Current Practice

DISEASE OF THE DIGESTIVE SYSTEM

Management of Bleeding Oesophageal Varices

ROGER WILLIAMS,* M.D., M.R.C.P. ; JOHN DAWSON,† M.S., F.R.C.S.

Patients with bleeding oesophageal varices die from exsanguination or liver failure or a combination of these factors. The development of liver failure is inevitable if bleeding is allowed to continue, and the first aim of treatment must be to stop haemorrhage by the fastest and simplest method available. Unfortunately bleeding tends to recur unless the portal venous pressure can be permanently lowered ; but in practice the timing and choice of correct operation are difficult. Throughout the care of these patients medical and surgical therapy are closely linked, and the importance of combined consultation of physician and surgeon with formulation of a definite plan for each patient cannot be overstressed.

Initial Assessment

The differential diagnosis of the various types of portal hypertension and cirrhosis is beyond the scope of this article. In patients who are bleeding from varices it is important to determine as soon as possible whether the cause is:

- (1) extrahepatic portal hypertension, due to thrombosis of the splenic or portal vein ;
- or (2) intrahepatic portal hypertension due to cirrhosis—by far the commoner cause in Britain.

The chances of successfully controlling the acute bleed and the prospect of long-term survival are higher in the first group, though the types of surgery possible are more limited since a portacaval anastomosis with a few exceptions cannot be performed.

It is also important to be certain that the bleeding is coming from varices. Cirrhotic patients have an increased incidence of peptic ulcer, and in various series published from America more than half the cirrhotic patients admitted with bleeding have either an alcoholic gastritis or a chronic peptic ulcer. In Britain alcoholism is a less common cause of cirrhosis, so probably more patients with cirrhosis who bleed do so from varices. Though a barium examination is often helpful direct visualization of a bleeding point at oesophagoscopy is the only certain way of making the diagnosis. The procedure is unpleasant for the patient and bleeding may have stopped by the time of examination, so that our practice is to perform oesophagoscopy or gastroscopy only when there is clinical doubt as to the site of bleeding. The control of bleeding by a Sengstaken tube is a useful confirmatory test.

Biochemical tests such as the bromsulphalein (B.S.P.) retention have also been suggested as aids to differential diagnosis. However, Enquist *et al.*¹ showed in a large series that B.S.P. excretion can be impaired in patients with acute gastrointestinal haemorrhage not due to liver disease—presumably as a result of a decreased liver blood flow. The finding of a raised blood

ammonia level points to underlying liver disease or an extensive collateral circulation or both, but again does not prove that it is the varices which are bleeding.

Treatment of Acute Bleeding

Haemorrhage causes decreased hepatic blood flow, and the resulting liver cell anoxia together with the absorbed load of nitrogenous breakdown products from the blood draining the bowel frequently produces hepatic encephalopathy.

The principles of treatment are to restore the blood volume, to prevent breakdown of blood within the bowel, and to arrest the haemorrhage.

Restoration of Blood Volume

Adequate blood transfusion is essential to prevent further impairment of liver cell function. If possible fresh blood should be used for this will make up, albeit temporarily, the deficiency of platelets and clotting factors usually present in cirrhosis. Vitamin K₁ (10 mg. intramuscularly daily) should also be given routinely.

Prevention of Protein Breakdown

The bowel lumen should be emptied by purging with magnesium sulphate orally and by enemata until normal bowel contents are obtained. Neomycin (1 g. four-hourly) should be given by mouth to decrease the bacterial breakdown of blood in the gut. Sedation is best avoided, but if the patient is greatly distressed phenobarbitone (60–200 mg. intramuscularly), which is largely excreted by the kidney, or chlorpromazine (50–100 mg. intramuscularly) is probably the least harmful. Occasional patients—usually those with extrahepatic obstruction—will stop bleeding spontaneously or after blood transfusion.

Arrest of Haemorrhage

Vasopressin lowers portal blood flow and pressure as a result of vasoconstriction of the splanchnic arterioles. *Vasopressin* is given as an intravenous infusion of 20 units in 100 ml. of 5% dextrose over 20 minutes. The effect lasts up to an hour. Initially there is also systemic arteriolar constriction with transient pallor of the skin and a rise in arterial blood pressure. Intestinal colic is another immediate effect, which may be useful in emptying the bowel of blood. The dose can be repeated after four hours, but its efficacy tends to decrease with successive doses. The use of *vasopressin* is contraindicated in patients with cardiac ischaemia, and another disadvantage is that the reduced hepatic blood flow may further impair the circulation to the cirrhotic nodules. The synthetic derivative phenylalanine²-

* Physician and Medical Tutor, King's College Hospital and Medical School, London.

† Surgeon and Surgical Tutor, King's College Hospital and Medical School, London.

lysine⁸-vasopressin (Octapressin) has less effect on the systemic circulation, but its advantages over vasopressin have not been established in a controlled trial.²

Balloon Tamponade.—The Sengstaken tube with its gastric and oesophageal balloons was introduced in 1950, and much has been written about its use. Traction is advised by some to keep the gastric balloon in position in the fundus of the stomach, and this can be judged by screening the patient during inflation of the balloons. The tube is undoubtedly effective but is unpleasant for the patient, and there is also a risk of pulmonary complications and oesophageal ulceration.³ Repeated pharyngeal suction to remove saliva and other secretions is required, and on no account should the balloons be left inflated for more than 24 to 36 hours. Some workers find that a single gastric balloon is sufficient, and suggest that the pressure of the balloon against the diaphragmatic crura stops the blood flow from the gastric veins into the oesophageal varices.^{4, 5}

Gastric Hypothermia.—Hypothermia is produced by the circulation of an ethanol-water mixture through a gastric balloon at a temperature of 0° C. Cooling is maintained for periods of 24 to 72 hours. There is no change in portal pressure, and bleeding probably stops as a result of local vasoconstriction of small vessels.⁶ Cooling is tolerated better than the Sengstaken tube but shares the high incidence of pulmonary complications.

Published results show that any of these three techniques will stop bleeding in 70–80% of patients. Unfortunately their effect tends to be temporary, and bleeding recurs particularly in those patients with severe hepatocellular failure. Pitressin therapy is the simplest and least distressing to the patient, and as illustrated in the figure our practice is to use it once in all patients unless there are specific contra-indications. If bleeding stops and does not recur within the subsequent weeks the patient can be assessed for definitive surgery.

If bleeding recurs it should be controlled with the Sengstaken tube (or gastric hypothermia if available) and the patient prepared for surgical intervention. When to operate in the individual patient is a difficult decision. There is no doubt that with each further bleed and subsequent resuscitation the general

should be taken not to open the diaphragm or to disturb the oesophageal hiatus, as this may allow ascitic fluid to accumulate in the pleural space and cause much difficulty in the post-operative period. Once the oesophagus is mobilized a soft clamp is placed across the oesophago-gastric junction to lower the pressure within the varices. These may then be dealt with in either of two ways:

(1) *The Boerema-Crile Operation.*^{7, 8}—The lower oesophageal lumen is entered via a longitudinal incision. The columns of varices are then under-run with a continuous atraumatic catgut suture. The oesophagus is then closed in layers with catgut and a chest drain inserted.

(2) *The Milnes Walker Operation.*⁹—The muscle of the lower oesophagus is divided longitudinally down to the mucosa. The mucosal tube together with the varices which lie in the submucosa is then dissected free. This tube is then divided transversely as low down as possible and resutured with a continuous catgut suture. This suture and the subsequent healing process in the mucosal layer occlude the varices. The two suture lines lie at right angles to one another so that the closure is potentially safer than in the Boerema-Crile operation. The oesophageal clamp should be released before the oesophagus is closed, to confirm that the bleeding has been stopped whichever operation is done.

After both these operations a gastrostomy is useful for feeding. No oral fluid is given for about five days, at which time a gastrograffin swallow will confirm that the oesophagotomy closure is sound. The avoidance of a nasogastric tube allows the patient to co-operate with the postoperative chest physiotherapy more readily. It is usual for the patient to have a low-grade fever for some days, which may be due to thrombosis of the varices.

Other Methods.—An emergency portacaval shunt is occasionally worth considering in the patient with excellent liver function in whom the portal vein is known to be patent.¹⁰ Even in such cases the mortality rate will be higher than that for an elective shunt, and a more prudent course is to control bleeding by a simple ligation and do the shunt at a later date.

Another technique which has been employed is cannulation of the thoracic duct. The duct is exposed under local anaesthesia through an incision above the medial end of the left clavicle. Drainage of the lymph reduces the portal pressure. Variceal haemorrhage stopped in 8 out of 13 patients treated in this way by Dumont and Witte.¹¹ However, the cannula tends to get blocked and bleeding usually recurs.

Prevention of Further Bleeding

Though a few patients do not re-bleed for a number of years after a ligation of varices the majority do, and all should be assessed and considered for definitive surgery. The operation which confers the best long-term protection against further haemorrhage is the portacaval shunt. Ideally the patient should be: (1) less than 50 years old; (2) not jaundiced; (3) have a serum albumin of greater than 3 g./100 ml.; (4) show no neuropsychiatric disturbance even during the haemorrhage. Relatively few patients fulfil these criteria, and many surgeons accept less favourable patients knowing that the mortality and morbidity of the operation will be increased. It is important to remember that signs of liver failure developing during or after a bleed may improve once haemorrhage has been controlled. Even the portal pressure can fall with medical treatment, particularly when cirrhosis is accompanied by marked fatty change as in the alcoholic.¹²

Portal Venography.—The technique of percutaneous trans-splenic venography is relatively simple and the portal pressure can be measured at the same time, but it cannot be performed if the prothrombin time is more than two seconds prolonged over normal. Occasionally all the contrast medium is diverted into collateral channels and the portal vein does not fill even though patent. This happened in 6.5% of a series of 904

condition and liver function of the patient deteriorates and the chances of a successful outcome get less. Operation should therefore be done early rather than late, and every patient should be considered—except for those with deep jaundice, ascites, or coma, in whom bleeding is but one manifestation of terminal liver failure (Figure).

Operations to Stop Bleeding

Surgical ligation of varices is probably the simplest and most effective method available. The left chest is opened through the bed of the eighth rib and the inferior pulmonary ligament dissected upwards to expose the pleura overlying the lower oesophagus. The large collateral veins around the lower oesophagus are best suture-ligated to prevent troublesome bleeding during mobilization of the oesophagus. If the patient has ascites care

splenic venograms reported by Burchell *et al.*,¹³ though in the majority of these anatomical patency was subsequently proved at operation. In such cases or in patients in whom splenic venography is contraindicated the technique of arterio-portography is of value. The coeliac axis or superior mesenteric artery is selectively catheterized and serial films taken of the arterial and venous phases after injection of contrast medium.¹⁴ Superior mesenteric arterio-portography is particularly useful in patients who have had a splenectomy. Some surgeons prefer to do venography and pressure measurement at the time of operation. However, this prolongs operating time, the pressure is lower than that recorded pre-operatively, and it is preferable to have the information beforehand.

Shunt and Transection Operations

A portacaval shunt is usually done by anastomosing the end of the portal vein to the side of the inferior vena cava. A side-to-side anastomosis may be technically more difficult and may be associated with a higher incidence of post-shunt encephalopathy.¹⁵ It should be noted that all other shunts—for example, splenorenal and mesenteric-caval—are in effect side-to-side anastomoses, so that the whole of the portal blood is not diverted from the liver.

In patients with extrahepatic portal hypertension, either a splenorenal or a mesenteric-caval shunt¹⁶ may be the only type of decompression possible. The thrombosis rate after spleno-renal shunt, even if the vein is more than 1 cm. in diameter, is higher than after a portacaval shunt. A spleno-renal shunt may also be done in cirrhotic patients who have secondary thrombosis of the portal vein or in the few patients in whom secondary hypersplenism is the major problem.

In cirrhotic patients who are unfit for shunt surgery, or in children with extrahepatic block (in whom the splenic vein is too small for a splenorenal shunt) some degree of protection against further haemorrhage may be conferred by a transection operation—either the Milnes Walker type already described, or the more extensive porta-azygos disconnection described by Tanner.¹⁷ In this procedure all the external vascular connexions of the lower 5 cm. of the oesophagus and the upper 5 cm. of the stomach are divided. The upper stomach is then transected and re-anastomosed. This is often a difficult and prolonged operation, and a thoraco-abdominal approach may be necessary. Resection of the lower oesophagus to remove varices with oesophagogastric anastomosis has been used with success, especially in patients with an extrahepatic block.⁸ Like transection procedures these do not affect the underlying portal hypertension. The injection of sclerosant solution into the varices¹⁸ or into the submucosa around the varices has also been employed,⁸ but there are no controlled trials of its use.

Results

There is no doubt that a successful shunt can prevent further bleeding. Grace and his colleagues,¹⁹ who have recently analysed 154 papers in the world literature, found that the incidence of recurrent bleeding in 1,020 patients following a shunt operation was 6.7% (portacaval anastomosis 2.8%, splenorenal 19%). The overall incidence of hepatic encephalopathy was 19.0% and the incidence was higher after a portacaval anastomosis than after a splenorenal shunt. Clinical manifestations of hepatic encephalopathy varied from mild impairment of intellect to disabling organic neurological syndromes. The development of encephalopathy must to some extent reflect the progressive nature of the underlying liver disease, but there seems little doubt that the incidence is higher after shunt operations. Indeed, this is the price that some patients pay for the prevention of further haemorrhage. The symptoms in many

of these patients, however, are relatively easily controlled by dietary protein restriction and neomycin therapy.

The reported figures for long-term survival vary, but in three large series from America between 50 and 65% of patients were alive five years after a portacaval anastomosis.²⁰ In a recent analysis of 242 cases Hunt⁸ found a five-year survival of 48% and a ten-year survival of 27%.

Although the prognosis in selected groups of good risk patients treated by shunt operations is good, the overall results reported for bleeding varices are less encouraging. Hislop and colleagues²¹ found that 34 of 63 patients admitted with their first bleed from varices died, and in Sherlock's²² series 40 of 120 cirrhotic patients died within a year of their first haemorrhage. Indeed, according to Grace *et al.*¹⁹ there is little evidence at present to show that shunted patients survive longer than good risk cirrhotic patients who have bled but not had a shunt performed. These authors also note that the most enthusiastic claims for shunt surgery were made by those workers with poorly controlled series.

Very often patients have been considered for transection operations only when they have been deemed unsuitable for shunt surgery, and there are no controlled studies available. No further bleeding occurred in 14 of 25 cirrhotic patients with a porta-azygos disconnection reported by Tanner,¹⁷ and in 13 of 25 patients with extrahepatic portal vein obstruction treated by oesophageal transection by Milnes Walker.⁹ The greater risk of rebleeding may be compensated for by a lower incidence of serious encephalopathy, but whether this is so and whether survival is prolonged has not yet been answered.

Finally, no account of this subject would be complete without mention of the prophylactic portacaval shunt, the operation being done before bleeding has occurred. However, the results to date of two carefully controlled trials in America show that the overall survival of such patients does not differ significantly from that of controls, though the incidence of variceal bleeding is considerably decreased.¹⁹ Only one of the 68 patients with a prophylactic shunt has bled since the time of randomization as compared with 19 of the 73 in the control group. This is gained at the expense of an increased incidence of encephalopathy, the figures being 25% for the shunted group and 5.5% for the controls. Here again one has to balance the disability to the patient of encephalopathy as opposed to recurrent bleeding, and this perhaps is one of the most difficult problems to evaluate at present.

REFERENCES

- Enquist, I. F., Dennis, C., Fierst, S. M., and Karlson, K. E., *Amer. J. Surg.*, 1964, 107, 306.
- Tsakiris, A., Haemmerli, U. P., and Bühlmann, A., *Amer. J. Med.*, 1964, 36, 825.
- Read, A. E., Dawson, A. M., Kerr, D. N. S., Turner, M. D., and Sherlock, S., *Brit. med. J.*, 1960, 1, 227.
- Linton, R. R., and Ellis, D. S., *J. Amer. med. Ass.*, 1956, 160, 1017.
- Tanner, N. C., in Colston's papers, *Liver Diseases*, ed. A. E. Read. Blackwell, Oxford, in press.
- Walker, J. G., Williams, R., Condon, R., Thompson, E. N., and Sherlock, S., *Lancet*, 1964, 2, 328.
- Britton, R. C., and Crile, G., *Surg. Gynec. Obstet.*, 1963, 117, 10.
- Hunt, A. H., *St. Bart's Hosp. J.*, 1965, 69, Suppl. No. 11.
- Walker, R. M., *Surg. Gynec. Obstet.*, 1964, 118, 323.
- Shaldon, C., and Walker, R. M., *Lancet*, 1962, 2, 1003.
- Dumont, A. E., and Witte, M. H., in *Current Surgical Management*, III, ed. E. H. Ellison, 1965, p. 264. Saunders, London.
- Vennes, J. A., *Medicine (Baltimore)*, 1966, 45, 445.
- Burchell, A. R., Moreno, A. H., Panke, W. F., and Rousselot, L. M., *Ann. Surg.*, 1965, 162, 981.
- Kreel, L., and Williams, R., *Brit. med. J.*, 1964, 2, 1500.
- Reynolds, T. B., and Redeker, A. G., in *Progress in Liver Diseases*, 1965, Vol. II, ed. H. Popper, and F. Schaffner. Grune and Stratton, New York.
- Voorhees, A. B., and Blakemore, A. H., *Surgery*, 1963, 54, 559.
- Tanner, N. C., *Ann. roy. Coll. Surg. Engl.*, 1961, 28, 153.
- Macbeth, R., *Brit. med. J.*, 1955, 2, 877.
- Grace, N. D., Muench, H., and Chalmers, T. C., *Gastroenterology*, 1966, 50, 684.
- Shaldon, C., in *Recent Advances in Gastroenterology*, 1965, ed. J. Badenoch, and B. N. Brooke. Churchill, London.
- Hislop, I. G., Waters, T. E., Kellock, T. D., and Swynnerton, B., *Lancet*, 1966, 1, 945.
- Sherlock, S., *Brit. J. Surg.*, 1964, 51, 746.