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The Surgical Treatment of Portal Hypertension

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Portal hypertension remains to my mind one of the most obscure modern surgical problems in many of its aspects. Many of the clinical features of this disease are not capable of ready explanation. Why is it that patients with portal hypertension and bleeding from oesophageal varices seldom have bleeding from haemorrhoids? Why is it that patients who have oesophageal bleeding as a result of portal tension, and presumably a high portal pressure within the abdomen, do not at operation, when the contents of their abdomen are fully exposed to atmospheric pressure, present a cyanosed intestine and congestion and dilatation of the mesenteric venous trunks? It is said that the negative pressure within the thorax encourages dilatation of the unsupported oesophageal veins, yet in these cases, though the veins of the retroperitoneal space may be grossly dilated too, and pressures should be equal throughout the valveless portal tree, the mesenteric trunks are normal in appearance usually. Why do patients who develop a satisfactory Medusa head seldom develop bleeding oesophageal varices? These are some of the questions to which no answer can be readily found. One is tempted to believe that there is some local abdominal cause for at least certain of the cases of portal hypertension, and that the locality of greatest portal venous pressure varies from place to place. A hypothesis that would answer these questions reasonably satisfactorily would be the assumption that portal hypertension is due primarily to an extrahepatic arteriovenous fistula or series of such fistulae. No such fistula can usually be demonstrated, if indeed it has ever been satisfactorily demonstrated, but a varying localisation for such a shunt might well determine whether venous dilatation was greatest in the upper left abdomen, encouraging oesophageal varices, in the central part of the upper abdomen, encouraging a Medusa head, or in the lower abdomen, encouraging bleeding piles.

For practical purposes we can recognise three main varieties of portal obstruction. (1) *Thrombosis of the portal vein* or of one of its branches, notably the splenic vein, may be due to compression in the locality of the portal fissure, from over-active closure of the ductus venosus and umbilical vein, or to congenital inflammation of the umbilical vein, or it may be due to pressure of adhesions, cysts or tumours.

(2) *Thrombosis of the splenic vein* is sometimes due to chronic pancreatitis. A similar splenic thrombosis may occur in bilharzial infection of the pancreas; this is probably responsible for Egyptian splenomegaly. Sometimes splenic thrombosis seems to be post-traumatic, and sometimes the thrombosis is secondary to portal hypertension, or spontaneous and primary.

(3) A curious *cavernomatous transformation of the portal vein* has been described by Whipple as a cause of portal obstruction. Its pathology is not yet certain, but it may well be due to a congenital obstruction of the portal vein, perhaps by extension to it of the mechanism which closes the umbilical vein at birth, with a prolific development of local venous collateral circulation between portal tributaries outside the liver and portal branches within the porta hepatis.

Clinical Types of Portal Hypertension

(1) *Acute portal thrombosis* spreading from the portal vein into its tributaries may occur in the course of cirrhosis of the liver, in compression of a vein by a tumour or as a sequel of splenectomy, or as an inflammatory phlebitis

in the course of acute abdominal infection. The signs of this type of portal hypertension are those of infarction of the bowel.

(2) An *episodal form* of portal hypertension may occur at intervals over a period of years, each recurring extension of thrombosis being compensated by fresh collateral circulation until finally the capacity of a compensation is overstrained.

(3) *Chronic portal hypertension* may be due to cirrhosis of the liver or to extrahepatic obstruction. It may be symptomless or give rise to ascites (commonly) or oesophageal haemorrhage.

Clinical Investigations

Physical examination includes palpation of the spleen for splenomegaly, palpation of the liver for enlargement, and inspection of the anus for dilated haemorrhoidal vein. The skin of the anterior abdominal wall is inspected for dilatation of its veins. Opaque meal examination is undertaken to exclude other causes of haematemesis and to attempt to delineate dilated oesophageal veins. The blood picture is fully investigated. The function of the liver is explored by all available clinical methods, including measurement of the blood flow through the liver, if this technique is available.

Catheterisation of the hepatic vein indicates whether a portal hypertension is due to intrahepatic or extrahepatic obstruction. A cardiac catheter is passed by a sytematic vein through the right auricle to the vena cava and is guided radiologically into a hepatic vein to engage in one of its branches. If the pressure falls on engagement it may be assumed that the portal obstruction is extrahepatic. If the pressure is high when the catheter engages it may be assumed that the obstruction is intrahepatic. The pressure in the hepatic vein reflects the pressure in the liver sinusoids. It is extremely useful to have this information before operation; if it is known that the obstruction is intrahepatic, one may proceed directly by the shortest route to establish a portacaval anastomosis.

Liver puncture biopsy is invaluable to determine whether or not cirrhosis of the liver is already present. Excretion urography determines the presence, position and state of the kidneys in case operative intervention should require the use of the renal vein for anastomosis. Oesophagoscopy demonstrates directly the presence of oesophageal varices and also serves to exclude certain oesophageal causes of haematemesis.

Portal venography may be done before operation by the insertion of a needle into the spleen through the chest wall and diaphragm. Under local anaesthesia a 17 S.W.G. needle is introduced through the tenth or eleventh interspace while the patient holds a deep expiration. Thirty ml. of 70 per cent. diadone is injected rapidly, a first film being taken as the injection is completed and a second film three seconds later. The only risk is of subcapsular haematoma and subsequent haemorrhage. The films outline the spleen and splenic vein and those parts of the portal system of veins which are in communication with the splenic. An obstruction in the splenic vein itself, or in the portal vein, can be easily demonstrated.

Treatment

It is the fashion to-day to perform some variety of portacaval anastomosis for the relief of portal hypertension, but it is difficult to estimate how valuable this method of treatment is. Some patients have a few haematemeses in their youth, and then without any treatment at all survive for ten or twenty years without further trouble. Many cases of the episodic kind manifest this prolonged kind of intermission. Nor do we know whether it is the actual anastomosis, or some other element in the operation, which is responsible for post-operative improvement. At operation the duodenum and the pancreas are usually extensively mobilised, and there are numerous intraperitoneal and extraperitoneal manipulations. After operation great new collateral channels develop in the area where the operation has been performed, and huge collateral veins may subsequently run from the region of the pancreas to the retroperitoneal tissues, establishing important anastomoses between portal and caval circulation which are additional to the main anastomosis effected by the hand of the surgeon.

We have had one patient, explored after a long series of frequent and severe haematemeses from oesophageal varices, whose portal, splenic and left gastric veins were all solid with thrombus, so that no anastomosis could be undertaken. In the exploration of these veins the head, body and tail of the pancreas and the spleen were all mobilised, and an important new anastomosis must have developed between the radicles of the splenic vein and the systemic veins of the posterio abdominal walls. In any case this patient, on whom no direct portacaval anastomosis was performed, has remained free from haemorrhages now for three years.

In the early days of the operation, before the indications for it were well established, I explored a patient who had cirrhosis of the liver with ascites and who had been treated many years previously by the Talma-Morison operation. At the time of exploration there were so many enormous veins coursing from the omentum to the abdominal wall that it was felt unwise to divide them and to perform our portacaval anastomosis which might well in fact be narrower in girth than the summated area of anastomosis of all the huge collateral veins. The abdomen was accordingly closed. That patient, whose abdomen had been tapped twice daily for many weeks before operation, did not after it require tapping for several months. Our operation had no doubt established still more collateral veins between the abdominal organs and the parietes. Such collateral veins, developing after any abdominal operation, are not well established for a few weeks probably, and it has been our experience that in a proportion of patients after portacaval anastomosis there has been haematemesis in the immediate post-operative period, presumably from silting up or clotting at the point of anastomosis, yet these patients have gone on for long periods subsequently with no recurrence of the haematemesis. Presumably they are now running along on collateral channels which developed a few weeks after the operation. This point, whether it is the laparotomy or the actual anastomosis that makes for improvement, could be settled only by treating two parallel and unselective series of patients differently, alternating in successive cases simple laparotomy with the performance of a portacaval shunt, and comparing the long term results. Most physicians, however, are fully convinced of the efficacy of the portacaval shunt, and I do not think that any of them would be inclined to sanction this kind of experiment. It is humbling to reflect that portacaval shunts have been done now for only about ten years. It took us twenty or thirty years to find out that splenectomy was really of no value in the treatment of "Banti's disease." Perhaps it will take us as long properly to assess the value of portacaval anastomosis. There is one important positive indication that portacaval anastomosis is in fact of real benefit. The completion of the anastomosis is sometimes followed by a fall of 200 mm. in portal pressure; this is probably a greater fall than might be expected merely from the effects of operation and anaesthesia, though it should be remembered that in all operations portal flow falls progressively during the operative procedure. Only in a very few cases is there a substantial decrease in the oesophageal varicosities after operation and reduction in the size of the spleen.

Splenectomy is not predictable in its results and bleeding frequently returns after it, though it has had many successes, with postponement of bleeding for twenty years before recurrence; its disadvantage is that it abolishes the adventitious circulation which has usually developed naturally between the splenic and pancreatic veins on the one hand and the adrenal and renal veins on the other. It is legitimate to perform splenectomy if the splenic vein only is obstructed.

The injection of oesophageal varices is quite unsatisfactory; as Learmonth has shown, it is analogous to injecting varicose veins in the leg in the presence of deep vein thrombosis. Ligation of the left gastric vein is quite ineffective.

The Talma-Morison operation is perhaps valuable in mild cases of ascites due to a moderate degree only of cirrhosis of the liver; Grey Turner recorded a thirty-six years survival of a patient who had hobnail liver and repeated ascites. I have been impressed with the flow which may occur in collateral veins after this operation. The development of a Medusa head after this operation is a good sign, indicating a successful artificial anastomosis between the omental, visceral and hepatic veins and the systemic veins of the anterior abdominal wall. If a Medusa head develops, a simple estimation can be made of the rate of blood flow into the veins of the abdominal wall from the portal circulation; by computing the arithmetical sum of the diameters of visible veins it can sometimes be deduced that there is a flow through them of nearly two litres per minute.

Portacaval shunt is probably the most desirable treatment of obstruction within the liver or of the hepatic fissure. It has a mortality of 15 to 20 per cent. A complete Eck fistula in animals is not survived for long, but the effect on a patient with portal hypertension cannot be deduced from animal experiment. Splenorenal anastomosis is technically an easier alternative than a direct anastomosis between portal vein and vena cava, but end-to-end anastomosis entails nephrectomy, and end-to-side gives a less dramatic fall in portal pressure than does portacaval shunt.

Resection of the proximal part of the stomach and the lower end of the oesophagus, together with the varices in that locality, was widely performed by Phemister for repeated serious

haematemesis, but the young scar tissue at the anastomosis, of primitive mesoderm exposed on one side to a higher venous pressure, is transformed soon into a venous plexus which quickly furnishes a varicose anastomosis again. Haemorrhage usually recurs soon after the performance of this operation.

On the assumption that portal cirrhosis is due to the presence of arteriovenous fistulas within the liver, it has been advised that ligation be done of the hepatic artery. It is also said that the hepatic artery and portal vein compete for sinusoid space in the liver and that to ligate the hepatic artery leaves freer drainage for the portal branches. Hepatic artery ligation has unquestionably been fatal sometimes when done accidentally in cholecystectomy, in cases with poorly functioning livers, but there have been many survivors and a few ameliorations recorded after hepatic artery ligation in portal hypertension. On the whole it is felt that this operation is discredited.

Portacaval anastomosis is usually undertaken for the treatment of recurrent haemorrhage from oesophageal varices. It is contraindicated if there is gross cirrhosis of the liver. Most authorities do not now advise a shunt for the treatment of cirrhosis of the liver with ascites. The mortality is very high and not much improvement is to be expected. Blakemore advises that a shunt be done if the serum albumen can be raised above 3 per cent. and sodium retention avoided.

For the emergency treatment of bleeding from oesophageal varices during the actual bleeding, a cuffed gastric tube is of advantage. The tube carries two cuffs, one of which is passed just into the stomach and inflated, while the other lies in the lower oesophagus, inflated also to compress the bleeding oesophageal veins. Attempts have been made to interrupt the anastomosis between the portal and the systemic circulations by the division of the left gastric vein, the splenic vein and the left gastro-epiploic vein. This operation is not very successful, for new venous anastomoses open up and bleeding usually recurs. Norman Tanner advises rather "trans-gastric porto-azygos disconnection"—transection and resuture of the stomach, with division of the vasa brevia. The disadvantage of this operation is the same as that of oesophago-gastrectomy; the stomach wound heals by young mesoderm which is easily transformed into a vast venous plexus, renewing the gastro-oesophageal varices in exaggerated form after a purely temporary improvement.

Operative Procedure

When the abdomen is opened, the liver is explored for cirrhosis, the portal vein is inspected and examined for block, and the pressures in the portal, splenic, coronary and superior mesenteric veins are measured. Venography of the portal system is performed on the table with the abdomen opened, if it has not been done before operation. The portal pressure is regarded as abnormal if it is more than 140 mm. of water, though this pressure is sometimes normally exceeded in certain parts of the portal system—the superior haemorrhoidal vein in defecation, for example. Portal pressure is higher after meals. The pressure in the portal vein usually varies in normal subjects between $\pm$ 65 mm. of water; the normal pressure in the hepatic vein is from 0 to 10 mm. of water. In portal hypertension the venous pressure may rise in the portal vein and its tributaries to 500 mm. of water.

There may be considerable variation in the anatomy of the tributaries of the splenic vein. The splenic vein itself is often buried in and obscured by the pancreas, and sometimes it is replaced by a large inferior pancreatic vein running along the lower border of the pancreas. Very often short gastric veins drain directly into the spleen. The left gastro-epiploic vein usually passes into a splenic tributary of the splenic vein. The left gastric vein in 50 per cent. of cases enters the superior aspect of the junction of the splenic and superior mesenteric, in 20 per cent. into the portal vein and in 12 per cent. into the splenic vein; sometimes it disappears into the portal fissure separately from the other veins draining into the liver. Usually the upper 5 cm. of the portal vein has no branches.

Splenectomy is performed if thrombosis is restricted to the splenic vein. After this operation there is a risk of extension of thrombosis, so the ligatures should be applied to the vessels directly and gently; clamps should not be used and no tension should be exerted on the splenic vein. The splenic vessels are tied as close as possible to the spleen so that any post-operative thrombosis in the splenic vein will be as far as possible from the main portal trunk.

Portacaval shunt is the procedure of choice in obstructions within the liver or of the portal fissure. Provided a clear pre-operative diagnosis has been made of intrahepatic obstruction, one can proceed directly to the operation with the patient lying half over on his left side, by an abdominothoracic approach. The ninth rib

is excised from its angle to the costo-chondral junction, the diaphragm is incised in the same line, the right triangular ligament is divided, and the liver is dislocated upwards into the chest. The portal vein is inspected, its upper end is ligated and a clamp is applied as distally as possible below the liver. The vena cava is inspected behind the portal vein and a segment of it is controlled by two clamps. End-to-side anastomosis is then performed between the portal vein and the vena cava. If the portal vein and vena cava are very far apart, a vein graft may be inserted, as Theron has advised, to join them, giving the effect of a lateral anastomosis. The join may be regarded as effective if it reduces the pressure by 200 mm.

Spleno-renal anastomosis is technically a rather more simple procedure, but it is usually followed by a less decrease in portal pressure. The spleen is mobilised, the splenic artery is tied close to the spleen and divided. The tributaries of the splenic vein in the pedicle are dissected and the longest of them is selected for anastomosis. The spleen is removed, the splenic vein is controlled by a bulldog clamp and the lumen of the vein is washed with heparin. The left kidney is then mobilised, and first its artery and then its vein are controlled by bulldog clamps. An end-to-side anastomosis is then effected between splenic and renal vein. If it appears that the veins will not lie nicely end to side, nephrectomy is performed and an end-to-end anastomosis made, with the veins lying in a wide comfortable curve. Care is taken to preserve the adrenal vein and the testicular or ovarian veins, for these are important anastomotic channels. Alternative anastomoses sometimes suggest themselves if thrombosis is extensive in the splenico-portal area—mesenteric vein to ovarian, for example, or left gastric to renal, but these afford so narrow an anastomosis that no great benefit can be expected, and, in cases where they might be applied, further thrombotic episodes are likely to occur in the portal field in spite of them.