

# Major Hepatic Vein Ligation Necessitated by Blunt Abdominal Trauma

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An unusual case involving avulsion of the middle and left hepatic veins secondary to blunt trauma is presented. Operative management of the injury resulted in ligation of both veins without resection of the parenchyma acutely deprived of venous outflow. The postoperative evaluation and review of the literature indicate that liver tissue will survive acute interruption of a major portion of its venous outflow. This case demonstrates that if after ligating major hepatic vein, the condition of the obstructed parenchyma stabilizes or improves under direct observation and if the condition of the patient does not deteriorate, resection may not be required.

**D**ISRUPTION of major hepatic veins is one of the most challenging injuries confronting the surgeon. Several recently described adjunctive techniques have improved the management of this grave injury.<sup>1,10,12,13,15</sup> Ideally, intraluminal bypass of the retro-hepatic vena cava allows isolation and repair with restoration of flow within the injured vein. When reconstruction is technically impossible, ligation of major hepatic veins is required. Consideration must then be given to resection of the parenchyma drained by the interrupted hepatic veins. Several authors have stated that resection of hepatic tissue acutely deprived of hepatic venous outflow is mandatory to prevent complications secondary to parenchymal necrosis.<sup>3,4,6,12,15</sup> The following case and review of the literature suggests that hepatic tissue will survive despite acute interruption of its venous drainage.

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## Case Report

On July 6, 1974 a 19-year-old man was seen at the Naval Regional Medical Center, Philadelphia, 4 hours after an automobile accident. He had obvious facial burns and abdominal pain. Vital signs were: blood pressure 90/60, pulse 110, respirations 24. Diffuse abdominal tenderness was noted. Abdominal paracentesis demonstrated non-clotting blood. After appropriate preparation, exploration was initiated via an upper midline abdominal incision. Approximately two liters of blood were found along with an actively bleeding 6 cm laceration on the antero-superior surface of the liver slightly right of the falciform ligament. A large clot was evacuated from the hepatic rent and several actively bleeding arterial branches were suture ligated; however, dark blood continued to accumulate in the depths of the laceration. Massive hemorrhage resulting from attempts to visualize the source of bleeding suggested retro-hepatic vena cava and/or hepatic vein disruption. The porta hepatis was occluded and the bleeding tamponaded with multiple laparotomy packs. Median sternotomy was performed followed by introduction of a 36F chest catheter into the retro-hepatic vena cava via the right atrial appendage in the manner described by Schrock.<sup>12</sup>

After rapid transfusions through the large bore catheter had resulted in effective resuscitation, the diaphragm was split to the vena cava and the tamponade slowly removed. The rapidity of blood loss was markedly reduced. Completion of the traumatic hepatic fracture allowed exposure of the anterior surface of the retro-hepatic vena cava. Avulsion of the middle and left hepatic veins was found. Repair was impossible and control required ligation of the middle and left hepatic veins. The defect in the vena cava was closed with a continuous suture.

After control of the hepatic vein injury further blood replacement resulted in stabilization of the vital signs. Consequently, the intraluminal shunt was removed and 90 minutes of intermittent porta hepatis occlusion terminated. Immediately after ligation of the middle and left hepatic veins the anatomical left lobe became cyanotic

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Submitted for publication June 14, 1975.

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TABLE 1. Liver Function Tests

Time After Injury	SGOT Normal (0-40 IU)	Alk. Phos. Normal (9-35 IU)	Total Bilirubin Normal (0-1.5 mg%)
1 day	130		0.8
4 days	110	24	0.7
10 days	58	31	1.1
14 days	72	34	0.6
28 days	34	35	0.7
40 days	44	49	0.6
10 months	34	35	0.6

and turgid; however, 15-30 minutes after restoration of flow within the porta hepatis, decrease of turgor and improved color were noted. Left hepatectomy was not performed because of the parenchymal improvement and the added morbidity of anatomical left hepatectomy in a patient who had sustained multiple transfusions (25 units) and prolonged periods of hypotension and oliguria. Suitable sump drainage was established and the incisions closed.

The postoperative course was relatively uncomplicated. A transient right pleural effusion responded to thoracentesis. Minor liver function abnormalities were noted, but were never of major concern. The liver function tests done are summarized in Table 1. Ten months post injury the patient has no clinically detectable hepatic abnormalities and is normally active.

*Postoperative Contrast Studies:* Serial hepatic scans were used as an aid in the postoperative evaluation of the patient. The first scan, as seen in Fig. 1, was performed 10 days after surgery; there was non-visualization of the left lobe and poor uptake within the right lobe. The second and third scans (Figs. 2 and 3) done 24 and 50 days post injury demonstrated near normal perfusion of the right lobe but there was delayed and slightly decreased perfusion of the left lobe. Ten months after operation the final scan (Fig. 4) was essentially normal except for a small wedge-shaped defect superiorly.

The only abnormalities demonstrated by hepatic arteriography (Fig. 5), 74 days post injury, were truncation of several arterial branches within the medial segment of the left lobe corresponding to those ligated at operation. Though not shown, the superior mesenteric artery injection during the same study delineated a patent portal vein.

Hepatic venography (Fig. 6) 93 days post injury illustrated patency

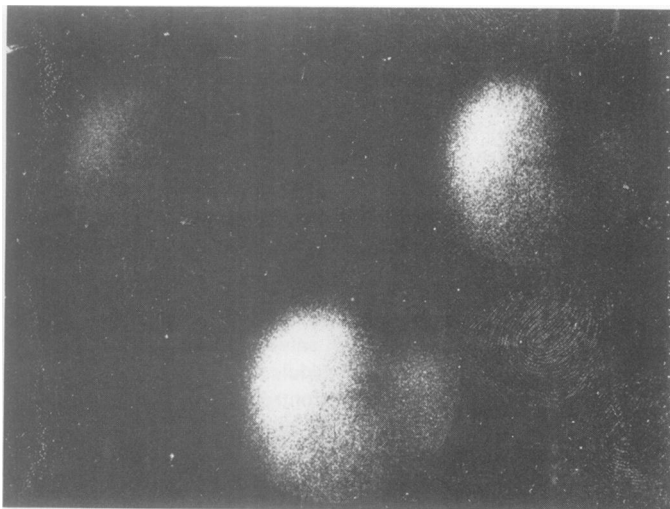


FIG. 1. Liver scan 10 days after injury.

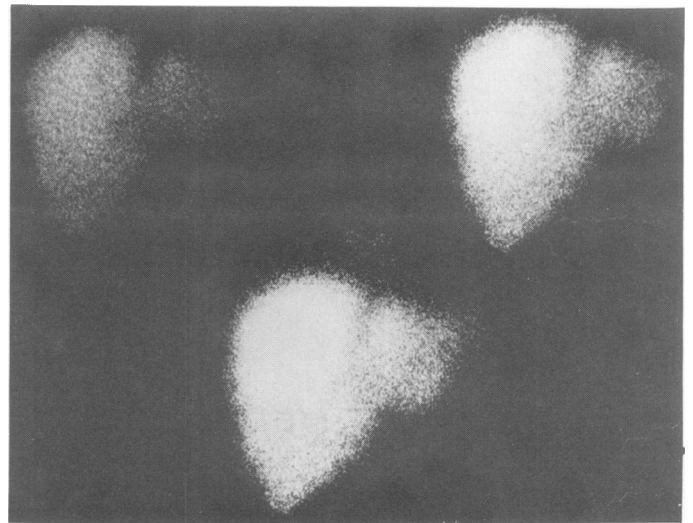


FIG. 2. Liver scan 24 days after injury.

of the right hepatic vein with non-visualization of the middle and left hepatic veins.

### Discussion

Routine gross or microscopic anatomical studies fail to demonstrate hepatic vein intercommunications.<sup>3,4</sup> However, corrosion cast technique does illustrate hepatic vein intercommunication.<sup>5,9,14</sup> Corrosion cast studies may be inaccurate because of the unphysiological pressures used to exhibit intraparenchymal hepatic vein collaterals. However, ligation of major hepatic veins, with continuation of portal vein and hepatic artery inflow, may elevate the pressure within the obstructed hepatic vein to levels that approximate those attained with the corrosion cast technique consequently opening intraparenchymal hepatic vein collaterals.

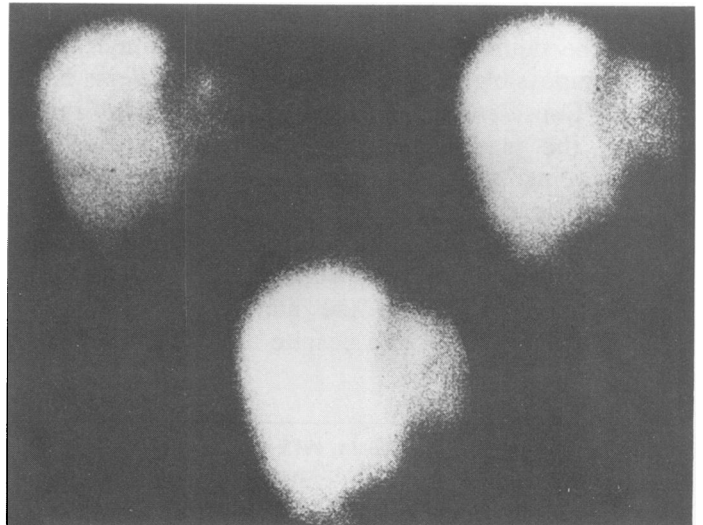


FIG. 3. Liver scan 50 days after injury.

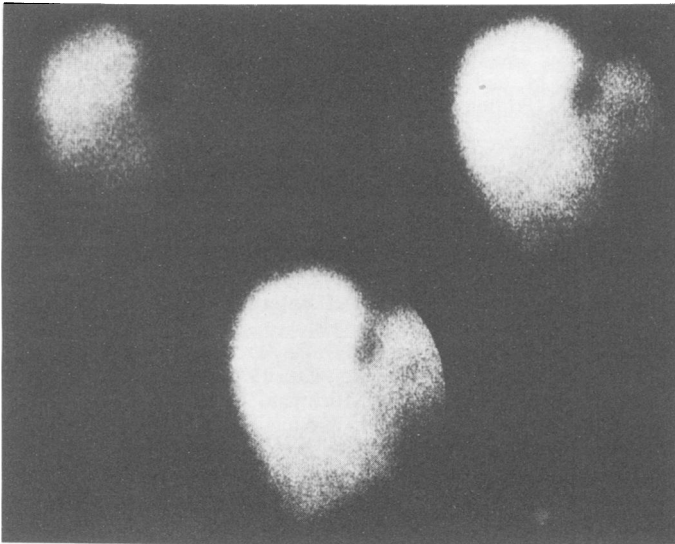


FIG. 4. Liver scan 10 months after injury.

Studies using pig liver, which closely resembles the vascular anatomy of the human liver, do demonstrate collaterals between the left and right hepatic veins when one or the other is ligated.<sup>7</sup> Post mortem venography has been used by Hirooka<sup>5</sup> to illustrate left to right intra-parenchymal hepatic vein collaterals in a human liver with membranous obstruction of the left hepatic vein.

Canine studies indicate that ascites resulting from near total occlusion of hepatic venous outflow will resolve if portal pressure does not acutely rise to lethal levels.<sup>11</sup> Resolution of ascites is associated with return of portal pressure to normal suggesting that decompression of hepatic venous outflow obstruction is achieved via collateral circulation.<sup>11</sup>

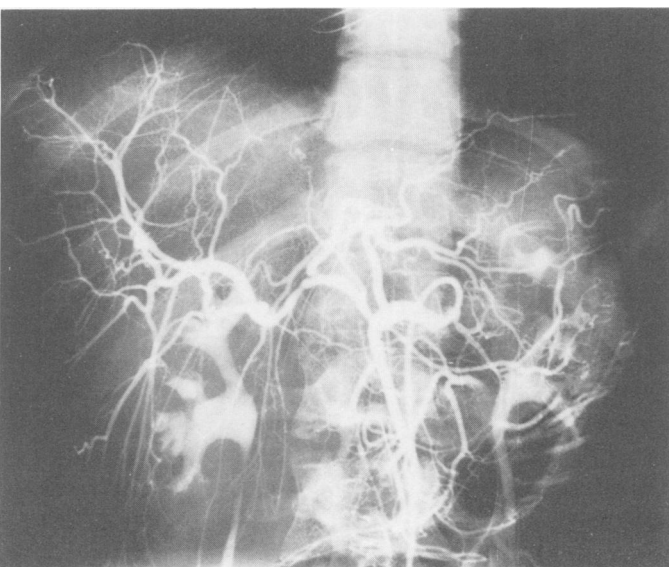


FIG. 5. Hepatic arteriogram 74 days after injury.



FIG. 6. Hepatic venogram 93 days after injury.

Autopsy studies of patients with neoplastic occlusion of major hepatic veins frequently fail to demonstrate ascites, illustrating the capacity of human liver with partially obstructed hepatic venous outflow to develop collaterals.<sup>8,14</sup>

In 1966 Feldman<sup>2</sup> reported a case of penetrating liver trauma resulting in left hepatic vein ligation without parenchymal resection. The patient survived with no adverse sequelae. Objective information relative to the function of the injured liver was not presented.

The postoperative studies in the present case demonstrated patency of the hepatic artery, portal vein and right hepatic vein, and occlusion of the middle and left hepatic veins. Intrahepatic collaterals were not visualized between the left and right hepatic system. Inability to illustrate intra-parenchymal collaterals probably reflects failure of hand injection venography to generate sufficient pressure to cause retrograde flow from the patent right hepatic venous system across collaterals to the obstructed left system.

The abnormalities illustrated by the liver scan, 10 days

and 24 days post injury, may be interpreted as poor distribution of isotope related to compromised hepatic venous outflow. Subsequent near normality of isotope distribution shown in the later scans probably reflects improved hepatic venous drainage secondary to collateral development.

### Conclusion

Compulsory hepatic resection after major hepatic vein ligation has been advised by most authors.<sup>3,4,6,12,15</sup> The present case suggests that if after ligating a major hepatic vein the condition of the obstructed parenchyma stabilizes or improves under direct observation and if the general condition of the patient does not deteriorate, resection may not be required.

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