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MAJOR HEPATIC RESECTION AND PORTAL PRESSURE

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IN RECENT REPORTS in the surgical literature, an increase in portal pressure following major resections of the liver has been described, and this elevated pressure has been related to an increased morbidity and mortality. However, in the University of Colorado series of 30 liver resections—including 17 trisegmentectomies, five right lobectomies, five left lobectomies and three left lateral segmentectomies—all performed without mortality, no evidence of splanchnic sequestration was observed. To document this clinical experience, portal pressures were obtained both before and after resection on three consecutive patients undergoing hepatic trisegmentectomy in whom 80 per cent or more of the liver was removed.

REPORTS OF PATIENTS

PATIENT 1. This 57 year old male had a large tumor of the liver found on routine physical examination three weeks prior to admission. Angiography revealed a large tumor blush in the right lobe of the liver, and on 20 March 1975, he underwent hepatic trisegmentectomy. After initial mobilization, portal pressure measured directly through a portal vein cannula was 180 millimeters of saline solution.

He then underwent an anatomic trisegmentectomy; the resected specimen weighed 1,200 grams. The estimated blood loss was 1,500 milliliters which was replaced with 4 units of whole blood plus 2 liters of lactated Ringer's solution. At no time during the procedure was he hypotensive, and the central venous pressure remained in the range of 17 to 21 centimeters of saline solution. During this six hour operation, there was never any evidence of splanchnic engorgement. Approximately 45 minutes after the completion of the resection, the portal pressure measured 172 millimeters of saline solution. The postoperative course was uncomplicated, and he was discharged on the 19th postoperative day.

PATIENT 2. This 40 year old female was considered for hepatic trisegmentectomy when she was found to have invasive carcinoma of the gallbladder after cholecystectomy for cholelithiasis. A hepatic trisegmentectomy was performed on 29 April 1975. The initial portal vein pressure obtained before extensive dissection was 200 millimeters of saline solution. The entire procedure lasted ten hours, with an estimated blood loss of 3,500 milliliters. This was replaced with 7 units of whole blood. In addition, the patient received 1,800 milliliters of crystalloid and 200 milliliters of fresh frozen plasma. No hypotension was encountered during the procedure, and the central venous pressure remained relatively constant. The resected specimen weighed 1,295 grams and contained a solitary metastasis deep within the parenchyma at the junction of the true right and left lobes.

After completion of the resection, the portal pressure measured 195 millimeters of saline solution. At no time during the procedure was splanchnic engorgement observed. She recovered uneventfully, but the disease has recurred one and one-half years after the operation was performed.

PATIENT 3. This 12 year old girl was referred from Germany with the diagnosis of sarcoma of the liver. Following a confirmatory work-up, an hepatic trisegmentectomy was performed on 24 May 1975. Exploration revealed a bulky tumor was replacing most of the right lobe of the liver and was extending slightly to the left of the falciform ligament. Before any significant dissection was performed, the portal vein was cannulated, and a pressure of 160 millimeters of saline solution was obtained.

Because of the bulkiness of the tumor, dissection proved difficult, with an estimated blood loss of 3,500 milliliters and a total operating time of ten hours. The systolic blood pressure remained fairly constant at 110 millimeters of mercury until the time of hepatic transection when it fell to 70 millimeters of mercury. This fall was associated with brisk bleeding from the liver bed and responded rapidly to volume replacement. The central venous pressure, which had also remained constant throughout the procedure, likewise fell at this time but also responded rapidly. Total fluid replacement was 3,500 milliliters of whole blood plus an additional 3 liters of crystalloid solution. The resected specimen weighed 1,760 grams and represented approximately 80 per cent of the liver of this patient.

One hour following the completion of the resection, when blood volume had again normalized, the portal vein pressure measured 154 millimeters of saline solution. At no time was there clinical evidence of engorgement of the splanchnic bed. The postoperative course, although protracted because of persistent bile and fluid drainage from the wound, was benign, and she made an uneventful recovery.

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DISCUSSION

Reports of splanchnic congestion following ligation of the portal vein and partial hepatectomy in animals have caused understandable concern among clinical investigators. The observation that splanchnic pooling occurred following occlusion of the portal vein was first made by Oré (7) in 1856. Thereafter, numerous other investigators, including Bernard (2) and Lautenbach (5), reported similar results, and by the early 1900s, the hypothesis of splanchnic pooling, decreased venous return with a decrease in effective circulating volume, was accepted as the mechanism of death in these animals.

In dogs, rabbits and rats, occlusion of the main portal vein invariably caused death. Investigators assumed that incomplete interruption of portal venous flow by partial hepatectomy would also partially impede splanchnic venous return. With the dog being used as a model, Aronsen and colleagues (1) reported a 33 per cent reduction in cardiac output and a 50 per cent reduction in stroke volume following a 50 per cent hepatectomy. In other studies, sustained portal hypertension was reported following substantial liver resections in dogs and rats.

After reviewing hepatic resections in 38 humans, Stone (9) emphasized the importance of splanchnic bed pooling and advised transfusing volume that exceeded the calculated loss to prevent shock following a 30 per cent hepatectomy. Stone (9) further suggested that splanchnic sequestration played a major role in the early deaths following major hepatic resection.

The hypothesized physiologic mechanisms for splanchnic sequestration were the reduction of hepatic outflow caused by a reduction in the total number of hepatic veins or a diminution in the hepatic mass available for egress of splanchnic flow. However, the ability of the liver to handle rather marked alterations of hepatic flow without changes in portal pressure is well known. Results of in vivo studies by Bloch (3) have shown that only 60 per cent of sinusoids are functioning in the steady state. This would theoretically allow for some loss of liver mass without alteration in portal pressure. That the normal liver can withstand even greater splanchnic flows was pointed out by Hopkinson and Schenk (4), who noted a 76 per cent increase in portal flow without evidence of increased portal pressure following a test meal.

In addition, it is dangerous to extrapolate from the dog to man with regard to splanchnic hemodynamics. For example, although acute occlusion of the portal vein is uniformly fatal in the dog, it is not immediately fatal in monkeys, according to Milnes and Child (6), or in other species that have true retroperitoneal portions of the gastrointestinal tract and associated organs.

Results of our previous experience (8) have shown that, with careful anatomic dissection and the absence of clinical disease in the nonresected liver remnant, major hepatic resections produce no clinical evidence of increased portal pressure or splanchnic sequestration.

SUMMARY

Although other reports have stressed the inevitability of portal hypertension and splanchnic pooling following major liver resections, clinical observations during 30 major hepatic resections and measurements of portal pressure in three consecutive trisegmentectomies fail to support this contention. If the remaining liver is normal and careful anatomic dissection is used, major resections can be performed without inducing portal hypertension.

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