The portal pressure-blood volume relationship in cirrhosis

DAVID S. ZIMMON AND RICHARD E. KESSLER

From the Medical and Surgical Services, Veterans Administration Hospital, and New York University School of Medicine, New York, New York

SUMMARY Portal pressure-blood volume curves were derived in 13 cirrhotic patients with portal hypertension and oesophageal or gastric varices by measuring portal pressure at two levels of blood volume. Portal pressure varied directly with blood volume. In seven patients where portal pressure was measured at three levels of blood volume separated by 500 ml or more the portal pressure-blood volume relationship was found to be approximately linear.

Life-threatening haemorrhage from oesophageal or gastric varices occurs in patients with cirrhosis and portal hypertension. The frequency of this feared complication increases with increasing portal pressure (Rousselot, Moreno, and Panke, 1959; Jackson, Perrin, Felix, and Smith, 1971). Expansion of the blood volume raises portal pressure (Losowsky, Jones, Lieber, and Davidson, 1963). To quantitate the relationship between portal pressure and blood volume in cirrhosis we have studied the response of portal pressure to rapid augmentation or diminution of the intravascular volume.

Methods

All patients had oesophageal varices demonstrated by oesophagoscopy and were being evaluated for portal-systemic shunt surgery after suspected or documented variceal haemorrhage. The presence of cirrhosis with postsinusoidal portal hypertension was ultimately confirmed in every patient by liver biopsy, wedged hepatic venous pressure measurement, or necropsy.

Immediately before study $^{131}$I albumin was injected intravenously. Peripheral venous blood samples at 10 and 12 minutes measured plasma volume. Whole blood volume was calculated from the peripheral venous haematocrit performed in triplicate and corrected for trapped plasma, and the difference between peripheral and whole body haematocrit (Nadler, Hidalgo, and Bloch, 1962; Lieberman and Reynolds, 1967). Portal pressure was measured by umbilical venous or wedged hepatic venous catheterization. Central venous pressure was monitored through an additional central venous catheter. Pressures were recorded through Statham P-37 perfused strain gauges with zero pressure level taken as 12 cm above the couch. When the umbilical vein was catheterized, it was used for phlebotomy or infusion. Otherwise the central venous catheter was used for infusion and the femoral vein catheterized for phlebotomy. Whole blood was withdrawn into acid-citrate-dextrose solution and reinfused. To increase vascular volume whole blood, packed cells, or 6% Dextran were infused. Since large-bore catheters and large vessels were used it was possible to infuse or withdraw 500 ml in less than five minutes. These rapid changes in vascular volume minimized compensatory shifts of fluid into or out of the vascular space. Thus, change in vascular volume was calculated from the initial isotopic measurement and the volume of fluid withdrawn or infused during the brief period of study.

Results

Within the range (8.6-3.6 litres) of whole blood volume studied portal pressure varied directly with blood volume. In seven patients portal pressure-blood volume curves were constructed by measuring portal pressure at three different quantities of blood volume separated by 500 ml or more. The portal pressure-blood volume relationship was approxi-
mately linear (fig 1). In six additional patients curves were constructed from two portal pressure measure-
mments at blood volumes differing by at least 500 ml. The range of pressure-volume slopes in 13 patients
was 0·6 to 2·8 cm change in portal pressure per 100
ml change in blood volume. The mean change in
portal pressure was 1·4 ± 0·7 (mean ± SD) cm per
100 ml change in blood volume. Although central
venous pressure increased or decreased in response
to volume loading or withdrawal in individual
patients, it remained within the normal range
(4-14 cm).

Discussion

The inadequacy of central venous pressure measure-
ment in reflecting changes in vascular volume has
been well documented (Prout, 1968; Irvin, Hayter,
Modgill, and Goligher, 1972). Thus, it is not surpris-
ing that central venous pressure failed to indicate the
induced changes in blood volume or portal pressure.

The prime factor responsible for portal hyperten-
sion in cirrhosis is increased hepatic outflow
resistance. Plasma volume expansion by salt and
water loading (Losowsky et al, 1963), infusion of
albumin (Losowsky and Atkinson, 1961) or dextran
(Boyer, Chatterjee, and Iber, 1966) aggravates portal
hypertension. Boyer et al (1966) emphasized the
prolonged elevation in portal pressure that may
follow dextran infusion in cirrhotic patients unable
to excrete a volume load. Conversely, blood volume
depletion through haemorrhage, phlebotomy, or
fluid removal reduces pressure (Kessler, Santoni,
Tice, and Zimmon, 1969).

Cirrhotic patients have an increased plasma
volume and proportionately increased whole blood
volume (Lieberman and Reynolds, 1967). The
quantity of plasma volume that does not serve
essential functions in cirrhotic patients and con-
tributes to increasing portal pressure with the
attendant risk of variceal haemorrhage ought to be
considered excess volume. Particularly during
episodes of fluid retention or inadequately moni-
tored blood or fluid replacement, intravascular
excess volume produces elevations in portal pressure
that are avoidable and could precipitate variceal
haemorrhage (Taylor, 1954).

Normal subjects tolerate acute reduction in whole
blood volume approaching 1 litre without signifi-
cant change in cardiac dynamics or hepatic venous
pressure (Price, Deutsch, Marshall, Stephen, Behar,
and Neufeld, 1963). Repeated episodic depletion of
plasma volume and presumably reduction of portal
pressure occurs covertly during diuretic therapy of
cirrhotic patients with ascites (Shear, Ching, and
Gabuzda, 1970). As evidenced by the favourable
clinical response to diuretic therapy, the majority of
cirrhotic patients with fluid retention may achieve a
reduction in portal pressure without reducing plasma
volume to the point where cerebral, renal, or hepatic
perfusion is impaired.

We have observed individual patients with severe
portal hypertension, probably due to markedly
increased hepatic outflow resistance, who are not
amenable to these manoeuvres. Depletion of plasma
volume to a level where urine output ceases and
shock supervenes leaves portal pressure markedly
elevated (Kessler et al, 1969). On the other hand,
patients presenting with oedema and ascites fre-
quently have a significant amount of excess plasma
volume that increases portal pressure and can be
removed with immunity.

By emphasizing the portal pressure-blood volume
relationship we wish to achieve precision in the
management of patients with portal hypertension at
risk from variceal haemorrhage. One hopes to tread
the narrow path between the hazards of plasma
volume depletion leading to hepatic and renal
malfunction and plasma volume excess leading to
increased portal pressure and its complications.

References


Irvin, T. T., Hayter, C. J., Modgill, V. K., and Goligher, J. C.
Lancet, 2, 446-449.
The portal pressure-blood volume relationship in cirrhosis


