Experience with the Finger Fracture Technique to Achieve Intra-Hepatic Hemostasis in 75 Patients with Severe Injuries of the Liver

H. LEON PACHTER, M.D., FRANK C. SPENCER, M.D., STEVEN R. HOFSTETTER, M.D., GENE F. COPPA, M.D.

The most important concept emerging from the management of complex hepatic trauma is that direct suture ligation of severed blood vessels and bile ducts is the most effective treatment. Three essential maneuvers are necessary: (1) the use of the finger fracture technique to expose the laceration widely, so that individual ligation of severed blood vessels and bile ducts can be accomplished under direct vision; (2) occluding the portal triad for 20 to 60 minutes; (3) closure of the hepatic incision over a viable omental pedicle. Two hundred consecutive patients with hepatic injuries were treated at the Trauma and Shock Unit of Bellevue Hospital between July 1976 and January 1982. One hundred and twenty-five injuries (63%) could be managed by superficial suture and drainage alone; 75 (37%) more extensive injuries required additional therapy; 47 of the 75 injuries required inflow occlusion for periods of up to 60 minutes, with the mean occlusion time of 30 minutes. All patients were pretreated with 30 to 40 mg/kg of Solu-Medrol® prior to cross-clamping the portal triad. In addition, the liver was cooled to 27–32 C° topically by pouring 1 liter of iced Ringer's lactate directly on the liver surface, monitoring the temperature with an intra-hepatic probe. Ischemia time exceeded 20 minutes in 70%, 30 minutes in 40% and 60 minutes in 7% of patients. This approach, with complex hepatic trauma, has been dramatically effective. There were only four deaths (5.3%). One (1.3%) patient required reoperation for bleeding; three patients (4%) developed perihepatic abscesses; and two patients (3%) developed biliary fistulae that spontaneously closed. An extended right hepatectomy was necessary in the one patient who required reoperation for bleeding. This represents the only case of a formal hepatic resection in this series. Hepatic artery ligation was not employed in any case. These experiences strongly endorse the direct approach to the treatment of major hepatic lacerations by opening a lacerated liver sufficiently to ligate lacerated blood vessels and bile ducts, followed by closure over an omental pedicle. The wide-spread adoption of this technique will probably lower the mortality from massive liver injuries to 5–10%.

From the Department of Surgery, New York University Medical Center and the Trauma and Shock Unit, Bellevue Hospital Center, New York, New York

Presented at the Ninety-Fourth Annual Meeting of the Southern Surgical Association, December 6–8, 1982, Palm Beach, Florida.

Reprint request: H. Leon Pachter, M.D., New York University Medical Center, 530 First Avenue, Suite 6-C, New York, New York 10016.

Submitted for publication: January 24, 1983.

BETWEEN JULY 1976 AND JANUARY 1982, 200 consecutive patients were treated for injuries to the liver at Bellevue Hospital. One hundred twenty-five of these were simple injuries, readily controlled by suture and drainage; 75 were complex injuries, with major parenchymal disruption and hemorrhage. One of the authors participated in the operation whenever a complex hepatic injury occurred.

In 1976, discouraged with the high mortality following conventional methods of treating hepatic injuries, the finger fracture technique was adopted to widely expose severe hepatic lacerations in order to obtain adequate hemostasis. Occlusion of the portal triad in excess of 30 minutes was necessary in 45% of the patients. With this approach, only one death occurred in the first 22 consecutive patients.[32] Since this initial report, 115 additional patients with hepatic injuries have been treated, 53 of whom were classified as complex. This report summarizes our total experience with 75 complex hepatic injuries over a 6-year period.

Classification

Confusion continues to plague the surgical literature with regard to the classification of hepatic injuries—rendering comparison of different reports virtually impossible.[2,13,26] Hepatic injuries at our institution are classified as follows: grade I—simple injury, managed by superficial suture and drainage or drainage alone; grade II—major intra-parenchymal injury with active bleeding; grade III—major intra-parenchymal injury with massive bleeding, or extensive parenchymal destruction; grade IV—juxtahepatic venous injury.

0003-4932/83/0600/0771 $01.20

TABLE 1. *Cause of Hepatic Injuries*

Stab wound	41	(55%)
Gunshot wound	25	(33%)
Blunt	9	(12%)
Total	75	

Clinical Material

From July 1976 to January 1982, 200 consecutive patients with hepatic injuries were treated. Of these, 125 injuries (63%) were classified as simple and 75 (37%) as complex. Of the 75 complex injuries, 41 (55%) resulted from stab wounds, 25 (33%) from gunshot wounds, and nine (12%) from blunt injuries (Table 1).

Injuries to other organs were present in 65.3% of these 75 patients. Eighteen patients (24%) had one other organ injury, 14 patients (19%) had two, 14 patients (19%) had three, one patient (1.3%) had four, and two patients (3%) had five or more associated injuries. Both of the patients with more than five injuries died during the operation. Seventeen patients (23%) had an associated hollow viscus injury—six colonic, three enteric, and eight gastric (Table 2).

Six patients (8%) sustained juxtahepatic venous injuries. An atrial caval shunt was used in four patients. In the two other patients, hemorrhage was controlled without the use of a shunt.

Treatment

The operative approach in managing complex hepatic injuries consists of seven consecutive steps: (1) temporary hemostasis achieved by compressing the injury until hypovolemia and acidosis are corrected[23] (2) occlusion of the portal triad (Pringle maneuver),[35] (3) finger fracture of the liver[24,41] to achieve adequate exposure within the depths of the hepatic laceration, (4) ligation and repair of the lacerated vessels and bile ducts under direct vision,[11,32,33,42,43] (5) debridement of non-viable hepatic parenchyma (6) filling the dead space within the liver with a viable pedicle of omentum[10,40] and (7) wide dependent drainage.

TABLE 2. *Associated Injuries among 75 Patients with Complex Hepatic Trauma*

No. of Organs	Patients
1	18 (24%)
2	14 (19%)
3	14 (19%)
4	1 (1.3%)
5	2 (3%)
Total	49* (65.3%)

* Seventeen patients (23%) had hollow viscus injuries; stomach (8) colon (6), SB (3).

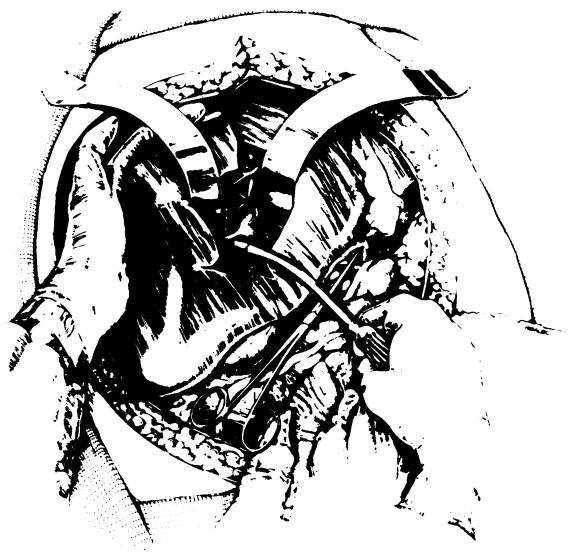

FIG. 1. The mobilized liver is brought forward with the first assistant's left hand. At the same time, a suction apparatus in the first assistant's right hand aspirates and exposes the injury. The porta hepatis is occluded with an atraumatic vascular clamp.

Resuscitation was often completed in the operating room after the abdomen had been opened, initially compressing the margins of the liver manually to control bleeding. Once the hypovolemia and acidosis were corrected, the liver was inspected in detail to assess the injury. Usually, the liver could be brought manually forward into the incision by having the first assistant place his left hand behind the liver and bring the mobilized organ forward. At the same time, a suction apparatus in the first assistant's right hand aspirated and exposed the injury. The next critical step was to incise the liver by the finger fracture technique of Lin[24] in the direction of the laceration to provide adequate exposure so that lacerated blood vessels and bile ducts could be controlled under direct vision. (Fig. 1) Incisions of the liver parenchyma varied in size, with a mean length of 15 cm and depth of 7 cm. One of the largest incisions of normal liver parenchyma was 25 cm in length and 12 cm in depth, indicating the extent of incision of normal hepatic parenchyma that may be required to obtain adequate exposure. Care should be taken to keep in mind the anatomic position of the main, right, and left hepatic ducts, so that they are not injured inadvertently. In 28 (37%) of the 75 patients, adequate exposure and hemostasis were readily obtained by appropriate finger fracture incision of the adjacent hepatic parenchyma. In the other 47 patients (63%), however, significant bleeding required temporary occlusions of the portal triad. Prior to occluding the portal triad, all patients were given 30 to 40 mg/kg of methylprednisolone succinate (Solu-Medrol®). In addition, the liver was cooled at 27–32 C° topically by pouring 1 liter of iced Ringer's lactate

directly on the liver surface, monitoring the temperature with an intra-hepatic probe. When the temperature rose above 32 C, additional iced Ringer's lactate was poured onto the liver. Usually, 500 cc of this solution was required every 15 minutes in order to maintain the desired hypothermia.

In 30 patients (70%), the portal triad was clamped for longer than 20 minutes. In 17 patients (40%), however, occlusion time exceeded 30 minutes, and in three patients (7%), it exceeded 1 hour. The mean inflow occlusion time was 30 minutes for the entire group (Fig. 2). Liver function tests were measured serially after operation in all patients. Although abnormalities in these tests were noted in some patients, all returned to normal ranges within a few days. There were no instances of hepatic failure.

Once hemostasis had been obtained, all non-viable liver tissue was debrided. A broad pedicle of omentum was then constructed, based upon either the right or the left gastro-epliploic vessels, as described by Stone.[40] The omentum was then used to fill the defect within the liver, after which the two free edges of hepatic parenchyma on either side were approximated loosely, over the omental pedicle.[32,33] The hepatic injury was then drained widely. A closed suction drain (Jackson-Pratt) was placed anterior and posterior to the injury. (Fig. 3) In addition, two large Penrose drains were placed inferior to the injury in the most dependent position. The closed suction drains were removed on the fifth postoperative day if drainage had ceased. The Penrose drains were mobilized on the tenth postoperative day, and removed 4 days later.

Results

Mortality

There were four deaths in the 75 patients for an overall mortality of 5.3% (Table 3). No deaths or serious complications occurred among the 28 patients with a grade II injury (major hepatic lacerations with active bleeding, not requiring inflow occlusion). Four patients died, however, with grade III and grade IV injuries (major hepatic lacerations with massive bleeding requiring inflow occlusion; or juxtahepatic venous injuries). Three patients died during operation, and one died 4 days later. The clinical data of these four patients are summarized below.

Patient 1. Patient 1 was shot through the back with a 30.06 rifle. An extensive hepatic laceration was repaired, but death resulted from massive retroperitoneal injuries (spine, paraspinal muscles and kidney).

Patient 2. This patient had multiple gunshot wounds from a 9-mm handgun. On admission, no blood pressure could be obtained. Death resulted from injuries of the liver, small bowel, hepatic artery, portal vein, and the retrohepatic cava.

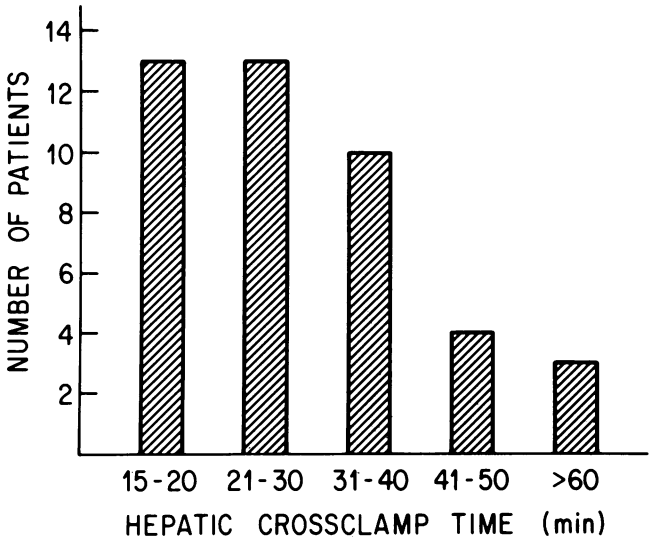

FIG. 2. Hepatic ischemia time.

Patient 3. This patient had multiple injuries from a 45-caliber pistol. He was admitted without an obtainable blood pressure, and died from injuries of the liver, retrohepatic vena cava, colon, small bowel, stomach, pancreas, and spleen.

Patient 4. The fourth patient had multiple injuries from stab wounds, was admitted with a hematocrit of

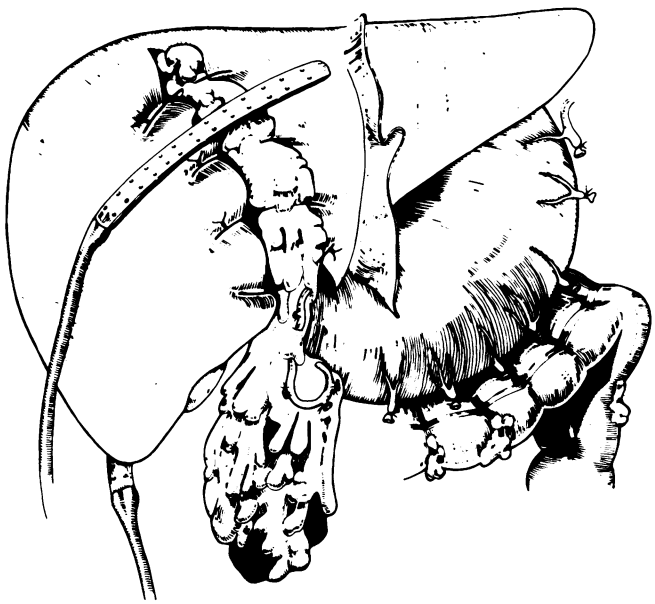

FIG. 3. Omentum has been inserted into the area of liver injury and is held in place by several interrupted liver sutures. Closed suction drainage anteriorly and posteriorly is accomplished by a pair of Jackson-Pratt drains (Courtesy of Pachter HL, Spencer FC. Ann Surg 1979; 190:423.

TABLE 3. *Complex Hepatic Trauma—Management and Results in 75 Patients*

	Mortality	Morbidity Abscess	Rebleeding	Bile Fistula
Grade II 28 (37%)	0	0	0	0
Grade III 41 (55%)	2 (4.9%)	3 (7.3%)	1 (2.7%)	2 (4.9%)
Grade IV 6 (8%)	2 (33.3%)	0	0	0
Total 75	4 (5.3%)	3 (4%)	1 (1.3%)	2 (2.7%)*

* Spontaneous closure within 2 weeks.

10 and no palpable blood pressure. Injuries included the liver, portal vein, hepatic artery, external and internal jugular veins, and left common carotid artery.

Complications (Table 3)

Biliary Fistula

Two patients (2.7%) drained moderate amounts of bile through the Jackson-Pratt drains, first noted 5 to 7 days following operation. The biliary drainage in both patients, however, stopped spontaneously by the 14th postoperative day.

Postoperative Abscess

Three patients (4%) developed localized abscesses, treated by surgical drainage 10 to 14 days following operation: two were subphrenic and one intrahepatic.

Re-Bleeding

Only one of the 75 patients (1.3%) required reoperation for bleeding. Hemostasis was achieved initially, but bleeding returned 12 hours after surgery and continued. Hemostasis could be obtained only by an extended right hepatectomy, after which the patient recovered satisfactorily. This was the only formal lobectomy necessary in the entire group of 75 patients.

Discussion

The mortality rate of only 5.3% in this series of 75 patients with complex hepatic injuries treated over a 6-year period is far lower than that reported by others.[8,22,29,42,43] A precise comparison of experiences from different groups is not possible, however, because of variance in classification of the severity of injury.

Hemorrhage has been the major cause of death in virtually all experiences reported. Bleeding can arise from three sources: branches of the hepatic artery, the portal vein, or the hepatic veins. The main obstacle to effective hemostasis has been the fear of incising normal hepatic parenchyma. As a result, indirect measures have been used, including hepatic artery ligation, hepatic lobectomy, and packing. It seems probable that the low mortality rate (5.3%) in this series is primarily due to the ability to control hemorrhage. Three distinctive operative techniques were used to achieve this result.

I. Finger Fracture Technique

The finger fracture concept is the most important. With adequate exposure, lacerated blood vessels can be treated readily. This, of course, is the basic approach for traumatic lacerations in any area of the body. The finger fracture technique was reported by Lin[24] in 1958 and, subsequently, by Ton[41] in 1963. To the authors' knowledge, the technique has not been used widely in this country because of the widespread belief that incision of normal hepatic parenchyma is prohibitively dangerous if not preceded by ligation of the appropriate hepatic artery, portal vein, and hepatic vein. At the University of Rochester, Schwartz[38] performed 60 hepatic resections with the finger fracture technique in the past few years with no mortality. Balasegaram,[4] by contrast, specifically condemned incisions of the liver along non-anatomic planes because of the risk of bleeding.

The experiences cited in this report clearly show that the dangers from incising normal hepatic tissue are exaggerated greatly. The finger fracture technique has been employed now for over 5 years in more than 70 patients, without significant problems. In 47 of the 75 patients (63%), occlusion of the portal triad was necessary in conjunction with the finger fracture technique. Only six of the 75 patients (13%) had continued bleeding following occlusion of the portal triad, indicating that a juxtahepatic venous injury was present. These data indicate that in over 85% of patients, occlusion of the portal triad will temporarily stop the bleeding. This maneuver, coupled with the finger fracture technique to expose lacerated blood vessels so that they could be controlled under direct vision, is responsible for the dramatic decrease in mortality from exsanguination.

II. Safety of Clamping the Portal Triad

Fear of clamping the portal triad for longer than 15 to 20 minutes under normothermic conditions undoubtedly has restricted its use with hepatic injuries. This limited time frame was established initially in animal models.[36] Experimental studies in dogs now are recognized as not applicable to man—primarily because of differences in splancnic venous circulation.[18,19,31] How long the portal triad can be safely occluded in man is unknown, but published data indicate that periods of up to 1 hour are safe.[18,19,31] In 1978, Huguet,[19] in France, reported on 20 cases of hepatic resection. In nine of the 20 patients, the portal triad was clamped between 24

and 65 minutes, with a mean occlusion time of 38 minutes. Abnormalities in hepatic enzymes returned to normal, and no patient demonstrated signs of hepatic failure.

Normothermic hepatic vascular exclusion for elective hepatic surgery cannot be extrapolated to trauma patients with certainty for two reasons: (1) spontaneous collaterals which are usually present in large hepatic tumors may be responsible for the liver's ability to tolerate normothermic ischemia, and (2) the severely injured, hypotensive patient may not tolerate normothermic ischemia as well as the patient undergoing an elective hepatic resection.

Methods of extending normothermic ischemia. **Hypothermia.** Both Bernhard[5] and Goodall[17] have shown that dogs subjected to normothermic occlusion for longer than 40 minutes sustained a 100% mortality rate. With hypothermia of up to 28–32 C, however, virtually all of the dogs survived following 1 hour of portal triad occlusion. Fortner,[15] using a complex technique of complete vascular isolation and hypothermic perfusion in 29 major hepatic resections, was able to achieve occlusion time of up to 147 minutes without untoward effects. The value of hypothermia in increasing the liver's tolerance to ischemia is, therefore, well-documented.

The effectiveness of topical hypothermia, described in this paper, is uncertain. Despite keeping the surface temperature of the liver between 28 C and 32 C by pouring iced Ringer's lactate onto the liver, wide temperature gradients within the liver must exist. Therefore, the beneficial effects of our technique cannot, at present, be evaluated adequately.

Steroids. Thirty to 40 mg/kg of methylprednisolone succinate (Solu-Medrol) was given to each patient in this report prior to occluding the portal triad. The rationale for its use was based on reports by Delpin[9] and Figueroa.[12] Their findings showed that occlusion of the portal triad in rabbits for 30 minutes resulted in a 100% fatality rate, but pretreatment with methylprednisolone succinate lowered the mortality rate to 10%. In addition, 1 hour of hepatic occlusion resulted in only a 50% mortality rate, if steroids were given prior to inflow occlusion. Starzl,[39] on the other hand, with his broad experience in liver transplantation, does not consider steroids to be of any benefit in extending ischemia time.

As the upper limit of ischemia time to the liver is unknown, we continue to favor topical hypothermia and large doses of steroids. Randomized trials, in the future, will be required to establish whether these methods of extending hepatic ischemia time are necessary.

III. The Viable Omental Pack

The use of a pedicle of omentum as an autogenous pack in 37 patients with hepatic lacerations was reported by Stone[40] in 1975. A subsequent report by Fabian and Stone,[10] in 1979, described experiences with 115 patients. The omentum was used as a pack, instead of the traditional gauze pack frequently used in World War II. The omental pack was inserted simply and sutured in place with minimal debridement of hepatic parenchyma or ligation of lacerated vessels. The fact that this method of "packing" controlled bleeding in 105 of 113 cases would seem to indicate that the source of bleeding in the vast majority of patients is from lacerated veins and not from arteries.

In order to avoid peri-hepatic and intra-hepatic abscesses, meticulous hemostasis must be achieved first, as well as adequate debridement of all non-viable tissue before the omentum is inserted.

Theoretically, the omentum may be of value in other ways. It fills dead space within the liver with viable tissue, perhaps lessening the risk of sepsis. It also introduces a rich source of macrophages which, in themselves, may be beneficial.[20] Jurkiewicz[20] reported on the use of free omental grafts for extensive reconstructive surgery of the face, further supporting the value of omentum in both obliterating dead space and minimizing infection.

Juxtahepatic venous injuries. Fortunately, injuries to the retrohepatic cava and hepatic veins are rare, but they constitute one of the most complex surgical challenges because of the danger of exsanguination. A number of ingenious shunts have been devised to control hemorrhage.[28,34,37] the most popular being the atrial-caval shunt, inserted through the right atrium into the vena cava, after a median sternotomy incision has been made.[16] Although theoretically sound, all reports describe a mortality rate in excess of 50%, even among the most experienced trauma surgeons.[8,11,21,43]

With increasing experience with the finger fracture technique, the frequency with which a shunt is required is uncertain. In the last two patients with major hepatic vein or retrohepatic caval injury in this series, rapid finger fracture through hepatic parenchyma to expose the site of injury permitted direct suture of large lacerations without the use of a shunt.

Hepatic lobectomy and hepatic artery ligation. **Hepatic lobectomy.** The concept of hepatic lobectomy along anatomic planes of cleavage is an attractive approach for an extensive laceration that has destroyed much of the liver parenchyma. The mortality rate associated with hepatic resection for trauma, however, remains prohibitively high,[8,22,26,42,43] and is not recommended with enthusiasm by American authors. Balasegaram,[4] however, reports a mortality rate of 10% in 85 cases of hepatic resection for trauma, and Blumgart[6,7] reports a mortality rate of 21% in 19 cases. While the low mortality rate, achieved by these authors, with hepatic resection is impressive, it does not appear to represent the general experience with this approach.

With the use of inflow occlusion and the finger fracture technique to expose lacerated blood vessels and bile ducts, a lobectomy would seem necessary only in unusual circumstances. One patient in this series, the only one in whom reoperation was necessary for bleeding, was treated with a right hepatic lobectomy. Resectional debridement, on the other hand, of non-viable tissue is essential if postoperative hepatic necrosis and abscess formation are to be avoided. Anatomic hepatic lobectomy for trauma should be restricted to two specific instances: (1) complete parenchymal destruction and (2) failure of all other conventional methods to arrest bleeding.[28]

Hepatic artery ligation. The historical events leading to the erroneous belief that ligation of the hepatic artery was prohibitively dangerous were summarized by Walt.[43] The fact that the hepatic artery can be ligated without untoward complications has been well-established.[1,13,14,27,28,43] The initial enthusiasm for its use has waned, and with the exception of Flint[13] its use for hepatic trauma ranged between 0% and 2%.[30] Mays et al.[1,14] popularized this technique, reporting excellent results in controlling hemorrhage in 60 patients with hepatic trauma.[1,14] Subsequently, Flint and Polk[13] reviewed their experience in 540 patients with hepatic trauma in whom selective hepatic artery ligation was employed in 94 (17%). Selective hepatic artery ligation failed to control bleeding in 15 patients (16%), with a subsequent mortality rate of 47% in these patients. The authors confirmed the ineffectiveness of selective hepatic artery ligation to control bleeding from either lobar branches of the portal vein or from major hepatic veins or their intra-hepatic tributaries. In addition to the inability of selective hepatic artery ligation to control venous bleeding or retrograde bleeding from the hepatic veins or retrohepatic cava, sepsis and hepatic necrosis have both been reported.[3,25]

Although ligating the hepatic artery is safe, its use in hepatic trauma seems questionable. The addition of hepatic artery ligation in a hypotensive patient with a resultant decrease in perfusion to the liver may render the liver sufficiently ischemic, resulting in subsequent necrosis and sepsis. Whatever the basis for the effectiveness of hepatic artery ligation in stopping bleeding, however, it seems to be unnecessary in the vast majority of patients.

Conclusion

The data in this report, 75 complex injuries treated over a 6-year period with a mortality rate of 5.3%, indicates that hemorrhage can be controlled in over 90% of massive hepatic lacerations with the technique described. The three key features are (1) the use of the finger fracture technique to expose the laceration widely, incising as much as 25 cm by 12 cm of adjacent hepatic parenchyma, (2) occluding the portal triad for 20 to 60 minutes, and (3) the subsequent insertion of a viable omental pedicle.

Both the fear of incising normal hepatic parenchyma and clamping the portal triad for longer than 20 minutes seem exaggerated greatly. Wide-spread adoption of these techniques will probably lower the mortality rate from massive liver injuries to 5–10%. Failures are usually due to juxtahepatic venous injuries. These may be approached directly by extensive finger fracture or by the insertion of an intra-caval shunt. Future studies are necessary to determine the optimal treatment for these devastating injuries.

Acknowledgments

The authors would like to thank Dr. Allan E. Dumont for his critical review of the manuscript and Miss Rachel Weitman, and Mrs. Claudette Curtis for their assistance with preparation of the manuscript.

References

1. Aaron WS, Fulton RL, Mays ET. Selective ligation of the hepatic artery for trauma of the liver. Surg Gynecol Obstet 1975; 141:187–189.
2. Aldrete JS, Halpern NB, Ward, SA, Wright, JO. Factors determining the mortality and morbidity in hepatic injuries: an analysis of 108 cases. Ann Surg 1979; 189:466–473.
3. Atik M. Operative cholangiography and special technical consideration in the management of severe injury of the liver. Surg Gynecol Obstet 1976; 143:741–748.
4. Balasegaram M, Joishy SK. Hepatic resection: the logical approach to surgical management of major trauma to the liver. Am J Surg 1981; 142:580–583.
5. Bernhard WF, McMurrey JD, Curtis GW. Feasibility of partial hepatic resection under hypothermia. N Engl J Med 1955; 253:159–164.
6. Blumgart LH, Drury JK, Wood CB. Hepatic resection for trauma, tumour and biliary obstruction. Br J Surg 1979; 66:762–769.
7. Blumgart LH. Personal communication.
8. Defore WW, Mattox KL, Jordan GL, Beall AC, Jr. Management of 1,590 consecutive cases of liver trauma. Arch Surg 1976; 111:493–497.
9. Delpin EA, Figueroa I, Lopez R, Vazquez J. Protective effect of steroids on liver ischemia. Am Surg 1975; 41:683–695.
10. Fabian TC, Stone HH. Arrest of severe liver hemorrhage by an omental pack. South Med J 1980; 73:1487–1490.
11. Feliciano DV, Mattox KL, Jordan GL Jr. Intra-abdominal packing for control of hepatic hemorrhage: a reappraisal. J Trauma 1981; 21:285–290.
12. Figueroa I, Delpin EA. Steroid protection of the liver during experimental ischemia. Surg Gynecol Obstet 1975; 140:368–370.
13. Flint LM Jr, Polk HC. Selective hepatic artery ligation: limitations and failures. J Trauma 1979; 19:319–323.
14. Flint LM Jr, Mays ET, Aaron WS et al. Selectivity in the management of hepatic trauma. Ann Surg 1977; 185:613–617.
15. Fortner JG, Shiu MH, Kinne OW, et al. Major hepatic resection using vascular isolation and hypothermic perfusion. Ann Surg 1974; 180:644–652.
16. Fullen WD, McDonough JJ, Popp MJ, Altemeir WA. Sternal splitting approach for major hepatic or retrohepatic vena cava injury. J Trauma 1974; 14:903–910.
17. Goodall GW, Hyndman WWB, Gurd FN. Studies on hypothermia in abdominal surgery. Arch Surg 1957; 75:1011–1019.
18. Huguet C, Nordlinger B, Galopin JJ, et al. Normothermic hepatic vascular exclusion for extensive hepatectomy. Surg Gynecol Obstet 1978; 147:689–693.
19. Huguet C, Nordlinger B, Bloch P, Conard J. Tolerance of the

human liver to prolonged normothermic ischemia. Arch Surg 1978; 113:1448–1451.
20. Jurkiewicz MJ, Nahai F. The omentum—its use as a free vascularized graft for reconstruction of the head and neck. Ann Surg 1982; 195:756–765.
21. Kudsk KA, Sheldon GF, Lim RC. Atrial-caval shunting after trauma. J Trauma 1982; 22:81–85.
22. Levin A, Gover P, Nance FC: Surgical restraint in the management of hepatic injury: a review of charity hospital experience. J Trauma 1978; 18:399–403.
23. Lim RC, Lau G, Steele M. Prevention of complications after liver trauma. Am J Surg 1976; 132:156–162.
24. Lin TY, Hsu KY, Hsieh CM, Chen CS. Study on lobectomy of the liver. J Formosan Med Assoc 1958; 57:750–769.
25. Lucas CE, Ledgerwood AM. Liver necrosis following hepatic artery transection due to trauma. Arch Surg 1978; 113:1107.
26. Lucas CE, Ledgerwood AM. Prospective evaluation of hemostatic techniques for liver injuries. J Trauma 1976; 16:442–451.
27. Madding GF, Kennedy PA. Hepatic artery ligation. Surg Clin North Am 1972; 52:719–728.
28. Mays ET. Hepatic trauma. Curr Probl Surg 1976; 13(11).
29. McInnis WD, Richardson, JD, Aust JB. Hepatic trauma. Arch Surg 1977; 112:157–161.
30. Moore EE Jr, Eiseman B, Dunn EL. Current management of hepatic trauma. Contemp Surg 1979; 15:91–116.
31. Nordlinger B, Douvin D, Javaudin L, et al. An experimental study of survival after two hours of normothermic hepatic ischemia. Surg Gynecol Obstet 1980; 150:859–864.
32. Pachter HL, Spencer FC. Recent concepts in the treatment of hepatic trauma. Ann Surg 1979; 190:423–429.
33. Pachter HL, Spencer FC. The Management of Complex Hepatic Trauma. Controversies in Surgery II, Philadelphia: WB Saunders, 1983.
34. Pilcher DB, Harman PK, Moore EE Jr. Retrohepatic vena cava balloon shunt introduced via the sapheno-femoral junction. J Trauma 1977; 17:837–841.
35. Pringle JH. Notes on the arrest of hepatic hemorrhage due to trauma. Ann Surg 1908; 48:541–549.
36. Raffucci FL. The effects of temporary occlusion of the afferent hepatic circulation dogs. Surgery 1953; 33:342–351.
37. Schrock T, Blaisdell FW, Mathewson C Jr. Management of blunt trauma to the liver and hepatic veins. Arch Surg 1968; 96:698–704.
38. Schwartz SI. Personal communication.
39. Starzl TE. Personal communication.
40. Stone HH, Lamb JM. Use of pedicled omentum as an autogenous pack for control of hemorrhage in major injuries of the liver. Surg Gynecol Obstet 1975; 141:92–94.
41. Ton TT. A new technique for operation on the liver. Lancet 1963; I. 192–193.
42. Trunkey DD, Shires GT, McClelland R. Management of liver trauma in 811 consecutive patients. Ann Surg 1974; 179:722–727.
43. Walt AJ. The mythology of hepatic trauma—or Babel revisited. Am J Surg 1978; 135:12–18.

Discussion

DR. SEYMOUR I. SCHWARTZ (Rochester, New York): I hope that Dr. Spencer has added a nail—the final nail—to close the coffin related to the importance of hepatic inflow to the liver and the issue that the liver would tolerate no more than twenty minutes of occlusion. There is a historic point of interest in the concept which we bought for many, many years. Presented at the First Surgical Forum at the American College of Surgeons, it represented a study of six dogs that were unconditioned, unprepared, received no appropriate antibiotics, and had the hepatic artery and portal vein compressed for twenty minutes.

I think there is no question, shown by Huguet and others and the authors' own personal experience, that the liver will, in fact, tolerate occlusion of both the portal vein and the hepatic artery up to ninety minutes, with little alteration of transaminase levels and other enzymes that can be studied.

The other important statement that he made relates to digitoclasia, or finger-fracture technique; his statement is actually truthful when he says that when this article appears in the *Annals of Surgery,* it will represent one of the first declarations in the American literature regarding the efficacy of this technique.

I agree essentially with all of the points made in the presentation and in the manuscript with one minor exception; the authors routinely have used steroid therapy in the management of their patients, and we have never advocated the use of steroids, either for trauma patients or for the elective resection of the liver for tumor.

Our own approach in reference to a bleeding stellate laceratus is rapid occlusion with a vascular clamp of the hepatoduodenal ligament, followed by finger-fracture technique, exposing the culprit. If the bleeding continues in the face of this approach, we have avoided the issue of the intracaval shunt, finding it very cumbersome, and have preferred to directly occlude the aorta at the level of the hiatus; we have been pleased with that approach, which allows us to expose the hepatic vein or the cava at the appropriate level.

We live in a more bucolic atmosphere in New York State, when we compare our series with Dr. Spencer's, and an important differential must be made. As you read the manuscript, you will note that most of these are either gunshot or knifewound injuries. In upper New York State, most of the hepatic trauma with which we deal relates to vehicular trauma, either the automobile or the motorcycle. And this represents a more extensive trauma, one that more frequently requires resectional debridement. Actually, as we reviewed our own data related to vehicular trauma, the results are not as good as we would like to believe; we have an overall mortality rate of 25% for patients who have required resectional debridement of at least a lobe.

My own experience, and a point which Dr. Spencer alluded to, relates more to the elective resection of primary and metastatic tumors of the liver, a circumstance that is actually burgeoning very rapidly, in view of the more aggressive attitudes to resection of lesions primary in the colon, or of the Wilms' tumor and, occasionally, other tumors that have metastasized to the liver, since now we know that these may be associated with a 25% 5-year survival.

And I have adopted this technique, having had the opportunity to work with Professor Lin in Taipei. We approach the patient who is undergoing a segmental resection of the liver, including the notorious Segment 5, which incorporates the gallbladder bed, using inflow occlusion with a vascular clamp, followed by digitoclasia, or finger fracture. For anything less than a trisegmentectomy, this works quite well.

For the trisegmentectomy, in which one is looking at approximately 15% to 20% of the liver which the patient will require for survival, I think it is more appropriate to directly dissect the interlobar vessels.

There is a distinct advantage to the finger-fracture technique which merits emphasis; the hepatic veins, that are truly the *bete noir,* or the black beast, of hepatic surgery, are picked up at an appropriate level. They are picked up at a point that one does not divorce the remaining portion of the liver from the effluent conduit. This circumstance can be a real problem if the hepatic veins are addressed posteriorly, dissecting the inferior vena cava itself.

Over the past five years, we have done over 60 elective resections using this approach, and have had no perioperative mortality. However, we have been plagued with complications, and one of the complications relates to bleeding, and we had to reexplore four patients.

I would like to bring to your attention one point, namely, that in the patient who has undergone hepatic trauma, with necrosis of hepatic tissues, one of the issues which might relate to persistence of bleeding is the fact that the liver will generate fibrinolysin; on two occasions, we have managed to stem the bleeding with the introduction of epsilon-aminocaproic acid.

The other issue, one which is really disturbing to me and on which the authors should be complimented, is their low incidence of infection. Even in elective surgery where the patients are prepared on antibiotics, we are now living with a complication rate of slightly over 15%. Fortunately, we are now no longer addressing it surgically, but calling our radiologic confreres to ultrasonographically direct a pigtail