Occlusive Hepatic Venous Catheterization in the Study of the Normal Liver, Cirrhosis of the Liver and Noncirrhotic Portal Hypertension

By W. Jape Taylor, M.D., and J. D. Myers, M.D.

The technic of occlusive venous catheterization has been applied to the liver. The pressure, measured in a small occluded hepatic vein and termed the wedged hepatic venous pressure, correlates well with the portal venous pressure in cats. An elevation of the wedged pressure in man, in the presence of a normal central venous pressure, is diagnostic of cirrhosis. This has proved to be of clinical value in the differentiation of intrahepatic from extrahepatic portal hypertension. The higher wedged hepatic venous pressures in cirrhosis are correlated with the presence of varices or jaundice, but not with the presence of ascites.

The anatomic location of the portal vein is such that measurements of portal pressure in man have been possible only at laparotomy. The relative inaccessibility of the portal vein has been a particular handicap in the study of Laennec’s cirrhosis in which the effects of deranged intrahepatic circulation with resultant portal hypertension are of great importance in the course and ultimate outcome of the disease. Various indirect methods for giving an indication of portal pressure have not proved fully satisfactory, and the need for a suitable technique for estimating portal venous pressure has become more pressing with the advent of direct surgical means for attacking the problem of portal hypertension. A method for direct needling of the portal vein in unanesthetized man has recently been reported but its applicability would appear to be limited.

From the Department of Medicine, Duke University School of Medicine Durham, N. C.

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Dr. Taylor was a Research Fellow of the American College of Physicians, 1950-51 and is a Research Fellow of the U. S. P. H. S. National Heart Institute, HF-5074, 1954-55.

Occlusive venous catheterization as applied in the pulmonary circuit has proved to be a valuable tool in the study of cardiac physiology and disease. Its application to the hepatic vascular system seemed worthy of trial despite the disadvantage of a dual afferent blood supply to the liver.

In 1951, the technic of occlusive hepatic venous catheterization in man was described with a report of preliminary results in man and in the cat. The present paper provides observations obtained by this method from a group of patients with Laennec’s cirrhosis, from a control series of subjects without liver disease, from a number of patients with extrahepatic portal hypertension and from a few patients with congestive heart failure. Data from animals, comparing this occluded hepatic venous pressure with the directly measured portal pressure, are also given. Factors important in assessing the relationship of this pressure to the portal venous pressure are discussed and a correlation of an elevation of the pressure with the complications of cirrhosis is presented.

The pressure obtained in an occluded small hepatic vein was originally termed the hepatic "sinusoidal" pressure with full realization of the anatomic inaccuracy of this name, but with the belief that the pressure gradient between the sinusoids and the occluded vein is small under our experimental circumstances, as discussed later. Friedman and Weiner
arrived separately at this same designation in reporting the use of a similar technique in dogs. Subsequent similar studies in man have been reported by Paton, Reynolds, and Sherlock, by Atkinson and Sherlock, by Krook and by Reynolds and co-workers. Reynolds and his colleagues have designated this occluded pressure the “wedged hepatic vein pressure” and a similar descriptive term is now frequently used in reference to the occluded pulmonary artery pressure. In the interest of uniformity, the name, wedged hepatic venous pressure, will be used throughout this paper.

METHODS

In man an open-end venous catheter (usually no. 7 French) is passed with fluoroscopic visualization via an antecubital vein into a right hepatic vein by the usual technique. The catheter is then extended peri-pilherally as far as possible with moderate pressure so as to occlude a small hepatic vein. The exact position varies with the individual subject, but the catheter tip usually appears to lie within 2 to 4 cm. of the lateral abdominal wall (fig. 1). Since fluoroscopy has been done only in the posteroanterior projection, localization in this plane has not been feasible. If much kinking of the catheter in the right atrium is produced in attempting to wedge the catheter peripherally into the hepatic vein, artefacts are produced presumably by the slapping of the catheter with cardiac contractions. These artefacts have a contour suggestive of an arterial pulse wave and were originally believed to be possibly such waves transmitted through the sinusoidal system. Subsequently, such waves have been recorded almost at will merely by kinking the catheter in various positions in the right atrium.

On withdrawing the catheter into a proximal, large hepatic vein, the operator frequently is aware of an abrupt “giving” sensation indicating that the catheter was well wedged into the small vein. When a catheter is similarly extended into a hepatic vein of an autopsy specimen, it is difficult to keep it in the occlusive position. The “giving” sensation on withdrawal is not present in the dead liver suggesting that in life there may be some muscular contraction of the small vein about the catheter. However, the occluded vascular bed, intravital, is not in striking spasm as fluid can be readily and without much resistance perfused through the catheter into the occluded vascular bed. Sizeable blood samples can usually be withdrawn using gentle suction. Blood aspirated through the occluding catheter has, in general, the same oxygen content as true hepatic venous blood from that same general area of the liver (tables 2 and 3). When aspiration is too vigorous, the oxygen content of the blood becomes higher than that of hepatic venous blood. This is presumably because the blood is drawn so rapidly through the hepatic parenchyma that normal metabolic exchange does not have time to take place.

Pressures were measured with either a Hamilton manometer or with a Lilly electromanometer, using for the baseline a point 5 cm. below the suprasternal notch. Usually the wedged hepatic venous pressure was constant, therefore being recorded as a straight line. When there were significant respiratory variations, mean pressure was determined by integration of the area with a polar planimeter. This latter technic was also used for determination of mean arterial pressure (measured through an inflying needle in a femoral artery) and for atrial pressures.

Measurements were made to the nearest 0.5 mm. Hg.

In the individual subject, the wedged hepatic venous pressures measured from different sites in the liver vary by only 1 to 2 mm. Hg. Therefore, when multiple determinations were made, an average figure is given. These small differences make a greater per cent variation in normal subjects, since their pressures are of smaller magnitude. In the study of induced changes in the wedged hepatic venous pressure, it is desirable that the catheter site remains unchanged.

Patients were studied in the postabsorptive state. Usually phenobarbital (90 mg.) had been given orally one hour before the procedure. The study was performed in an air conditioned room. It was accomplished generally without discomfort to the patient and could usually be completed within 30 minutes, except in those cases in which hepatic blood flows and other determinations were subsequently done. In no case has there been any untoward complication.
The group of patients with Laennec's cirrhosis had the usual and typical clinical picture of that disease, in general at a moderately advanced stage. Twenty-five of the 27 cirrhotic patients entered the hospital because of complaints referable to the cirrhosis, (ascites, bleeding varices; jaundice, etc.). In the other two, hepatosplenomegaly and deranged hepatic function, indicative of cirrhosis, were found incidental to study for pulmonary disease. The diagnosis of cirrhosis was confirmed in 17 of the 27 cases by needle biopsy of the liver. Derangement of liver function was severe in all except one case, and that individual was in the group with an hepatic biopsy diagnostic of cirrhosis.

Esophageal and/or gastric varices were considered to be present, if demonstrated by x-ray study, at operation or at autopsy, or if there was a clearcut history of massive hematemesis without other demonstrated cause. The control group was a mixed hospital population of subjects with mild or convalescent illnesses, not expected to produce major hepatic disorders or alteration of portal venous pressure.

Hepatic blood flows were estimated by the bromsulphalein method of Bradley and colleagues with the exception that in many of the cirrhotic subjects, hepatic extraction of bromsulphalein was too poor to permit estimation of hepatic blood flow. In a few of these, blood flow was estimated by the urea technic as previously described; urea concentrations in blood and urine were measured by the colorimetric method of Archibald. Plasma volumes were estimated by the Evans blue method of Gibson and Evans, using a single 10-minute arterial sample after the dye injection. Blood volumes were calculated from plasma volume and hematocrit and the blood volumes were then expressed as per cent of normal, using Gibson's normal values. Oxygen contents of blood samples were determined by the spectrophotometric method of Hickam and Frayer and serum bilirubin by the technic of Mallory and Evelyn. Bromsulphalein excretion is expressed in terms of bromsulphalein (BSP) clearance, as previously defined in per cent of normal clearance.

In animal experiments, cats were anesthetized with pentobarbital, 30 mg. per kilogram intraperitoneally, and a no. 6 French, open-end catheter was placed via the right jugular vein, through the right atrium, occlusively into a small branch of an hepatic vein. A laparotomy was done and a no. 25 hypodermic needle with a hardened plastic extension was placed in the superior mesenteric, portal or splenic vein, with the tip pointing toward the liver. Paired pressures were then recorded from the occluded hepatic vein, via the catheter, and from the portal vein or its tributary within 30 seconds of one another. Since the objective was the comparison of these paired pressures from the two sites rather than their absolute values, the cats were not uniform in regard to fasting, degree of anesthetia, or conditions generally, although most of them remained in a good state throughout the procedure.

Results

Agreement was good between the paired portal venous pressures and the wedged hepatic venous pressures in 18 observations on 10 cats, with the mean wedged pressure being 8.6 mm. Hg and the mean portal venous pressure 8.0 mm. Hg (table 1). This difference is not significant, and the coefficient of correlation is 0.772 (p < 0.01). Epinephrine, given directly into the portal vein, produced a rise in the portal venous pressure, but the agreement among 10 paired pressures remained good (r = 0.663) (p < 0.05), although the wedged hepatic venous pressures were higher than the portal venous pressures to a greater extent than without epinephrine. These points are demonstrated in figure 2 where portal venous pressure is plotted against wedged hepatic venous pressure.

The wedged hepatic venous pressure in each of the 27 cirrhotic patients was higher than the pressure recorded in any one of the group of 18 control patients. In the cirrhotic subjects, the pressures ranged from 11.0 mm. Hg to 50.0 mm. Hg with a mean of 22.3 mm. Hg, while the pressures in the 18 control subjects varied from 0 to 9.5 mm. Hg with a mean value of 5.2 mm. Hg (tables 2 and 3). The difference between the means for the two groups (p < 0.01) is highly significant statistically. No correlation exists between the estimated hepatic blood flow and wedged pressure.

There is a significant difference (p < 0.01) between the mean hepatic venous pressure in the cirrhotic group as compared with the controls, although no overall difference in the right atrial or femoral arterial pressures were found. The elevated hepatic venous pressure could not be simply correlated with the presence or absence of ascites, probably because of other factors some of which increased intraabdominal pressure while certain others augmented central venous pressure and, thereby, hepatic venous pressure.

Study of the gradient between wedged hepatic venous pressure and central hepatic
venous pressure provides in general much higher gradients in the cirrhotic group than in the control subjects, the mean figure being 16 ± 1.7 versus 1.8 ± 2.1, a highly significant difference. Only one gradient in the control group exceeded 6 mm. Hg (the normal mean of 1.8 mm. Hg plus two standard deviations), this being 7.5 mm. Hg in patient C.B. The reason for this high gradient is not apparent. Only one gradient (3.5 mm. Hg in patient E.G.) in the cirrhotic group fell below a value of 6 mm. Hg. The possible meaning of the low gradient in E.G., who had proven cirrhosis, is discussed later. In general, the wedged-circular hepatic venous pressure gradient reflects the wedged pressure alone and computation of the gradient provides no additional diagnostic information.

Figure 3 is a diagrammatic presentation of the wedged hepatic venous pressures in the cirrhotic patients separated into groups, according to the presence or absence of ascites and/or varices in various combinations. It can be seen that most of the higher pressures occurred in

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OCCLUSIVE HEPATIC VENOUS CATHETERIZATION

Fig. 2. Relationship of the wedged hepatic venous pressure to portal venous pressure in cats. The heavy line represents absolute agreement of the pressures.

TABLE 2.—Wedged Hepatic Venous Pressure and Related Circulatory Data in Subjects Without Liver Disease

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<th>Subject</th>
<th>Diagnosis</th>
<th>Age Yrs.</th>
<th>Wedged hepatic venous pressure (mm. Hg)</th>
<th>Central hepatic venous pressure (mm. Hg)</th>
<th>Gradient</th>
<th>Rt. atrial pressure (mm. Hg)</th>
<th>Mean arterial pressure (mm. Hg)</th>
<th>Estimated hepatic blood flow (ml. min./sq. M.)</th>
<th>Hemoglobin (Gm. per 100 ml.)</th>
<th>Arterial-wedged hepatic venous oxygen difference (Vol. %)</th>
<th>Arterial-central hepatic venous oxygen difference (Vol. %)</th>
<th>BSP clearance (% normal)</th>
<th>Blood volume (% normal)</th>
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No........................................... 18   18   18   9   14   15   17   17   15   16
Mean........................................ 5.2  3.4  1.8  2.5  80.1 736 12.9 4.6 80.0  +5.3
S.D......................................... 2.4  2.1  2.1  2.1 14.2 194 3.4 1.0 11.8  10.0
S.E......................................... 0.6  0.5  0.5  0.7  3.8  50  0.8  0.2  3.1  2.5

individuals having varices. This association proved to have statistical significance, the difference between the mean pressures of the group with varices and those without varices having a p value of <0.01. The presence of ascites, on the other hand, is not correlated with any significant trend in the wedged pressure, as compared to those individuals who have cirrhosis without ascites.

The blood volume in the group of cirrhotic patients was elevated to a mean value of 17 per cent above normal. The larger blood volumes are, in general, associated with the higher wedged hepatic venous pressures. However, some striking deviations from this are apparent and the coefficient of correlation (r) is only 0.35.

A few of the patients with cirrhosis were moderately jaundiced at the time of the study. This was associated with an exacerbation of their disease and, in general, with very large livers. An arbitrary separation was
made between those patients with total serum bilirubin greater than 3.0 mg. per 100 cc. and those with less than 3.0 mg. per 100 cc., since this division seemed to fit best with a clinical separation of these two groups. The more deeply jaundiced patients had much higher pressures than the nonjaundiced or lightly jaundiced patients, the mean pressures being 27.1 mm. Hg and 19.3 mm. Hg, respectively ($p < 0.01$). In the statistical analysis which correlates pressures with the complications of cirrhosis, the last patient (B.S.) is omitted because he clearly represented a unique circumstance, as is subsequently discussed.

Table 4 summarizes 11 observations on 10 patients with portal hypertension clinically who eventually proved not to have hepatic cirrhosis. Five of these, who had hemorrhaged from esophageal varices, were thought to have cirrhosis on clinical grounds, but in all five wedge hepatic venous pressures were normal and all five subsequently had normal

<table>
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<tr>
<th>Subject</th>
<th>Age Yrs</th>
<th>Wedged hepatic pressure (mm. Hg)</th>
<th>Central hepatic pressure (mm. Hg)</th>
<th>Gradient (mm. Hg)</th>
<th>Mean arterial pressure (mm. Hg)</th>
<th>Hemoglobin (Gm. per 100 ml.)</th>
<th>Arterial-wedge hepatic venous oxygen difference (Vol. %)</th>
<th>Arterial-central hepatic venous oxygen difference (Vol. %)</th>
<th>BSP clearance (% of normal)</th>
<th>Ascites</th>
<th>Esophageal varices</th>
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* Hepatic blood flow estimated by urea method.
† Determinations made one year previously.
‡ Post-necrotic cirrhosis. Not used in statistical analysis of complications of cirrhosis. See text.
liver biopsies. In the five other patients with the syndrome of portal hypertension (four with esophageal varices and one with ascites and splenomegaly), clinical evidence provided a diagnosis of portal hypertension of extrahepatic cause. Again, all five had normal wedged hepatic venous pressures and no cirrhosis was demonstrated by liver biopsy. Laparotomy in 8 of the above 10 patients confirmed the presence of extensive portal venous collaterals, and direct pressure measurements in peripheral branches of the portal venous system demonstrated portal hypertension in all eight.

Measurements of the wedged hepatic venous pressure were made in four patients with congestive heart failure of diverse etiology. The mean right atrial pressure in this group was 14.5 mm Hg and the central hepatic venous and wedged hepatic vein pressures 14.5 and 15.0 mm Hg, respectively. These data, (table 5) are in agreement with those of Kook,10 and Paton, Reynolds and Sherlock, demonstrating that an elevation in central venous (i.e., right atrial), pressure is accompanied by a comparable increase in central hepatic venous and wedged hepatic venous pressures. There is no elevation of the gradient between wedged pressure and the central hepatic venous pressure.

**Table 4.—Wedged Hepatic Venous Pressure and Related Circulatory Data in Patients with Extrahepatic Portal Hypertension**

<table>
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<tr>
<th>Subject</th>
<th>Etiology of portal hypertension</th>
<th>Age Yrs.</th>
<th>Wedged hepatic venous pressure (mm. Hg)</th>
<th>Central hepatic venous pressure (mm. Hg)</th>
<th>Gradient (mm. Hg)</th>
<th>Rt. atrial pressure (mm. Hg)</th>
<th>Mean arterial pressure (mm. Hg)</th>
<th>Hemoglobin (Gm. / 100 ml.)</th>
<th>Arterial-wedged hepatic venous oxygen difference (Vol. %)</th>
<th>Arterial-central hepatic venous oxygen difference (Vol. %)</th>
<th>BSP Clearance (% normal)</th>
<th>Blood volume (% normal)</th>
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TABLE 5.—Wedged Hepatic Venous and Central Venous Pressures in Heart Failure

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<th>Age (Yrs.)</th>
<th>Diagnosis</th>
<th>Wedged hepatic venous pressure (mm. Hg)</th>
<th>Central hepatic venous pressure (mm. Hg)</th>
<th>Gradient (mm. Hg)</th>
<th>Right atrial pressure (mm. Hg)</th>
<th>Mean arterial pressure (mm. Hg)</th>
<th>Arterial-central hepatic venous oxygen difference (vol. %)</th>
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No. . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . .
normal persons may be less than the true portal venous pressure, possibly because of the richness of the anastomotic runoff, although the observations of Paton and coworkers do not support such a discrepancy.

Direct measurements of portal venous pressure in cirrhotic subjects have also been made at laparotomy. It is of note that such pressures are of the same general magnitude as the wedged hepatic venous pressures of the present cirrhotic group.

Two factors are of paramount importance in considering the relationship of the wedged hepatic venous pressure to portal venous pressure: (1) the influence of the hepatic arterial pressure on the total afferent blood pressure to the liver and (2) the collateral circulation within the liver, which determines the degree to which the wedged catheter obstructs blood flow.

It is apparent that the hepatic arterial pressure and the portal venous pressure must come into equilibrium at the point where the two systems merge or there could be no portal venous flow. Thus one may look upon the two afferent hepatic pressures, portal venous and arteriolar, as being in equilibrium in the proximal, i.e., more portal areas of the sinusoidal bed.

The next consideration, therefore, must be the pressure gradient between this proximal portion of the sinusoidal bed and the occluded hepatic vein. In the normal liver the pressure in the obstructed area will just barely exceed that in the neighboring unobstructed areas because of the richness of the sinusoidal anastomoses and the interconnections from one small hepatic vein to another, i.e., between hepatic veins smaller than that which the catheter occludes. The measured wedged hepatic venous pressure can be lower than the true afferent pressure (portal venous and hepatic arteriolar pressures in equilibrium) but by no more than the pressure gradient across the sinusoids and their anastomoses. Since this gradient is normally quite small, the sinusoidal bed being a low resistance system, the wedged hepatic venous pressure approximates the true portal venous pressure in the normal liver. That this is a low resistance system has been established by the observations of others as well as ourselves that postmortem perfusion of the liver through the portal vein is easily accomplished in large volume at low pressure. Furthermore, we have found that reverse perfusion through the hepatic vein with the portal vein as efferent is about as easily accomplished.

In cirrhosis of the liver, the fibrous disorganization of the hepatic architecture and vasculature reduces significantly the inter-sinusoidal and intervenous anastomoses from one hepatic segment to another. If the anastomotic channels are very severely constricted, it might be expected that the pressure in the occluded hepatic vein would of necessity approach hepatic arterial pressure. In actuality the wedged hepatic venous pressure in cirrhosis rises approximately to the portal venous pressure and further rise is prevented by dissipation of pressure into such collateral channels as are still present within the liver, as, for example, the portal venous to hepatic venous communications demonstrated by Popper and associates. Furthermore, the large volume portal venous bed with its extrahepatic collaterals does not allow any rise of the wedged hepatic venous pressure above the portal venous pressure.

In some circumstances, however, the wedged hepatic venous pressure does not give a true estimate of portal venous pressure in cirrhosis. The last cirrhotic patient, (B.S.), with “postnecrotic” scarring, had a liver estimated to be one fifth of normal size at laparotomy and a portal venogram demonstrated that the portal vein was patent up to the liver, although occlusion of the portal vein within the liver could not be excluded. Such large collateral veins were present that a more effective portacaval shunt could not have been produced surgically. The wedged hepatic venous pressure, which had been measured previously, was 50 mm. Hg, greatly exceeding the directly measured portal venous pressure of 15 mm. Hg. The hepatic blood flow was reduced to 340 ml. per minute per square meter of surface area, which very likely indicates a greatly diminished portal venous flow through the liver. Instances of
portal collaterals without portal hypertension are rare, however, and it remains our opinion that in the great majority of cases of unoperated cirrhosis the wedged pressure reflects with remarkable accuracy the elevated portal venous pressure.

It was the hope preliminarily that repeated measurements of wedged hepatic venous pressure would be useful in the postoperative evaluation of various surgical procedures for portal hypertension of cirrhotic origin. However, studies thus far indicate that, in severe cirrhosis associated with portal hypertension, the wedged pressure remains high after complete diversion of portal venous blood from the liver through an end-to-side portacaval anastomosis. This is interpreted to mean that in advanced cirrhosis the obstruction to flow is so marked that a high intrasinusoidal pressure is provided by the hepatic arterial different alone. Under such circumstances the wedged pressure cannot, of course, reflect portal venous pressure nor can it give any idea of the efficacy of the portacaval shunt in reducing portal venous pressure.

That the wedged hepatic venous pressure does ordinarily give a good estimate of portal venous pressure in cirrhosis indicates that the vascular obstruction in cirrhosis is either sinusoidal or postsinusoidal. If the obstruction were presinusoidal, the wedged pressure would be much lower than the portal pressure.

A secondary factor, the intra-abdominal pressure, would be expected to affect the wedged hepatic venous pressure. In our studies, no measurements of intraabdominal pressure have been made. However, it would be expected to affect the wedged pressure and central hepatic venous pressure alike; thus, there would be no effect on the wedged-central hepatic venous pressure gradient, which would remain small in the normal person and high in subjects with cirrhosis. The clinical and diagnostic significance of a high wedged hepatic venous pressure would not be masked by an increase in intra-abdominal pressure. Likewise, any influence which elevates the systemic central venous pressure raises the wedged hepatic venous and portal venous pressures concomitantly, and the gradient is not disturbed. This is best illustrated by congestive heart failure where the wedged hepatic venous pressure is elevated but only by an increment equal to the increase in systemic venous (including central hepatic venous) pressure.

Although some of the theoretic aspects are not subject to direct proof, the practical usefulness of occlusive hepatic venous catheterization seems well established. The separation of the high pressures in Laennec's cirrhosis from the low pressures in patients without advanced liver disease has been absolute to the point that an elevation of the wedged hepatic venous pressure has been useful in differential diagnosis. A special corollary of this ability to distinguish cirrhosis is the separation of cirrhotic from noncirrhotic (extrahepatic) portal hypertension. Frequently, this differentiation is of crucial importance in planning surgical treatment for portal hypertension and, at times, a correct decision may be made by other means only with considerable difficulty. To date, the accuracy of the wedged hepatic venous pressure in separating the portal hypertension of cirrhotic from that of noncirrhotic etiology has been absolute.

The fact that the wedged hepatic venous pressure is no higher in cirrhotics with ascites than in those without it is further evidence that an increased portal pressure, per se, is not the determining factor in ascites formation.

Esophageal varices are generally believed to be a result of increased portal venous pressure. The difficulty in relating the observations herein reported to the pathogenesis of varices is that the varices are already present when the study is made and the portal pressure may have been quite different during the development of the varices. An example of this is seen in the patient, J. P., whose wedged hepatic venous pressure changed from 37 to 20 mm. Hg over a period of several months of medical therapy, during this time, ascites and jaundice disappeared, liver function improved, and his liver decreased in size. However, varices persisted and even bled at this later time. Nevertheless, as pointed out earlier, the demonstration of higher wedged pressures in
the group of patients with cirrhosis and varices, than in those having cirrhosis but without varices, supports the view that the level of the portal pressure is important in the formation of varices. It is well known clinically and recently reemphasized by Walker\(^\text{19}\) that the correlation of the degree of elevation of portal venous pressure and the intensity of the cirrhotic process by pathologic criteria is by no means close. Patient E. G., in the present series is a striking example of such a discrepancy. This patient, who had extensive esophageal varices and repeated massive hematemeses, had the lowest wedged hepatic venous pressure in the entire cirrhotic group. His cirrhosis had been proven by biopsy, but he had a normal bromsulphalein clearance one year previous to the pressure measurements and a normal routine 45-minute bromsulphalein test at the time of the study. The explanation for his relatively low pressure is not clear. It is possible that a recent severe hemorrhage had reduced his portal venous and wedged hepatic venous pressures, or that some extrahepatic factor, such as a portal venous thrombus, influenced the formation of varices without much rise in intrahepatic vascular pressure.

Noteworthy is the correlation in liver disease of a higher wedged hepatic venous pressure with the presence of jaundice. Each of the cirrhotic patients who was jaundiced had a very large liver which was presumably due in part to fatty metamorphosis (this was demonstrated microscopically in some instances). It seems reasonable that intrahepatic obstruction under these circumstances would be greater, producing a higher portal pressure. This is in line with Dock’s observation\(^\text{29}\) on the decreased perfusability of a large fatty liver.

The correlation between increased wedged hepatic venous pressure and an elevated blood volume in some instances of Laennec’s cirrhosis is interesting, but not unexpected, since a similar volume-pressure relationship exists in the vascular system under other conditions.\(^\text{38, 39}\) The meaning of this correlation is, however, difficult to ascertain. It is conceivable that the increased blood volume contributes to an increased pressure analogous to the changes found in a distended closed section of vein and in the pulmonary circuit. In many instances, the greatly dilated portal venous bed would seem to accumulate the increment in blood volume without per se producing portal hypertension. The most likely explanation of the volume-pressure relationship seems to be that they are not causally related, but merely coexisting manifestations of a severe stage of the same disease.

**Summary**

1. The technic of occlusive venous catheterization of the liver in man is described.
2. The pressure obtained in an occluded small hepatic vein has been termed the wedged hepatic venous pressure.
3. In cats, the wedged hepatic venous pressure and portal venous pressure are essentially the same.
4. In 29 observations on 27 patients with Laennec’s cirrhosis, the wedged hepatic venous pressure has, in each instance, been higher than any observation in 18 control subjects, thus establishing diagnostic significance for an elevated pressure.
5. The procedure has been of practical value in the separation of cirrhotic from noncirrhotic (extrahepatic) portal hypertension.
6. The wedged hepatic venous pressure is particularly high in those cirrhotic subjects with esophageal varices and with jaundice, but the degree of elevation cannot be correlated with the presence or absence of ascites.

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**Summario in Interlingua**

Le technica del occulsive catheterisation venose eseva applicate al hepate. Le pression mesurate in un occludite minor vena hepatic (designate como le “cuneate pression hepato-venose”) se monstrava ben correlatoate con le pression del vena portal in cattos. In homines, un elevate pression cuneate in association
con un normal pression venose central es
diagnostic pro cirrhosis. Ist facto se ha
provate de valor in le differentiation de intra-
hepatic e extrahepatic hypertension portal. Le
plus alte cuneate pressiones venose hepatic
que occurre in cirrhosis es correlationate con
le presentia de varices o ictero sed non de
ascites.

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W. JAPE TAYLOR and J. D. MYERS

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