Case 88 A massive haematemesis

A married woman aged 50 years, who used to be a shop assistant but who is now out of work, was brought by ambulance to the Emergency Department having vomited several basins full of bright red blood and clots. She complained of feeling very faint and dizzy, but was not in any pain. She had seen bright red blood in her stools several times in the previous couple of months but had done nothing about this.

Her husband explained that his wife had been an alcoholic for the past 10 years, drinking a bottle of gin – or any other spirits she could lay her hands on – each day. Efforts by her family and friends, her family doctor and the local psychiatric unit to control the habit had been completely ineffective. On several occasions she had attended Alcoholics Anonymous, but each time defected after the first session. She had been told by the doctors that 'she was poisoning her liver' and the psychiatrist told her husband that the liver tests they had carried out were 'very bad'. Over the last year she had eaten very little and had lost a great deal of weight.

On examination, the patient was thin, looked 10 years older than her chronological age, was deathly pale, covered in a cold sweat and with a definite icteric tinge to her skin and especially to her conjunctivae. Her pulse was 110 beats/min and blood pressure 94/60 mmHg. The abdomen was distended and there was shifting dullness in the flanks. The spleen was palpable a couple of finger-breadths below the left costal margin but the liver could not be felt. There was pitting oedema of the ankles.

What is your clinical diagnosis in this sad case, and what is the anatomical basis of her massive haematemesis?

The whole clinical picture is that of severe cirrhosis of the liver resulting from her alcohol abuse – the jaundice, ascites and ankle oedema. Portal hypertension due to obstruction of the portal venous channels in her liver has resulted in her splenomegaly and the development of portosystemic anastomoses. The normal portal venous pressure is between 5 and 10 mmHg, this rises to 30 mmHg or more in portal hyper-tension.

Why do varices develop at the oesophago-gastric junction?

The lower oesophagus and the cardia of the stomach are the site of the most important of the anastomoses between the portal and systemic venous systems – here between the oesophageal branch of the left gastric vein (which drains into the portal vein) and the oesophageal veins draining into the azygos veins (systemic circulation).

Other anastomoses are between the superior (portal) and inferior (systemic) rectal veins in the anal canal, over the surface of the liver, and at the umbilicus, where the recanalized umbilical vein in the round ligament drains into the epigastric veins to form the caput medusae – dilated varices over the anterior abdominal wall. These anastomoses are shown in Fig. 88.1.

A blood transfusion was commenced in the Emergency Department, but she had another massive vomit of blood and her blood pressure became unrecordable. What emergency procedures would be used in an to effort to control the bleeding varices?

The patient was resuscitated aggressively with intravenous fluids, including blood to replace that lost, and her coagulation was corrected with fresh frozen plasma and platelets; she proceeded to endoscopy once cardiovascularly stable. At fibreoptic endoscopy, bleeding oesophageal varices were identified and controlled by variceal band ligation, where a small elastic band is secured around the base of the varix. Injection of the varices with a sclerosant solution at endoscopy is sometimes required where banding is technically difficult. Intravenous vasopressin analogues, such as terlipressin or glypressin, or somatostatin analogues such as octreotide, are frequently

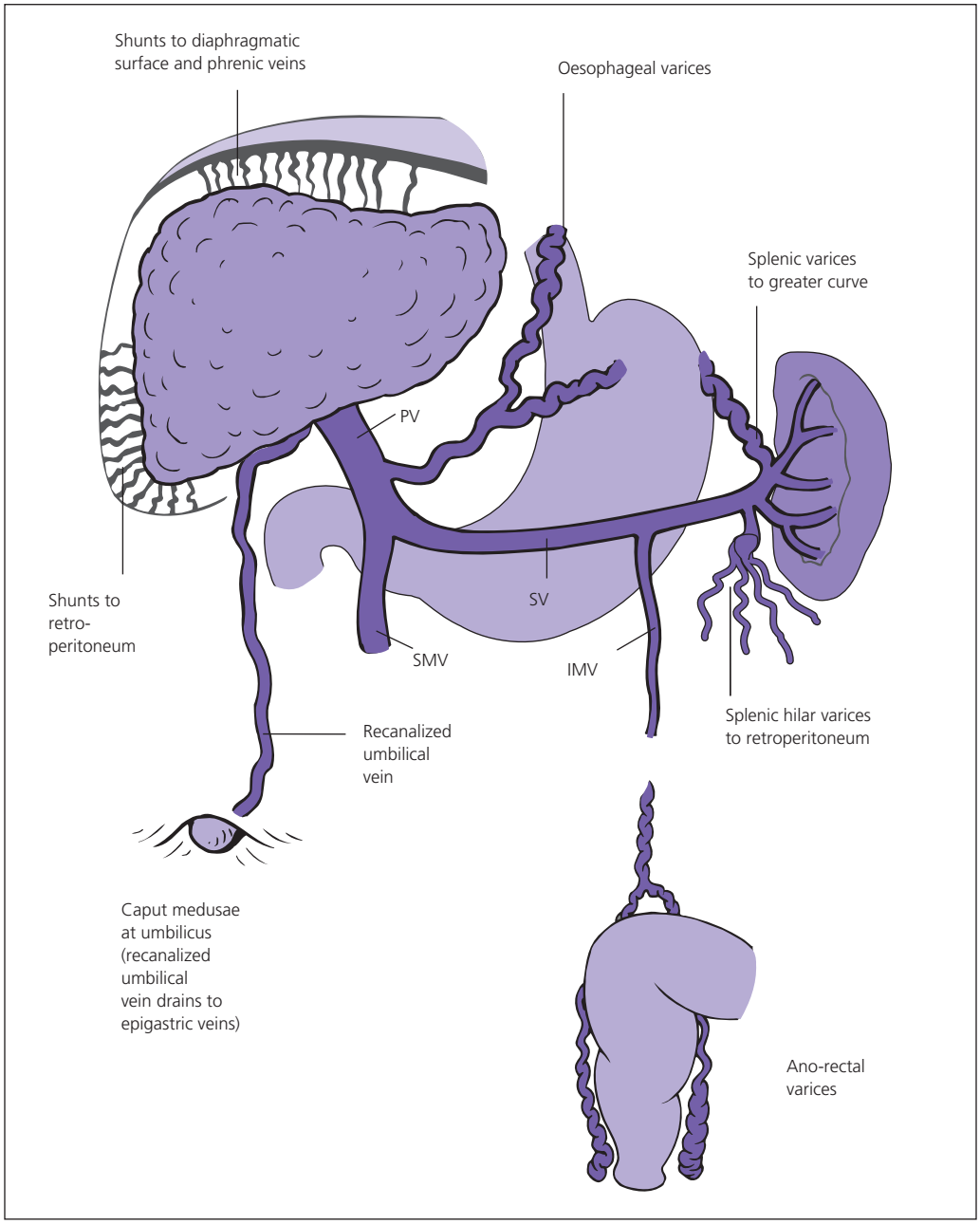

Figure 88.1 The sites of occurrence of portosystemic communications in patients with portal hypertension. IMV, inferior mesenteric vein; PV, portal vein; SMV, superior mesenteric vein; SV, splenic vein.

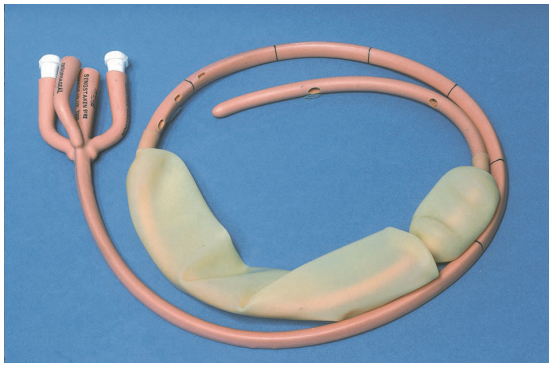

Figure 88.2 A Sengstaken–Blakemore double balloon catheter.

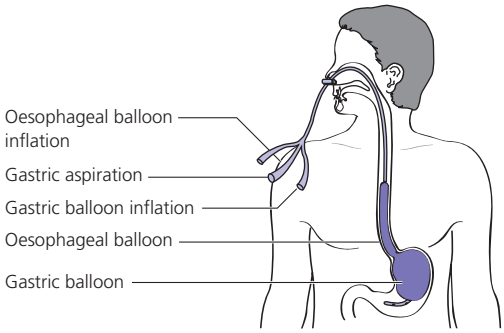

Figure 88.3 The double balloon catheter procedure.

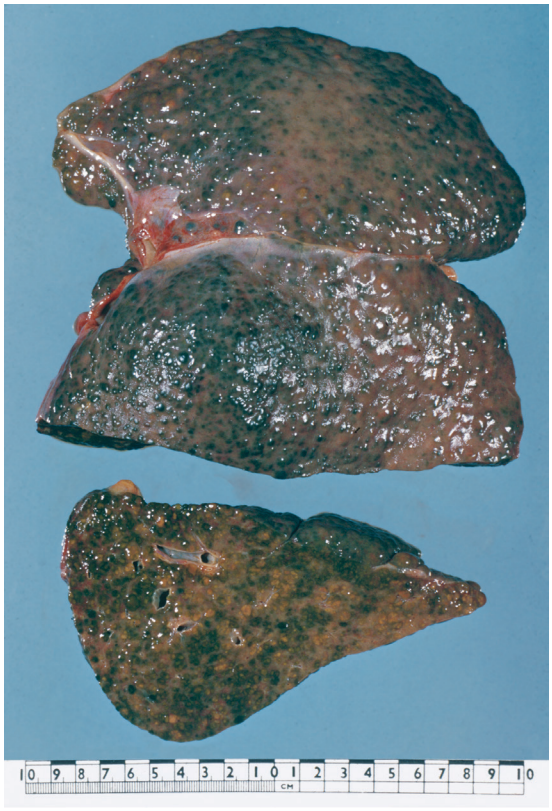

Figure 88.4 Slice of the patient's liver.

given in conjunction to produce a fall in portal venous pressure by mesenteric arteriolar constriction.

What other techniques are available to control the haemorrhage in these often desperate cases?

A Sengstaken–Blakemore* double balloon catheter (Fig. 88.2) was passed into the stomach and inflated. The lower balloon is inflated and the tube pulled back so that this balloon impacts at the oesophago-gastric junction (Fig. 88.3). Compressing the oesophago-gastric junction reduces portal blood flow into the oesophageal varices and in most cases arrests the bleeding. If bleeding continues the upper balloon is inflated to directly tamponade the oesophageal varices, although this is rarely necessary. The distal end of the tube is used to aspirate the stomach contents. Bleeding often restarts hen the balloon is deflated, so a definitive treatment needs to be available. Transjugular intrahepatic portosystemic shunt (TIPSS) formation or surgical portosystemic shunt formation may be required if band ligation is not possible.

Sadly, this patient died in spite of the attempts to stop the bleeding and replace her massive blood loss. The coroner was notified and an autopsy performed. Figure 88.4 shows a slice through her liver.

Describe the appearance of the liver in Fig. 88.4 and what would a section of it look like under the microscope?

The liver is coarsely nodular. The nodules are of varying

*Robert Sengstaken (b. 1923), neurosurgeon, New York; Arthur H. Blakemore (1897–1970), surgeon, Columbia Presbyterian Medical Center, New York.

size and comprise rounded areas of regenerating liver cells separated by fibrous septa.

This patient's spleen was palpable on clinical examination and her splenomegaly was confirmed at autopsy. How large must a spleen be before it is likely to be palpable?

The normal spleen 'fits into the palm of the hand'. It is tucked against the left leaf of the diaphragm in front of the 9th to 11th rib. A spleen must be at least three times its normal size before it can be detected clinically, so an easily palpable spleen represents considerable enlargement.