the Crosbie-Clunie button, peritoneum-saphenous vein anastomosis and ileoentectropy may occasionally be of temporary benefit (Neumann *et al.*, *Trans Amer Surg Ass 75*:396, 1957; Girling, *Proc Roy Soc Med 52*:252, 1959).

More recently drainage of the ascitic fluid into the circulation has been attracting attention. The Spitz-Holter and Dahl valves, devised for the drainage of hydrocephalus, depend for their effect on being interposed between a fluid reservoir of increased pressure and a vascular receptacle of less or negative pressure. With the peritoneal cavity the drainage must be uphill, and the most promising of such operations depends on the use of a small pump devised by Drs. Hyde and Moosnick in Dr. Ben Eiseman's clinic (available from The Holter Company, Bridgeport, Pennsylvania) and having a stroke volume of 5 cc. or more (Fig. 5 *a, b, c*). The inflow tube takes in the ascitic fluid from the pelvis through a "basket" which prevents occlusion of the end, and the outflow tube is run up the vena cava to above the diaphragm via the saphenous vein, which is divided within 1 in. (2.5 cm.) of its proximal end. The pump itself is placed subcutaneously in a pocket over the symphysis pubis. The operation can be done under local anaesthesia. The method (Hyde and Moosnick, *JAMA 201*:264, 1967) is mentioned here because the state of these ascitic patients during the long, drawn-out terminal phase of their disease is truly pathetic. The Hyde valve provides reasonable palliation and gives the patient something to do to help himself. It does not produce portal decompression or materially alter the course of the disease. Disappointment with this final result must be tempered by a recognition of the place of such palliation in bringing consolation and some hope.

Procedures

There are certain procedures common to almost all radical operations for portal hypertension, e.g., the taking of the portal pressure and splenic and portal venography, which have already been described. After the abdomen is opened, the liver and the spleen and the regions where portal and systemic circulations come into contact must always be most carefully examined. At this stage a fragment of the liver is taken for biopsy, after careful palpation of the whole liver. If there is an area suggestive of primary tumour, the fragment is examined by frozen-section technique.

The operation to be done can usually be determined beforehand. However, it is not until the above preliminary investigations have been completed on the operating table that a final decision can be made on the exact diagnosis and the type and extent of the disease process. The first stage of the operation is done, therefore, through the anterior or abdominal part of the incision, with the patient in the dorsal position, with arms folded across the face and held in place with strapping. This "hornpipe position" (Hunt *et al.*, *Lancet 1*:881, 1956) allows free access to either portal or splenic vein. The cassette tunnel is so arranged under the patient that the film extends to include the nipple and umbilical planes. The lower thorax and abdomen are carefully covered with Vi-