

Effect of Hepatic Artery Ligation on Portal Pressure in the Normal and Cirrhotic Rat. (30669)

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The hepatic artery has been thought by some investigators(1) to be a significant contributor to the portal hypertension of cirrhosis, and at one time its ligation was advocated for treatment of that condition in certain cases(2,3). Most of the evidence in support of this concept, however, has been indirect, based on studies of pressure relationships in perfused, autopsy livers(4,5,6,7). For a recent study(8) on the isolated perfused calf's liver it was concluded that no reciprocal pressure relationship existed between the artery and portal vein in the normal liver.

The only *in vivo* measurements reported on the effect of hepatic artery ligation on portal pressure in the normal animal were those of McMichael(9), who measured the effect in the splenectomized cat, and Berman(10), who observed the effect in the normal dog. Both noted drops of 15-25 mm Hg after artery ligation. However, in both studies the gastroduodenal artery was also obstructed, thus the effect of pure hepatic artery ligation could not be observed.

There have been no studies reported to date measuring the effect in the cirrhotic subject.

Methods. Subjects were normal 300 g male Wistar rats, a normal 2 kg rabbit, and 18 cirrhotic rats. The latter had been exposed to CCl₄ vapor twice weekly for approximately 5 months, and were classified into an Early and Advanced Group according to histologic criteria. Under light ether anesthesia laparotomy was performed, the hepatic artery identified, and a fine silk suture passed under it by means of an attached cutting needle, taking care not to induce spasm in the vessel. A No. 1 needle, attached *via* heparinized cannulae to an electromanometer, was then inserted

into the portal vein. After achieving a stable pressure level, the hepatic artery ligature was pulled tight, indicating the precise point on the tracing (Fig. 1). Patency of the needle could be checked by the presence of normal respiratory pulsations in the tracing.

Results. The results are summarized in Table I.

The mean percentage drop in portal pressure after hepatic artery ligation in normal rats was $12.5\% \pm 4.7$ S.D., in the rabbit 13%.

There was no significant difference in mean absolute drop between the normal group and either cirrhotic group. The small differences in mean percentage drops were accounted for by the differences in initial portal pressure.

Discussion. Recent anatomic studies(11) on the nature of the intrahepatic termination of the hepatic artery have demonstrated conclusively the presence of microscopic arterio-portal shunts in normal mammalian liver. These had previously been seen in vital microscopic studies(12,13). It is *via* these shunts presumably that the arterial pressure is transmitted to the portal bed to the small but consistent degree noted in the normal liver. One might reasonably expect an exaggeration of this effect in cirrhosis, where the arterial bed is generally enlarged(14) and thus presumably the shunts as well. However, this was not observed in this study, even in those cases where the hepatic artery appeared dilated at the time of laparotomy. The possibility remains that a greater effect might have been observed in cirrhosis of much longer standing. At any rate, the findings of this study do not support a significant role for the hepatic artery in the pathogenesis of portal hypertension, at least in experimental cirrhosis.

Summary. Portal pressures were measured in 16 normal rats, one normal rabbit, and 18 cirrhotic rats, before and immediately after ligation of the hepatic artery. The normal

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