A New Method for Complete Hepatic Venous Occlusion in Dogs. (26203)

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Recent evidence has implicated obstruction of hepatic venous outflow as one of the important factors in pathogenesis of ascites in human cirrhotics(1). Previous experimental methods of producing ascites in dogs have utilized partial constriction of the supradiaphragmatic inferior vena cava, but with these methods, the effects of pure hepatic venous outflow cannot be assessed, since renal and adrenal venous hypertension are also produced. A 2-stage method has been devised for production of complete hepatic venous obstruction without these associated undesirable phenomena.

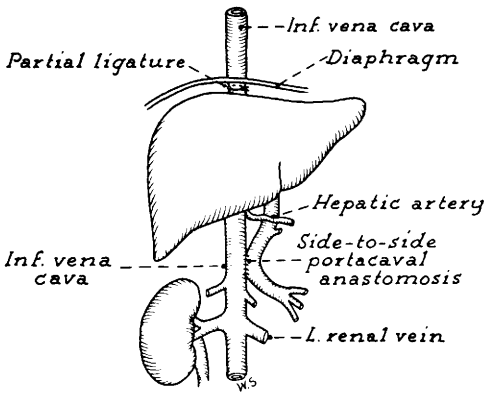
Methods. Healthy adult mongrel dogs were anesthetized with intravenous pentabarbital sodium. The first stage operation was carried out through a long right subcostal abdominal incision. A long side-to-side portacaval anastomosis was made and the inferior vena cava between liver and diaphragm was constricted to approximately ⅔ its diameter with a #0 silk ligature (Fig. 1).

Following a 2 to 3 week recovery period, the second stage operation was performed through a long thoracoabdominal incision utilizing very light anesthesia induced by a constant slow drip of a 2½% solution of sodium thiopental in isotonic saline. The inferior vena cava was transected immediately above the previous portacaval shunt. The hepatic side of the transected cava was doubly ligated and the distal end was anastomosed end-to-end with a crimped Dacron graft of suitable size. The supradiaphragmatic inferior vena cava was doubly ligated, transected, and the cardiac end anastomosed end-to-end with the upper end of the Dacron graft (Fig. 2). During this upper anastomosis, the descending aorta was occluded with tapes to prevent distal pooling. The graft was allowed to fill with blood by removing the vascular clamp on the abdominal vena cava, thus forcing any trapped air through the interstices of the graft before the upper vascular clamp was removed. The diaphragmatic incision was closed anteriorly only so that constriction of the graft was kept to a minimum.

Liver biopsies were obtained at each of the 2 operations and at intervals after the second stage. Girth and weight were recorded preoperatively and at weekly intervals after operation. Total protein, serum albumin, serum globulin, thymol turbidity, and bromsulfalein excretion were measured preoperatively and at intervals postoperatively in surviving animals. Venograms were obtained *via* the femoral vein to determine vena caval and graft patency in second stage survivors.

Results. Two-stage hepatic venous occlusion was accomplished in 18 dogs. Ten survived 18, 27, 30, 31, 67, 84, 101, 113, 150, and 270 days after the second stage operation. One healthy survivor died at 270 days during an exploratory celiotomy. This animal had been in excellent health throughout, had patent graft and portacaval shunt, no ascites, normal liver function tests, and a normal liver biopsy. Two animals died 3 and 5 days after second stage operation of empyema. The remaining 6 dogs died of severe hypotension within the first 24 hours after the second operation.

Six of the 10 long-term survivors had patent Dacron grafts and portacaval shunts as demonstrated at autopsy and by pre-mortem venograms. Only one of these dogs developed ascites. This occurred 90 days after the second-stage operation and consisted of 5250 cc at autopsy on the 101st day. The 4 remaining long-term dogs developed ascites at varying intervals after operation, the onset coinciding with thrombosis of the Dacron graft as demonstrated by venography before and after the ascites appeared. The only consistent change in liver function was rapid decline of serum albumin after the development of ascites when it occurred. All of the 6



FIRST STAGE HEPATIC VEIN OCCLUSION
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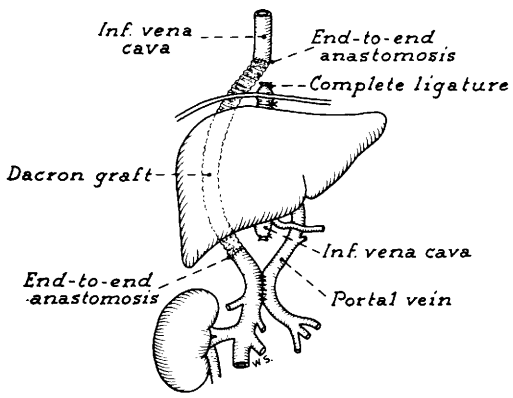

SECOND STAGE HEPATIC VEIN OCCLUSION
②

FIG. 1 and 2.

animals with patent grafts except the one which developed ascites had normal liver biopsies. The latter and the 4 dogs with thrombosed grafts showed marked sinusoidal congestion and lymphatic dilatation in their liver biopsies.

Discussion. The high immediate mortality rate caused by hypotension is unexplained, but is similar to the early deaths encountered by Child, *et al.*, when performing reversal of hepatic circulation(2). In all animals developing thrombosis of the Dacron grafts, massive progressive ascites developed, presumably because the portal vein could no longer function as an outflow tract for the liver(3). Only one of the 6 animals with patent Dacron vena cava grafts and portacaval shunts developed ascites. The reason for this one exception is unclear, particularly since the serum albumin remained relatively normal in this dog.

It should be recognized that although complete hepatic venous occlusion is accomplished anatomically by this operation, functionally, hepatic arterial blood is being shunted retrograde into the portal vein and thence into the vena cava through the portacaval shunt. The presence of the portacaval shunt is necessary since dogs do not survive acute hepatic venous occlusion without a shunt. The fact that most of the long term survivors did not develop ascites attests to the efficacy of such a shunt in decompression of the liver and may have clinical inferences supporting the concept that in patients with severe hepatic venous outflow obstruction manifested predominantly by ascites, side-to-side portacaval shunt rather than end-to-side shunt may be the procedure of choice. Since acute occlusion of the portacaval shunt in these dogs is uniformly fatal, further studies on the effects of gradual occlusion of the shunt or the portal vein cephalad to the shunt are being carried out.

Summary. 1. A 2-stage operation for production of pure complete hepatic venous outflow obstruction has been devised. Ten of 18 dogs survived from 18 to 270 days after operation. Crimped Dacron prostheses function satisfactorily in the low pressure venous system. 2. This preparation is presented as a satisfactory means of studying hemodynamic, hormonal, and chemical factors affecting development of ascites.

1. Madden, J. L., Lore, J. M., Gerold, F. P., Ravid, J. M., *S. G. and O.*, 1954, v99, 385.

2. Child, C. G., McDonough, E. F., Des Rochers, G. C., *Ann. Surg.*, 1959, v150, 445.

3. McDermott, W. F., Jr., *New Eng. J. Med.*, 1958, v259, 897.

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