MEDICAL PRACTICE

*Occasional Review***Medical treatment of portal hypertension and oesophageal varices**

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Portal hypertension exists when the pressure in the portal venous system is raised above the normal range of 5-10 mm Hg. It becomes clinically important at or above 15 mm Hg. An increase in portal vascular resistance is the usual mechanism for portal hypertension, and by far the commonest cause of this in Western society is hepatic cirrhosis,¹ most frequently alcohol induced.

The liver has a dual blood supply of about one litre per minute² through the hepatic portal vein and the hepatic artery. The hepatic portal vein supplies about 75% of hepatic blood flow. After entering the liver at the porta hepatis these vessels arborise throughout the parenchyma, making up the components of the portal tracts along with bile ducts. The bile ducts are ensheathed by capillaries derived from the hepatic artery, which drain into the hepatic portal vein. Little highly oxygenated arterial blood therefore enters the hepatic sinusoids directly.³

Several factors exert control over hepatic blood flow, including the inlet sphincters of vascular smooth muscle which control blood flow into sinusoids² and from conduits between the hepatic artery and hepatic portal vein as direct arteriportal connections. Functional interactions between these two vascular systems occur as well as anatomical connections.² For example, an increase in blood flow through one circuit produces an increase in vascular resistance in the other, a process called reciprocity. Thus ligation of the hepatic artery is succeeded by a reduction in portal pressure and increased portal blood flow,

whereas portacaval anastomosis results in increased hepatic arterial blood flow. The regulation of flow by myogenic and metabolic factors is ill understood.² Hepatic blood flow may also be influenced by circulating hormones—for example, glucagon, which produces hepatic arterial vasodilation and antagonises hepatic arterial vasoconstriction.⁴ This mechanism may be important in stress, when glucagon secretion would ensure a high arterial blood flow supplying the increased oxygen demands of hepatocellular glycogenolysis.⁴

Studies in animals indicate that the hepatic artery has both alpha and beta₂ adrenoreceptors, while the hepatic portal vein has only alpha receptors. High concentrations of circulatory catecholamines produce predominantly alpha receptor effects, with vasoconstriction of both the hepatic artery and the hepatic portal vein. At lower concentrations dilatation of the artery predominates, increasing hepatic blood flow.⁴ The balance is further affected by the adrenergic innervation of splanchnic arteries, which reduces inflow in the hepatic portal vein at high circulatory catecholamine concentrations.^{4, 5}

Oesophageal varices

The consequence of portal hypertension is the development of an abnormal collateral circulation. The anastomosis of most importance clinically is at the lower end of the oesophagus: oesophageal varices. There is no direct relation between the portal venous pressure and the potential for bleeding but larger varices are associated with an increased risk of haemorrhage.⁶ It is not known whether the pressure measured directly across the varix endoscopically⁷ is related to variceal size or the likelihood of haemorrhage. Despite many years of speculation we remain completely ignorant about those factors responsible for rupture of a varix and variceal haemorrhage.

Two approaches exist to the treatment of oesophageal varices—one aimed at reducing portal pressure and the other at occluding varices without affecting portal pressure.

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Portal pressure reduction

Portal pressure can be reduced pharmacologically or surgically. The best known drug for this purpose is vasopressin. Initially it was thought to act solely by constricting splanchnic arterioles, which decreased portal venous flow, but recent studies show that it has the unique property of reducing portal vascular resistance.⁴ An infusion of vasopressin directly into the superior mesenteric artery is no more effective, and less safe, than intravenous administration.⁸⁻⁹ A synthetic analogue of vasopressin, triglycyl lysine vasopressin (glypressin), has a more prolonged effect, may have fewer side effects, and is probably more effective.¹⁻¹⁰⁻¹¹ An interesting modification of this treatment is the addition of sublingual nitroglycerin to parenteral vasopressin treatment, which may reduce or prevent the toxic effects of vasopressin on systemic haemodynamics as well as enhancing the reduction in wedged hepatic venous pressure.¹²

Somatostatin, a hormone found in the hypothalamus and delta cells of the islets of Langerhans, reduces splanchnic blood flow in man¹³ and has been used successfully to treat acute bleeding from gastroduodenal ulcers.¹⁴ The drug has been suggested for the treatment of variceal bleeding, but doubts about its efficacy are raised by the observation that the wedged hepatic venous pressure in patients with variceal bleeding is not reduced when somatostatin is administered at the usual dosage (4 µg/min).¹⁵ Bosch *et al* have shown a reduction in portal pressure at higher doses.¹⁶ The disadvantages of somatostatin are that it is expensive and has to be given intravenously, which makes it unsuitable for the long term treatment of portal hypertension.

Propranolol has recently been shown to lower portal pressure, reduce liver blood flow and cardiac index,¹⁷ and increase sodium excretion in cirrhotic patients after an acute sodium load.¹⁸ Increased sympathetic activity, as determined by circulating catecholamine concentrations, correlates closely with sodium and water retention in cirrhotic patients.¹⁹ Propranolol may protect from the renal impairment induced by diuretics during treatment in cirrhotic patients.²⁰ The mechanism underlying the reduction in portal pressure is not entirely clear and may not be due solely to a reduction in cardiac output, because atenolol, a specific beta₁ antagonist, produces a less pronounced fall in portal pressure with a similar decrement in cardiac output.²¹ There is a direct relation between the reduction in hepatic blood flow produced by propranolol and the fall in portal pressure.²²

Probably an additional effect exists,²¹ perhaps mediated via vasoconstriction in the splanchnic circulation due to unopposed alpha receptor activity in the face of beta₂ receptor blockade. Similarly, unopposed alpha receptor activity on the hepatic artery producing vasoconstriction may, by the principle of reciprocity, reduce portal vascular resistance. A direct effect on the hepatic portal vein is unlikely because the portal vein has only alpha receptors; indeed, unopposed alpha stimulation would tend to increase portal pressure. Such an effect may explain the observation of Colman *et al* that propranolol causes no reduction in portal pressure in patients with decompensated cirrhosis.²³ Patients with decompensated cirrhosis have raised catecholamine concentrations,²⁴ which may result in a predominant alpha effect (vasoconstricting) in the portal system, which would counteract any reduction in portal pressure produced by reduced cardiac output and decreased splanchnic blood flow.²³ A delicate balance therefore seems to exist between beta blockade and catecholamine concentration, with a reduction in portal pressure achieved only in certain circumstances.

Despite these observations several studies indicate that propranolol reduces recurrent gastrointestinal haemorrhage in patients with cirrhosis.²⁵⁻²⁷ In one study of cirrhotic patients who had had a gastrointestinal haemorrhage and were treated with propranolol, at a dose sufficient to reduce the resting heart rate by 25%, the proportion who remained free of gastrointestinal bleeding after one year was 96%, compared with 50% in a placebo treated group.²⁷ Propranolol probably reduces the

bleeding from gastric erosions as well as variceal rupture in cirrhotic patients. Bleichner *et al* showed that intravenous propranolol reduces portal venous pressure and stops bleeding from varices and acute gastric erosions in cirrhotic patients with gastrointestinal haemorrhage.²⁸

The effects of propranolol in cirrhotic patients on their blood ammonia concentrations and on the precipitation of encephalopathy are controversial.²⁹⁻³³ Further studies are necessary to verify the efficacy of propranolol in portal hypertension and ascertain how safe long term beta blockade is in patients with cirrhosis and at what dosage. Should it ever become necessary to discontinue beta blockade once propranolol has been introduced to treat portal hypertension, the drug should be tailed off slowly to avoid precipitating variceal haemorrhage.³⁴⁻³⁵ The adverse influence that propranolol may have on cardiac output in patients bleeding from oesophageal varices by inhibiting an appropriate tachycardia has been recognised,³⁶ and treatment of these patients with agents such as glucagon, atropine, isoprenaline, or dobutamine has been suggested.³⁷

The use of drugs such as cimetidine and other histamine antagonists such as pyridamine which influence hepatic blood flow or portal pressure, or both,³⁸⁻³⁹ needs further evaluation before their use can be endorsed in the treatment of patients with portal hypertension.

SURGERY

The most effective of the several surgical methods for lowering portal pressure, is the creation of a shunt between the portal and systemic circulation. Portal pressure is appreciably lowered, but at the expense of a reduction in hepatic blood flow because of blood that bypasses the liver. Surgical shunting is associated with the development of hepatic encephalopathy in about one third of patients.⁴⁰⁻⁴¹ There are many variations of the shunt procedure, including mesocaval and distal splenorenal shunts; all have been introduced in an attempt to reduce the incidence of postoperative encephalopathy but generally they have not achieved this purpose.⁴²⁻⁴⁴ Predictors of an adverse result after an emergency portacaval shunt include the presence of ascites, raised serum aspartate transaminase activity, hypokalaemic alkalosis, and recent alcohol ingestion. Prophylactic shunts performed in patients with varices which have not yet bled do not prolong life.¹⁻⁴⁵⁻⁴⁷ The outcome of shunt surgery depends primarily on the age of the patient and on hepatocellular function, which tends to deteriorate after shunting.⁴⁸⁻⁴⁹ Therefore a portasystemic shunt remains hazardous for many cirrhotic patients.

Variceal occlusion

Numerous techniques exist to occlude varices. Temporary control of bleeding oesophageal varices can be achieved by direct compression with the Sengstaken-Blakemore tube.⁵⁰⁻⁵¹ A more permanent occlusion can be obtained by a variety of techniques, including sclerosis, embolisation, and transection of the varices.

Sclerosis can be effected by several approaches, of which direct endoscopic injection sclerotherapy has become the most popular. This is usually performed through a flexible endoscope (with or without a flexible outer sheath with a distal slot which allows the vein to prolapse into the lumen, thereby simplifying injection and subsequent compression). The sclerosant may be injected either directly into each varix or into the adjacent tissue. Sclerosis by this technique should be attempted only after the initial bleeding has stopped and once the patient's condition is stable. Repeated injections have to be made to prevent recurrence of the varices.

Most reports of endoscopic sclerotherapy for variceal haemorrhage and portal hypertension have reported a reduction in episodes of rebleeding and perhaps an improved rate of survival at one year.⁵²⁻⁵⁵ Complications of this technique include early

oesophageal ulceration of the site of injection, which normally heals without complication, but late formation of a stricture may occur.

Transhepatic sclerosis and embolisation may also be undertaken to reduce the incidence of variceal haemorrhage. These techniques have not been evaluated as thoroughly as transoesophageal sclerotherapy, which is generally regarded as the best procedure for variceal sclerosis. Other methods include oesophageal transection with the stapling gun and oesophageal transection with ligation of the left gastric vein (with or without splenectomy).⁵⁶

Conclusion

In conclusion, portal hypertension, the main complication of which is variceal haemorrhage, may be treated medically or surgically. Only portasystemic shunting and beta blocking agents are likely to reduce portal pressure in the long term. Although effective in reducing portal hypertension and subsequent gastrointestinal haemorrhage, portasystemic shunting is hazardous in many cirrhotic patients and has not been shown convincingly to prolong survival. Propranolol reduces portal hypertension and gastrointestinal haemorrhage in some cirrhotic patients, but its complications and long term efficacy need to be established. In contrast to surgical procedures, there is no evidence that propranolol, although lowering hepatic blood flow as well as portal pressure, increases portasystemic shunting. Hence beta blockade may be accompanied by a reduced risk of precipitating hepatic encephalopathy. Patients with poor liver function fare badly after portacaval shunt surgery, and the pharmacological reduction in portal pressure with propranolol may be a better alternative. Thus the severity of hepatic disease will influence the therapeutic options. The combination of propranolol with a method of variceal sclerosis may well prove to be particularly effective management for portal hypertension.

Although the most appropriate therapy for an individual patient remains uncertain the following points should be considered. Firstly, portacaval shunt operations are tolerated best by patients with mild hepatocellular dysfunction and poorly by patients with advanced or decompensated liver disease. Secondly, the use of the Sengstaken-Blakemore tube, vasopressin or glypressin infusion, and perhaps variceal sclerosis or intravenous somatostatin has a place in controlling acute variceal haemorrhage. Thirdly, endoscopic injection sclerotherapy is effective in decreasing the incidence of rebleeding, at least in the short term. Fourthly, propranolol treatment can lower portal pressure and reduce rebleeding in patients with compensated cirrhosis, but it may be hazardous or ineffective in patients with decompensated cirrhosis and in those who already have a portacaval shunt. Finally, the correct management in selected patients may well be the combination of variceal ablation with a pharmacological reduction in portal pressure.

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For Debate . . .

Sport for tall

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Abstract

Eight new events (handball, basketball, and six rowing events) were introduced for women in the Olympic Games at Montreal in 1976. Of 187 women rowers who competed at Montreal, none was shorter than the mean height (162 cm, 64 in) of women aged 18-24 in the United States. In team events only two out of 250 participants were shorter than the reference mean. Even among the tall, it was the taller participants who won medals. What does the slogan "Sport for All" mean in this context? Moreover, the physical size required of champion rowers and basketball players is not to be found in some Asian, African, and Latin American populations.

International contests in many such events therefore seem to be at variance with the first charter of the Olympic Games. An independent reviewing body is urgently needed to examine the merits of man made rules in many sporting contests.

Introduction

There is an overwhelming bias in favour of the very tall in many athletics, swimming, and team contests.^{1,2} An urgent

need to review the sporting rules was suggested in 1968 but no action has been taken.¹ On the contrary, additional events have been introduced which are even more heavily biased. To focus attention on the problem results are presented for the following events which have recently been introduced for women: in rowing, sculls (single and double), coxless pairs, coxed quadruple sculls, coxed fours, and rowing eights and the team games handball and basketball. The choice of these events appears to be at variance with the slogan of the Sports Council, "Sport for All," and also with the first charter of the Olympic Games,³ which states: "They assemble the amateurs of all nations in fair and equal competition."

Data

The data on height and weight of the competitors taking part in the new eight events for women were derived from publications of the organising committee of the Olympics, Montreal 1976. The heights of women aged 18-24 in the United States were used as a reference population.⁴

Results

ROWING

Coxwains are known to be short and light, so coxswains from all women's rowing events are grouped separately in the table. As expected, they were 6.8 cm (2.7 in) shorter than the reference mean; the other mean differences ranged from +10.2 cm (4 in) for double sculls to +14.3 cm (5.6 in) for rowing eights. Out of 187 rowers from all events none was shorter than the reference mean. Only eight teams, all of them from North America and Europe, competed in

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