THE TREATMENT OF ASCITES BY SIDE TO SIDE PORTACAVAL SHUNT*

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SEVERAL attempts have been made to treat ascites by surgical procedures but little success has been attained. Time does not permit a review of this history. It is important to point out, however, that there has been rather unusual agreement that portacaval shunts are of little value in the treatment of ascites and, indeed, they have been considered by some to be contraindicated. This opinion is based on the consideration that portal hypertension, *per se*, is not the cause of ascites and, therefore, splanchnic decompression of the portal system would have little effect on ascites. Clinical observations have largely borne out this contention. It should be mentioned in this regard that portacaval anastomosis is usually performed by the end to side technique in which case the portal vein is severed near the liver, its hepatic end tied and the splanchnic end anastomosed to the vena cava. This operation effectively decompresses the peripheral portal system but should have little effect upon reducing ascites if hypertension is not the cause of ascites.

A review of the mechanisms which may be involved in the production of ascites in hepatic cirrhosis leaves us with the impression that there are four possible causes. These are: 1) inflow block (portal hypertension), 2) decreased oncotic pressure of the blood associated with a low value for serum albumin; 3) retention of water and sodium associated with abnormalities of hormonal systems; and 4) outflow block (obstruction to the egress of blood from the liver). Originally, it was

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assumed that ascitic fluid arose from the splanchnic bed by transudation, the local mechanical derangement being a combination of portal hypertension and decreased osmotic pressure of the blood. This etiology is not tenable in view of certain known facts, namely, that high degrees of portal hypertension can exist without ascites in patients with cirrhosis of the liver and also with extrahepatic obstructive portal lesions. Also, there is no constant correlation between the blood colloidal osmotic pressure and the rate and volume of peritoneal fluid accumulation. The abnormalities noted in salt and water metabolism and in certain hormonal systems serve to give further information about the general metabolic effects of cirrhosis of the liver but do little to explain the localization of fluid in the peritoneal cavity. Ascites is a local phenomenon and a local mechanical derangement must be present to account for it. The more recent concept of outflow block postulates that there is a mechanical obstruction to the egress of blood from the liver, a diminution in normal efferent flow through the hepatic veins, the local mechanical derangement being a postsinusoidal block leading to venous congestion of the liver. McIndoe[1] showed by injection studies that in cirrhosis of the liver there is a decrease in both the portal venous system and the hepatic venous system within the liver. More recently, Madden, Lore, Gerold and Ravid[2] have demonstrated by injection studies of the hepatic vasculature that in cirrhosis of the liver associated with ascites there is an absolute decrease in the intrahepatic venous system. This relatively greater decrease in the hepatic venous system is not seen in cirrhotic livers taken from patients who did not have ascites. Kelty, Baggenstoss and Butt[3] have demonstrated that the regenerated hepatic lobule is another important cause of obstruction of the hepatic venous system. These observations are very important in establishing the fact of outflow block.

Experimental studies also support the concept that outflow block is the primary cause of ascites and that ascitic fluid is hepatic lymph. It is known that ascites can be produced consistently in dogs by constricting the vena cava above the diaphragm. The ascites which follows this procedure often is not associated with hypoproteinemia or marked elevations of the portal pressure in the initial stages. Clinically, the association of ascites with constrictive pericarditis and the Budd-Chiari syndrome indicates that the obstruction to the venous outflow does not necessarily have to be intrahepatic but may be anywhere between the termination

of the portal system and the right side of the heart. The actual origin of the fluid is probably sinusoidal where obstruction and venous congestion of the liver promote a high filtration pressure with the excessive formation of hepatic lymph which extravasates into the peritoneal cavity. Grindlay, Flock and Bollman[4] have reported on the dilatation of lymphatics in the porta hepatis and measured the markedly increased lymph flow pressure in dogs made ascitic by constriction of the vena cava. Hyatt and Smith[5] have shown the close relationship between the protein content of ascitic fluid and hepatic lymph in dogs with ascites. Many surgeons have noted the outpouring of fluid associated with dissection around the porta hepatis on patients undergoing portacaval shunt operations. This phenomenon is seen especially after the portal vein has been interrupted and during the time of performing the anastomosis, an important finding supporting the concept of outflow block. Mallet-Guy, Devic, Feroldi and Desjacques[6] in France have produced hydrothorax in dogs by constricting the supradiaphragmatic portion of the inferior vena cava and placing one lobe of the liver in the pleural cavity above the diaphragm. This work further supports the belief that ascites is hepatic lymph. The actual route of the intrahepatic lymph flow has not been completely determined. However, available data support the theory that consequent to congestion of the liver, there is engorgement of the subcapsular lymphatics and the lymphatics of the porta hepatis with extravasation of lymph from engorged lymphatics into the peritoneal cavity.

Our observations support the concept of outflow block as the basic mechanical factor in causing ascites. Aside from paracentesis and operations designed to drain ascites from the peritoneal cavity (unsuccessful to date) there are two rational approaches to the surgical treatment of ascites: 1) Reduction of hepatic inflow or 2) the production of additional outflow. Rienhoff and Woods[7] in 1953 proposed that ligation of the hepatic artery should be beneficial in the treatment of ascites. Assuming that ascites is secondary to hepatic venous obstruction, reduction in the afferent flow would serve to decompress the liver. Hepatic artery ligation has been given a trial but has not been accepted as a sound procedure because the consequences of arterial ligation negate the beneficial effects upon the ascites. Others have sought to reduce ascites by diverting the portal inflow. Eck originally devised his fistula as a means of treating ascites. Eisenmenger and Nickel[8] in 1956 reported

five cases in which ascites was present and in which end to side portacaval shunt had been done with apparent beneficial effect upon the ascites. This experience has not been universal and most surgeons have rejected the idea of portacaval shunt as having a predictable effect upon ascites. Nature, however, has provided a means by which it is possible to divert portal inflow and, simultaneously, a portion of the arterial inflow without incurring the disadvantages of total arterial occlusion. Two types of intrahepatic arterial portal anastomoses exist, arterioportal, which lie within the portal space, and arteriosinusoidal, which lie outside the portal space. Herrick[9] showed in 1907 that in cirrhosis of the liver the communications between the arterial bed and the portal system increased with the development of new abnormal arterioportal venous anastomosis. In cirrhotic livers, he showed that portal pressure rose markedly as hepatic arterial pressure was increased. He also showed that when the hepatic artery was perfused, there was a free flow through both hepatic and portal veins. In the normal liver, the portal return-flow was less in volume than from the hepatic vein. In the markedly portal cirrhotic liver, the portal return-flow was always greater than from the hepatic vein. We have found that upon occluding the portal vein in patients with ascites and measuring the portal pressure on the liver side of the occluding clamp, the recorded pressure will often be surprisingly high. In several of our cases, it has been over 250 mm. of water.

These findings suggest that the portal vein can act as a decompressive outflow route in certain circumstances. It occurred to us that if we could arrange to decompress the liver, using the portal vein as an exit for the excess of blood trapped within the liver, we might affect the formation of ascites. In an end to side portacaval shunt the distal end of the portal vein is ligated. However, in a side to side portacaval shunt the portal vein is left in continuity and both the splanchnic end and the hepatic end are allowed to drain into the low pressure caval system. Thus, a portion of the hepatic artery blood can drain into the vena cava via the natural intrahepatic arterial-portal and the artificially created portacaval anastomoses. The inflow to the congested liver would be further decreased by that amount of portal blood from the splanchnic bed which would be diverted into the vena cava by the same side to side portacaval anastomosis.

We, therefore, have performed side to side portacaval shunt in six

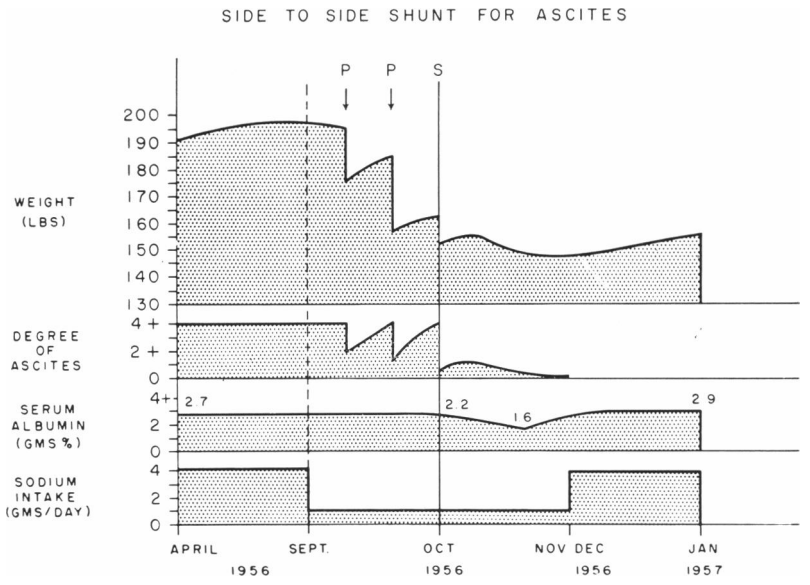

Figure 1.

Graphic representation of control of ascites after side to side portacaval shunt in a 47 year old male patient with alcoholic cirrhosis of the liver. P., paracentesis. S., portacaval shunt. Note the absence of ascites after portacaval shunt in the presence of very low serum albumin values. Also, the resumption of an intake of sodium chloride of over 4 grams did not cause re-accumulation of ascitic fluid.

patients with intractable ascites. In each case, the ascites has been relieved, although the follow-up has been short (eight months to two months). One patient was inadvertently given large doses of ammonium chloride at home for the treatment of leg edema and succumbed from ammonia intoxication. The others have done well. Their ascites has not re-accumulated and their liver function has improved. This group of patients had poorer liver function than patients usually subjected to portacaval shunt for the relief of hypertension and prevention of bleeding. Figure 1 graphically represents the course of one patient subjected to side to side shunt for ascites. It was particularly noteworthy that ascites did not re-accumulate even though the values for serum albumin were very low in the postoperative period (Figure 1). Of considerable significance is the fact that the five surviving patients have resumed a normal salt intake without precipitating a recurrence of ascites.

This communication is a preliminary report on a short-term follow-up of side to side portacaval shunt in the treatment of ascites, presented because the clinical results seem convincing to us and because the rationale seems adequate for a trial of this operation on other cases. Additional clinical investigation can be carried out on these patients before and after the shunt procedures. We have some indication that their ability to excrete sodium is considerably enhanced but more documentation is needed for establishing this as a fact.

Ascites is, of course, to be controlled by medical measures in all patients. Careful management can usually accomplish this. There are cases of intractable ascites which usually manifest themselves after a month or six weeks of medical treatment. Side to side shunt may be an adjunct to the treatment of such patients in the light of our recent experience with it. It also should prevent bleeding from varices if they be present since it simultaneously decompresses the peripheral portal system.

A review of the literature on side to side shunt reveals that although no one has recommended this procedure for the treatment of ascites alone, a few instances of relief of ascites have followed the procedure. Large, Johnston, and Preshaw[10] report three cases of ascites in which side to side shunt was performed with some amelioration of the ascites. Theron and Allan[11] used an "H" shunt, in which a vein graft is employed between the portal vein and vena cava, in five patients with portal hypertension and bleeding. Two of these patients had ascites and were relieved. Again, however, these authors did not recommend the operation for ascites alone although the "H" graft technique was designed by them to decompress the liver, a rationale similar to ours.

Some investigators state that a portacaval shunt has the disadvantage of depriving the liver of blood. However, it is well known that in many cases of cirrhosis with well developed collateral circulation, much of the portal blood is shunted around the liver. In cirrhosis with ascites, the added factor of outflow block is present and there is evidence that in certain of these cases the flow in the terminal part of the portal vein system may actually be retrograde, depending on the degree of outflow block and arterial mixing. In regard to depriving the liver of arterial blood by such a shunt, it can be said that the actual amount of blood needed by the liver is not known. It is agreed, however, that congestion of the liver is not beneficial. The amount of arterial deprivation af-

forded by this operation is difficult to determine because of the nature of the intrahepatic anastomoses but the amount of retrograde flow is dependent on the degree of outflow block and is to a great extent a self-regulating mechanism. Side to side shunt, therefore, offers a means of decompressing the liver which imitates nature. It shows promise in the treatment of ascites.

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