SURGICAL TREATMENT OF PORTAL HYPERTENSION

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When the flow of portal blood into or through the liver is gradually obstructed the hydrostatic pressure in the portal system of veins rises, the spleen enlarges and a collateral venous circulation develops to return the portal blood to the general circulation. This syndrome is given the name of Portal Hypertension. The site of the obstructing lesion may be inside or outside the liver. Intrahepatic obstruction is almost invariably caused by cirrhosis of the liver, the regeneration of the parenchyma and the growth of fibrous tissue which constitute the "healing" phase of this condition causing a gross distortion of the vascular tree and greatly increasing the resistance to portal blood flow through it. Extra-hepatic obstructions may be situated in the portal vein itself, in which case the hypertension and other changes affect the whole portal bed but the liver is normal, or in the splenic vein, when the changes will be localized to the splenic circulation and its connections with the greater curvature of the stomach. Obstruction of the superior mesenteric vein does not cause portal hypertension. The effect depends to a large extent upon the situation of the obstruction and may either be a massive intestinal infarction, which is usually rapidly fatal, or a transient melaena with few other symptoms or signs.

Clinical Features

The clinical features of portal hypertension are:

(1) Splenomegaly, often with a feeling of weight or recurring episodes of sharp pain in the left upper quadrant.

(2) Changes in the circulating blood picture (granulopenia and thrombocytopenia), which appear to be associated with the presence of an enlarged and over-active spleen and together comprise the syndrome of Hypersplenism.

(3) Episodes of alimentary bleeding which may be slight in amount and detected only as 'occult blood' in the faeces or may be a profuse haematemesis. This blood loss is the principal cause of the anaemia that is usually present.

(4) The presence of abnormal portal systemic communications. These may be seen on the anterior abdominal wall ('caput Medusae') or demonstrated as 'oesophageal varices' by oesophagoscopy or radiography after barium swallow.

(5) In more than 80% of cases the obstruction is within the liver, secondary to chronic hepatic disease, and symptoms and signs of such a condition are also evident.

Indications for Surgical Treatment

The prime indication for surgical treatment in portal hypertension is the occurrence of haematemesis or melaena. Operation may also be called for when an enlarged spleen is causing pain and discomfort as well as persistent granulopenia and thrombocytopenia or to prevent the onset of severe bleeding when oesophageal varices can be demonstrated and other features of the syndrome are present. In assessing the suitability of a patient for operation it is important to remember the various ways in which the condition may present. Most cases will be found to conform to one of three broad groups, each of which has a different prognosis:

(1) The block in the portal vein is extra-hepatic. The patient is often a young person and is usually in other respects normal. The progress without operation is characterized by repeated haematemesis from which recovery is generally rapid. Death may occur from an uncontrollable haemorrhage or from intestinal infarction. The outlook after operation for these patients is good.

(2) The block is intra-hepatic but there are few if any symptoms of hepatic disease, and the results of hepatic function tests are almost within normal limits. The liver is moderately enlarged and is rubbery and smooth to palpation and the spleen is often very large. There is no previous history of any serious hepatic disease. This group has been called Hepatolienal fibrosis and it constitutes the greater proportion of cases of portal hypertension in Great Britain. Operation should be done to prevent further haematemesis because with each episode of severe loss of blood further damage to the liver occurs and its powers of recovery are reduced. In many instances the hepatic condition appears to be almost static so that the prognosis
of operation depends upon the degree of hepatic damage already present and the successful prevention of further bleeding.

(3) The block is intra-hepatic. There is a history of severe or protracted hepatic disease a few years previously from which there has been a measure of clinical recovery, of exposure to hepatoxins or of prolonged overindulgence in alcoholic beverages. The liver is hard and irregular and the results of function tests show grave hepatic impairment. There may be such clinical signs of hepatic failure as weight-loss, jaundice, fluid retention or mental deterioration. The prognosis in these patients is poor and from the very nature of the condition cannot be much improved by operation. Indeed, the mortality after operations on patients with cirrhosis and ascites is so high that the presence of persistent fluid retention should be considered a contra-indication to surgery.

**Treatment**

From what has been said it is apparent that the factors which largely determine the prognosis in most cases of portal hypertension are the functional and pathological condition of the liver and its response to suitable treatment. Hence the basis of all treatment for this condition must be medical. The form this treatment may take is described elsewhere in this number. In most cases anaemia is also present and is of an-iron-deficiency type. It responds to iron in full therapeutic doses. In some patients with chronic hepatic disease absorption of iron from the alimentary tract is defective and the response to iron by mouth is poor. However, there is almost invariably improvement if the iron is given parenterally.

The essential preliminary to the treatment of active bleeding is a correct diagnosis. The presence of an enlarged spleen and a palpable cirrhotic liver is strong evidence in favour of portal hypertension, but when, because of a history suggestive of ulcer dyspepsia, bleeding from a peptic ulcer cannot be excluded, there should be no hesitation in passing an oesophagoscope to see whether the blood is coming from the stomach or the oesophagus. If the bleeding is seen to be from oesophageal varices treatment consists of (1) early restoration by transfusion of the lost blood; (2) sufficient sedative to take the edge off the patient’s anxiety but not so much as to make him unco-operative. Small doses of omnopon intravenously probably serve this purpose best; (3) control of any bleeding tendency secondary to the hepatic disease by intravenous injections of vitamin K₃-oxide; and (4) if these measures alone fail to check the bleeding, control of the bleeding point either by balloon tamponade (Sengstaken and Blakemore, 1950) or by oesophagotomy and suture of the enlarged veins (Crile, 1953). The virtue of this latter procedure is uncertain because further operation is always necessary and because experience has shown that bleeding from oesophageal varices frequently ceases after an operation such as exploratory laparotomy has been performed on a mistaken diagnosis of bleeding peptic ulcer.

Definitive surgical treatment is usually undertaken when the patient’s condition has been sufficiently restored by transfusion, diet, vitamins and iron to make him a reasonable operation risk. In patients with cirrhosis this process often takes 4 weeks or more. Operation may then take one of three forms:

1. Splenectomy alone. In patients with proven obstruction in the splenic vein splenectomy provides complete relief. The only satisfactory way of demonstrating such a lesion is by preoperative trans-splenic portal phlebography. Splenectomy is also a valuable operation when there are present splenomegaly and such severe granulopenia that the effects of what should be minor infections are magnified and prolonged and when it is not possible to demonstrate any collateral circulation in the cardio-oesophageal area. On the other hand, in patients who have already had episodes of haematemesis or melaena splenectomy is followed by recurrent bleeding so frequently that it cannot be considered as anything more than a palliative procedure.

2. Operations designed to divert the portal blood away from the varix-bearing area by means of an anastomosis between a large portal and a large systemic vein. The veins usually chosen are the portal vein and the inferior vena cava, and the splenic vein (after splenectomy) and the left renal vein. In order to prevent drainage of hepatic arterial blood back through the proximal end of the portal vein it is advisable to ligate the portal vein close to the porta hepatis and to anastomose its distal end with the side of the inferior vena cava. This operation diverts all the portal blood from the liver as well as from the varix-bearing area and for this reason there is the possibility that it may be more liable to be followed by neurological complications (Sherlock et al, 1954). When the spleen is so large or so active that splenectomy is essential it is preferable to perform an anastomosis between the end of the splenic vein and the side of the left renal vein. The blood supply to the kidney must be controlled by an arterial clamp while the suturing is being done. In our experience periods of occlusion up to 45 minutes have not resulted in any damage to renal function. Lieno-renal anastomosis is followed by adequate diversion of portal blood from the cardio-oesophageal area and as it also removes the enlarged spleen it is, in our opinion,
the operation of choice in cases considered suitable for portal-systemic venous anastomosis.

3. Excision of the varix-bearing area by removal of the segment of stomach drained by the left gastric and short gastric veins and the lowest 3 to 5 cm. of oesophagus followed by oesophago-gastrostomy, end-to-end or end-to-side. This procedure removes the site from which the bleeding occurs. The operation is a severe one and is liable to be followed by such unpleasant features as regurgitation of food and dysphagia. In most instances these complications have been temporary only and have been more than offset by the freedom from further haemorrhage. Limited oesophago-gastrectomy is particularly useful for the treatment of patients with recurrent bleeding after other operations and in children, but is poorly tolerated when the history or the results of hepatic function tests indicate a seriously diseased liver.

**Technique**

Certain points in the management of patients with portal hypertension during the operation period may be mentioned. At least 1 litre of matched blood must always be available and an intravenous infusion is set up before the operation is begun. A stomach tube is passed on the morning of operation and is left in place. Nothing interferes with the smooth course of operations on the spleen more than a stomach distended with air or anaesthetic gases. An operating table which can be tilted laterally is desirable for thereby the exposure can be greatly improved. The incision must be one which will permit examination of the liver, portal vein, stomach and spleen. Our own preference is to begin with a transverse upper abdominal incision. This is sufficient for exploration and a small extension over the left costal margin, not necessarily into the pleural cavity, gives a good exposure for splenectomy and lienorenal anastomosis. If the operation of choice proves to be portacaval anastomosis the patient is tilted to the left and the incision extended along the right ninth interspace. If oesophago-gastrectomy is chosen, the patient is turned on to his right side and the chest widely opened by extending the incision to the left through the bed of the ninth rib. When the pleural cavity has been opened it should always be drained for 48 hours after operation.

**Complications of Operation**

The particular danger of operation in portal hypertension is hepatic failure. The worse the condition of the liver before operation the more likely is hepatic failure to occur afterwards. If alimentary bleeding is constituting an additional threat to life in a patient with serious hepatic de-

compensation the least severe operation which is suitable should be performed. The only other precautions which can be taken are to choose an anaesthetic which allows a high concentration of oxygen to be given at the same time and to maintain the circulating blood volume by transfusion during operation. If the sign of hepatic failure is retention of fluid it may be controlled by a diet containing not more than 0.5 g. of sodium per day, supplemented if necessary and provided laboratory control is possible, by the administration of mercurial diuretics. The prognosis of hepatic coma after operation is bad. The basis of treatment is early recognition and continuous intravenous infusion of 5 per cent. glucose in water. The serum electrolytes are liable to rapid change and laboratory estimations should be done repeatedly so that the appropriate corrective solutions may be administered. Many other methods of treatment have been recommended but there is not yet any good evidence that they are more efficacious.

**Prognosis After Operation**

As a result of better selection of cases for operation, the choice of suitable anaesthetic agents, the proper use of blood transfusion and improvement in surgical technique, the actual operative mortality in clinics which take a particular interest in these formidable cases is now in the region of 10 per cent. Most of these deaths are due to acute hepatic failure. In a follow-up of 64 cases of portal hypertension with hepatic cirrhosis (Macpherson, Owen and Innes, 1956) it was found that of those who had been operated on more than 1 year ago there were surviving 78 per cent., more than 3 years 58 per cent., more than 5 years 40 per cent. and more than 7 years 18 per cent. The principal causes of death were hepatic failure and recurrent bleeding, frequently in combination. This survey included several patients who would not now be considered suitable subjects for operation. Consequently there is some reason to believe that when the surgical treatment of portal hypertension is reviewed in another 10 years time the results will be better. However, it must be recognized that survival in most cases of portal hypertension is determined not so much by improvements in surgical technique as by the functional capacity of the patient's liver and its power to respond to stress. Realization of this is not in itself a reason for the adoption of a defeatist attitude by physicians, but should be an incentive to physician and surgeon acting together to exercise a fine judgment in the choice both of the time for surgical intervention and of the operation most suited to the individual patient.

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by tremor. The drug must be given daily for many months. Tolerance to the drug varies. Some patients can be given doses up to 300 mg. per day; others will develop toxic reactions and prolonged therapy is not possible. Versene, although effective, has the disadvantage of requiring intravenous administration. Intramuscular versene is painful and not very effective; oral versene is ineffective. Penicillamine appears effective by mouth and, provided it can be produced cheaply, will undoubtedly prove of great value. All patients should be given a high protein diet, since the resultant increased amino acid excretion will itself increase the urinary excretion of copper. Ideally, the total copper content of the diet should be kept as low as possible. A diet containing less than 1 mg. of copper per day is generally unpalatable, and it is often simpler to compromise and merely exclude those articles from the diet which have a high copper content.

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