ANNALS OF SURGERY

Vol. 185 June 1977 No. 6

Selectivity in the Management of Hepatic Trauma

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Retrospective review of 178 consecutive patients who sustained hepatic injuries confirms that selective application of hepatic artery ligation is an efficient means of definitive hemostasis. Only two of 20 immediate deaths resulted from hepatic parenchymal hemorrhage. Over-all mortality (20%) was not significantly increased where hepatic artery ligation was used. Sepsis was the most frequent complication after severe hepatic trauma.

DESPITE THE RELATIVE protection from external injury offered by the rib cage, the liver is frequently injured. Furthermore, the structure and consistency of liver tissue is unsuitable for spontaneous hemostasis following parenchymal disruption. The physiologic impact of loss of a major portion of hepatic tissue is significant, but hepatic dysfunction commonly follows operation for liver injury regardless of whether hepatic resection is performed.

Although the mortality rate from liver trauma has declined steadily since the review by Madding, Lawrence, and Kennedy[13] following World War II, liver injuries continue to present vexing problems of intraoperative hemostasis and postoperative complications. Recent series cite mortality rates from 13 to 15%.[2,6] Mortality for acutely bleeding lesions necessitating hepatic lobectomy ranges from 40 to 60%. Reports by Trunkey and associates[19] and by Lucas and Ledgerwood[11] emphasize the importance of shock and parenchymal hemorrhage as causes of death in the patient with liver injuries. Post-operative complications in survivors are similarly challenging, with infection and sepsis-related organ failure being frequent and lethal.

The operative management of hepatic trauma has varied with the agent causing injury and with the surgeon's assessment of the extent of injury. Combinations of drainage and direct hemostasis have been used for most mild to moderate injuries. Bursting injuries with extensive destruction of parenchyma and massive hemorrhage often have required formal hepatic lobectomy. Mortality following hepatic lobectomy for trauma remains high. The search for an alternative means of hemostasis in such extensive injuries has been hampered by several factors. The actual extent of parenchymal destruction may be obscure in some injuries. Sandblom[16] documented delayed biliary tract hemorrhage (hemobilia) following destruction of hepatic tissue and formation of an intraparenchymal cavity and emphasized the relatively benign appearance of the outer surface of the liver at initial laparotomy.[6] Variability in the frequency of associated injuries and the incidence of postoperative complications, such as late bleeding and infection, have led to uncertainty concerning the relationship of management techniques to postoperative course. Moreover, co-existing extrahepatic injuries place an added burden on patient and surgeon. Mortality escalates when formal hepatic lobectomy accompanies other major organ system injuries requiring extensive operation.

Interest in selective hepatic dearterialization as a means of initial hemostasis began to evolve with the suggestion by Madding in 1954 that this modality might be useful.[12] Subsequent experience has confirmed the utility of hepatic artery ligation before elective hepatic operation.[7] Extensive studies have documented the prevailing anatomic pattern of the hepatic vasculature and potential collateral channels.[5] Clinical experience

Presented at the Annual Meeting of the Southern Surgical Association, December 5–8, 1976, Palm Beach, Florida.

Submitted for publication, December 10, 1976

TABLE 1. *Grades of Injury*

I	Capsular tears
II	Non-bleeding (<5 cm)
	Through and through missile tracts (non-bleeding)
III	Small actively bleeding lacerations
	Bleeding missile tracts
	Subsegmental tissue destruction (non-bleeding)
IV	Large fractures
	Lobar tissue destruction
V	Extensive parenchymal disruption with hepatic arterial or venous injury

and angiography have demonstrated that reperfusion of ischemic segments of liver rapidly follows selective hepatic artery ligation.[1] The relatively benign postoperative course in most patients who have hepatic artery ligation has led to its further examination as a means for controlling blood loss from the liver and preventing early death from hemorrhage.

Because of the need for clarification concerning the use of various modalities in the management of liver trauma, we undertook a retrospective review of 178 consecutive patients with major liver injuries to allow more precise definition of clinical circumstances in which available methods might be used singly or in combination to improve the outcome.

Materials and Methods

All patients with hepatic trauma seen between January 1, 1973 and January 1, 1976 were reviewed. Among the 130 male and 48 female patients who sustained liver injury and underwent exploratory laparotomy for repair, the median age was 29 years with a range of 6 to 81 years.

Our approach to patients with major abdominal injuries has become standardized. Resuscitation of hemorrhagic shock is begun with electrolyte solutions and continued with whole blood. Diagnosis of intra-abdominal injury is made on the basis of history, the mode of injury, and careful physical examination. Patients sustaining penetrating injuries in the lower chest and abdomen undergo exploratory laparotomy. Patients suspected of having intra-abdominal injury as a result of blunt trauma frequently undergo peritoneal lavage for early confirmation of significant intraperitoneal bleeding.

The patient undergoing laparotomy for hepatic trauma is prepared for a midline incision with ample skin preparation and draping to allow for extention of the incision as a median sternotomy. Preoperative antibiotics are used. Upon entering the abdomen, the hematoma and free blood are rapidly evacuated and bleeding is controlled from the hepatic parenchyma by gentle compression of liver tissue and application of moist packs. Once initial hemorrhage is controlled, the injury is inspected and the nature of the hemorrhage (arterial, venous, or a combination) is ascertained. Compression and packing is once again applied, and, if this maneuver is successful in controlling hemorrhage, attention is turned to other injuries. Resuscitation and replacement of blood loss is continued throughout the procedure. Liberal use of platelets and fresh frozen plasma largely prevents coagulopathy. Once an adequate examination for associated injuries has been conducted and the obvious sources of bleeding controlled, the hepatic wound is re-inspected. If continued arterial bleeding is encountered, the porta hepatis is occluded with an atraumatic vascular clamp (Pringle maneuver). Should this maneuver control the hemorrhage, direct ligature of the bleeding point within the hepatic substance is attempted. If direct ligature of bleeding points is not feasible, the arterial trunk to the bleeding segment of liver is exposed and individually clamped. Subsequently, the arterial trunk to the bleeding segment is doubly ligated with arterial suture. In most instances, parenchymal hemorrhage is controlled in this manner. Devitalized hepatic tissue is debrided. If hemorrhage continues unabated despite packing or after compression of the porta hepatis, hepatic venous injury is strongly suspected, which indicates the need for extention of the laparotomy incision to a median sternotomy for exposure of the retrohepatic vena cava and control of the bleeding sites. The intracaval shunt described by Schrock and colleagues[17] is used to obtain a more complete vascular isolation than that obtained by compression of the porta hepatis alone. Although systemic hypothermia is avoided, local hypothermia using iced saline lavage may be useful during hypotensive epidodes to protect liver and kidneys.[3]

A system for grading injury was designed to assess the severity of hepatic parenchymal damage. Five grades were defined according to the extent of damage and the amount of blood loss resulting from the liver wound (Table 1): Grade I injuries included capsular tears not associated with obvious intra-parenchymal hematoma. Grade II injuries consisted of small (less than 5 cm) shallow, non-bleeding lacerations and non-bleeding through and through missile wounds. Grade III injuries were small lacerations associated with moderate blood loss or sublobar fractures of the liver not associated with massive blood loss. Grade IV included major fractures of hepatic parenchyma involving one or both lobes and associated with

TABLE 2. *Hepatic Trauma Mode of Injury*

Grade	Stab	GS.W.	Blunt	Total
I	10	4	9	23
II	14	44	10	68
III	4	23	17	44
IV	0	11	21	33
V	0	6	4	10

massive blood loss. Patients sustaining hepatic arterial or venous injuries in addition to extensive parenchymal destruction were included in Grade V.

When statistical comparisons were appropriate, the chi square method for independent proportions was employed.

Results

Table 2 lists modes of injury for each patient group. Gun shot wounds were prevalent in Grades II through V. Blunt trauma was found more commonly in Grades III and V. The frequency with which patients presented in shock (systolic blood pressure of less than 80 mm Hg on admission) increased with grade of injury (Fig. 1). Shock was present in 21 of the 32 patients in Grade IV and in 9 of 10 patients in Grade V.

Associated injuries were common, with contiguous structures such as the pleural space, duodenum and pancreas most frequently involved.

Thirty-six patients died, establishing an over-all mortality rate of 20%. Mortality was closely related to severity of hepatic injury. Causes of death are listed in Table 3. Exsanguination from liver parenchyma was encountered in only two patients. Intraoperative hemorrhage from hepatic venous injuries accounted for 6 deaths. Bleeding from extrahepatic sites and sepsis were the most common causes of death.

Septic complications and deaths also became more frequent as grade of injury increased. Table 4 relates the mortality for each injury grade to the incidence of death attributable to infection in each grade. Particularly striking is the frequency of septic deaths among patients with Grade IV injuries where 8 of 11 deaths resulted from infection. Over-all infection rates, likewise correlated with the severity of injury. In patients with the most severe hepatic parenchymal injuries (Grades III and IV), infection rates were 36 and 41%, respectively. Of the 29 patients with infections in Grades III and IV, 21 developed perihepatic sepsis.

Hepatic wounds were managed by drainage, suture, resectional debridement, selective hepatic artery ligation (SHAL), and formal lobectomy, or combinations thereof (Table 5). As injury grade increased, SHAL was employed more frequently (Fig. 2.). Hepatic artery interruption was successful in controlling arterial bleeding from the hepatic parenchyma in *all* patients in whom it was employed. The operative records of 59 patients undergoing hepatic artery ligation disclose that in 82% the decision to employ SHAL followed the failure of conventional modalities to control arterial bleeding.

Hepatic artery interruption and subsequent hepatic parenchymal ischemia are potentially dangerous. The effects of SHAL on mortality and development of perihepatic sepsis were analyzed in the 59 patients who underwent SHAL. Mortality among these patients

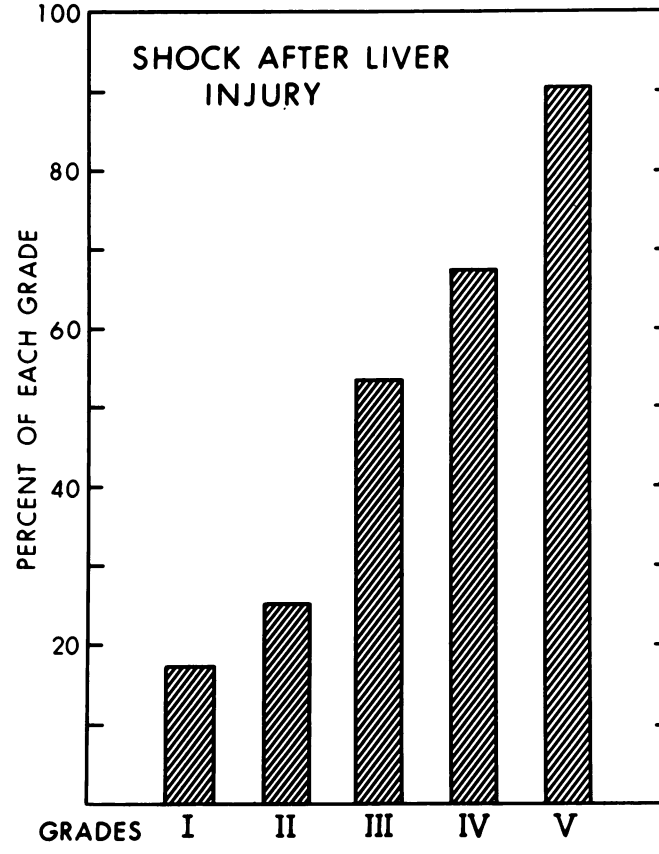

FIG. 1. The incidence of shock increased with the severity of injury.

was 31%, compared to an over-all mortality for all other treatment modalities of 22% ($P > .01$ N.S.). Similarly, the incidence of perihepatic sepsis following hepatic artery interruption was not significantly increased when compared to other modalities ($P > 0.1$ N.S.).

Rebleeding after successful initial hemostasis was observed in three patients. In one patient with a bilobar wound, the left lobar ramus was ligated and late bleeding occurred from the right lobe. All episodes of late bleeding responded to SHAL, and no delayed resections were necessary. The only patient who developed lobar hepatic necrosis underwent treatment of a severe hepatic fracture by large interlocking mattress sutures.

The serum biochemical disorders which regularly follow hepatic injury, hepatic resection, and hepatic ar-

TABLE 3. *Causes of Death*

Exsanguination from liver parenchyma	2
Exsanguination from hepatic veins	6
Exsanguination from extrahepatic sites	12
Sepsis and related complications	10
Head injury	3
Ruptured aneurysm	1
Unknown	1
Hepatic failure	1

TABLE 4. *Mortality After Hepatic Trauma*

Grade	Total	Deaths	Septic Deaths
I	24	2	1
II	68	11	2
III	44	5	3
IV	32	11	8
V	10	7	0

TABLE 5. *Hepatic Trauma*

I	8	0
II	10	2/8 (25%)
III	36	10/16 (63%)
IV	41	11/13 (86%)
V	10	1/1 (100%)

tery ligation have been reviewed in detail.[4] Of special interest was the finding in our patients of persistent hyperbilirubinemia (total bilirubin in excess of 3 mg% for more than five days) in septic patients. Thirteen of 19 patients with perihepatic sepsis were jaundiced while only three of 130 long-term, non-septic survivors were similarly affected.

Discussion

The data herein indicate that a selective approach to hepatic injury is desirable. The principles underlying successful management of patients with severe hepatic trauma include acquisition of early complete hemostasis, the debridement of devitalized tissue, and adequate provision for drainage of blood and bile from perihepatic spaces.

Previous studies have documented that intraoperative exsanguination from hepatic parenchymal injury is a frequent cause of death following hepatic trauma even when hepatic venous injury is excluded. Trunkey and his colleagues[19] observed that the cause of death in 26 of 34 patients dying of their liver injuries was hemorrhage. Similarly, Lim and associates[10] ascribed 38 of 71 immediate deaths from liver trauma to hemorrhage. We recorded 20 immediate deaths only two of which resulted from hepatic parenchymal hemorrhage. Selective application of hepatic artery interruption effectively controlled intraoperative hemorrhage. If arterial hemorrhage recurred after initial packing and compression of the injury, atraumatic occlusion of the structures in the porta hepatis resulted in cessation of hemorrhage in all cases. The subsequent identification and accurate interruption of an appropriate branch of the hepatic artery resulted in hemostasis, effectively prevented early death from hemorrhage, and decreased the need for hepatic lobectomy. SHAL was frequently used after other means had failed and yet mortality for these patients was not significantly increased when compared to similarly injured patients. Although anatomic lobectomy was seldom needed, limited debridement of devitalized liver tissue was indicated where peripheral tissue destruction had completely disrupted blood supply.

Hepatic dearterialization may be contraindicated when severe pre-existing liver disease is discovered. The cirrhotic liver may depend on increased arterial flow. Thus, interruption should be employed cautiously in patients with injured cirrhotic livers. The autogenous omental pack described by Stone and Lamb[18] may be of value in such instances. Furthermore, the decision to use SHAL is best made as soon as persistent arterial bleeding from liver tissue is evident. Extensive dissection of the supporting attachments of the liver interrupts potential collateral channels and must be avoided if SHAL is to be used. If such dissection is necessary, safe use of hepatic artery ligation is precluded.[9]

We have not employed second-look operations as such following hepatic artery interruption. On several occasions patients have undergone re-operation for other reasons during the initial 48 to 72 hours following hepatic artery ligation. The persistence of a dusky cyanosis of the hepatic tissue is noticeable in these cases. However, none of these patients have had hepatic resection, and the single patient in this series who developed lobar hepatic necrosis did so as a result of

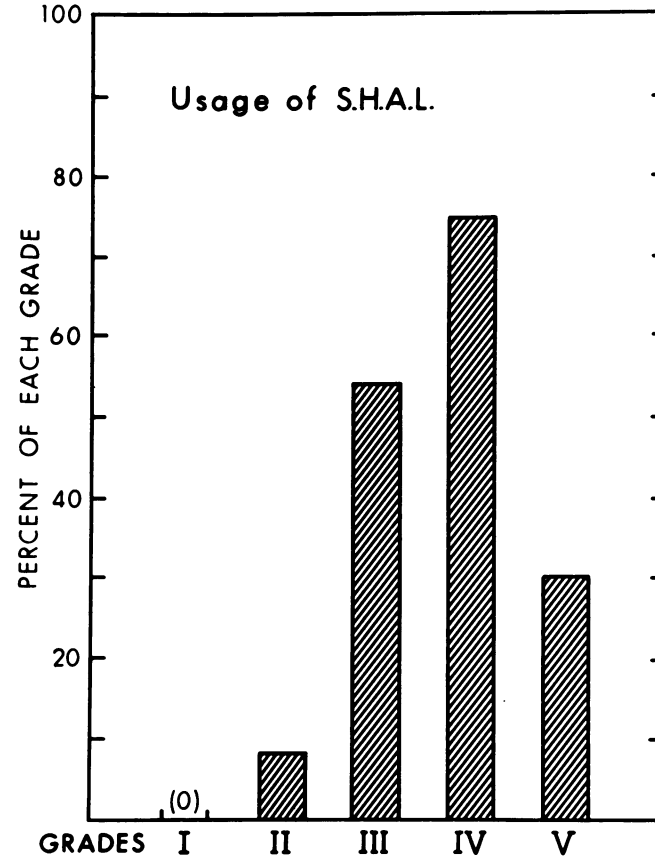

FIG. 2. Selective hepatic artery ligation was employed more frequently with increased severity of injury.

TABLE 6. *Management of Hepatic Wounds*

Drainage only	84
Suture	16
S.H.A.L.	59
Debridement	8
Lobectomy	3

extensive suturing and did not have hepatic artery ligation.

Early reperfusion of hepatic tissue via arterial collaterals has been repeatedly demonstrated following SHAL. Lewis and colleagues[9] angiographically visualized open collaterals four hours after hepatic artery ligation. This finding implies that early rebleeding from injured tissue is a potential hazard. We have observed no instances of postoperative arterial rebleeding where SHAL was used as outlined above.

Olcay and colleagues[14] have observed impaired clearance of blood-borne endotoxin following hepatic ischemia in baboons. Similarly, Saba[15] postulated that clearance of microorganisms might be impaired with hepatic ischemia. We could not confirm a significant increase in infection rate or frequency of perihepatic sepsis following hepatic dearterialization.

Significant disorders of serum enzyme levels follow hepatic artery interruption. The lactic dehydrogenase and serum glutamic oxaloacetic transaminase levels reach values more than four to six times normal during the initial 72 hours after hepatic arterial interruption. These abnormalities rapidly revert to normal. Hyperbilirubinemia is not seen regularly following hepatic arterial interruption. In our patients, persistence of hyperbilirubinemia often was associated with development of perihepatic sepsis.

The elements of successful postoperative care following operation for hepatic trauma include assurance of adequate hepatic nutrient flow through maintenance of blood volume and vigorous support of oxygenation, including the frequent use of adjuvant ventilators. Maintenance of normoglycemia is best accomplished by infusion of 5% glucose solutions. Glucagon infusion, theoretically valuable in maintaining portal flow, was not regularly employed. Maintenance of portal venous oxygenation through proscription of oral nutrients is apparently valuable. Postoperative complications may dictate the need for total parenteral nutrition.

The management of the severe liver injury following blunt or penetrating trauma should be consistent with the extent of parenchymal destruction and the amount of hemorrhage which accompanies the injury. Selective hepatic artery ligation is an efficient means of gaining hemostasis and avoiding death from hemorrhage when arterial bleeding persists or recurs in parenchymal lacerations. Devitalized hepatic tissue should be resected and, where possible, direct hemostasis within the hepatic parenchyma obtained. Preservation of functioning hepatic tissue through avoidance of formal lobar resection is possible in many patients and may ultimately lead to improved prognosis.

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DISCUSSION

DR. WILLIAM A. NEELY (Jackson, Mississippi): Mortality can be correlated with various things, such as distance from the emergency room, how quick the patient gets to the operating room. We found in our series of some 240-odd cases that, the quicker we took the patient to the operating room, the higher the mortality. But only six patients were taken immediately to surgery.

I see that resection has not been employed extensively by the presenter. We believe that a mushy, bleeding liver with a large central