# *Management of Blunt and Penetrating Injuries to the Porta Hepatis*

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**Injuries to the porta hepatis pose difficult problems in management, and transection of the bile ducts, portal vein and hepatic artery is among the most challenging. Twenty-one patients with severe injuries to the porta hepatis were treated over a ten-year period. Ages ranged from 13 to 56 years, and follow-up was up to nine years. Among the 14 patients with bile duct injury, eight were found to have complete transection, and five suffered a tangential laceration or incomplete disruption with a portion of a duct wall remaining intact. Five of the eight patients who had complete transection underwent primary end-to-end repair with T-tube splinting, while three were treated with primary Roux-en-Y choledocojejunostomy. All patients with incomplete disruption underwent primary repair with or without T-tube splinting. Of the five patients with complete disruption who were treated with primary end-to-end anastomosis of the bile duct in conjunction with T-tube splinting, all required secondary biliary tract reconstruction of some type. No patient with complete transection that was treated with primary Roux-en-Y biliary enteric anastomosis required reoperation. Partial transections were successfully treated with primary repair. Portal vein injury was encountered in ten patients. Injury was successfully managed by primary closure, interposition of a vein, or splenic–mesenteric vein bypass. Associated injuries to liver, pancreas, kidney and duodenum were common. In four patients there was injury to the main or left or right hepatic artery which was managed successfully by repair or ligation, with or without hepatic lobectomy. By adhering to the principles of management to be outlined, many patients with injury to the porta hepatis will survive, and the long term outcome can be gratifying.**

WHILE TRAUMATIC INJURIES to the porta hepatis are rare, and at times delayed in clinical presentation, more often these injuries are dramatic in their life-threatening potential and are some of the most challenging cases for surgical decision making and operative strategy. Among the structures involved in this type of injury, *i.e.*, the hepatic arteries, extrahepatic bile ducts and portal vein, the latter is most likely to lead to a fatal outcome, as has been shown in various reports.[8,19,20] However a most important factor which influences mortality is the large number of serious associated injuries which accompany porta hepatis injury. While the initial management of the ductal and arterial injuries is still controversial, standard approaches to therapy have been proposed.[16,26] On the other hand techniques for acute portal vein injury remain quite diverse[8] because of the varied hemodynamic and operative considerations that involve this injury.

In order to determine the appropriate method of management for these rare, complex and difficult injuries, the combined experience of two widely separated institutions, both with substantial experience in trauma, was analyzed. Thus the relatively recent cases of trauma to the structures in the porta hepatis at UCLA Hospital, Los Angeles, California and at Charity Hospital of Louisiana at New Orleans (Tulane Service) were reviewed retrospectively, and the findings, treatment and results are presented in order to formulate guidelines for the initial management of this injury. Because the early management will have late, as well as early, serious consequences, we believe that the analysis and results of our study benefit the surgeon who is occasionally called on to deal with these emergencies.

## Clinical Material

During the decade July 1968 through July 1978, a total of 21 patients who suffered from penetrating or blunt abdominal trauma that caused injury to structures

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TABLE 1. *Injuries to Porta Hepatis*

| Structure | Type of Injuries | | | Total |
|---|---|---|---|---|
| | Blunt | GSW | Knife | |
| Bile duct | 7 | 6 | 1 | 14 |
| Portal vein | — | 9 | 1 | 10 |
| Hepatic artery | — | 4 | — | 4 |
| | | | | 28 |

of the porta hepatis were treated at UCLA Hospital and at Charity Hospital. There were nine patients at UCLA and 12 patients at Charity. For the purpose of this analysis, these two separate geographic populations are considered collectively inasmuch as mechanism of injury and treatment modalities were quite similar. All were managed by a group of resident and staff physicians with varied experience in this type of serious trauma.

Among these patients, the male–female ratio was 17:4; the mean age was 29 years, ranging from 13 to 56 years. Follow-up has averaged four years with a range from seven months to nine years. One patient was lost to follow-up seven months after injury. There were a total of 28 injuries to structures of the porta hepatis, and the distribution is shown in Table 1. Gunshot wounds accounted for 19 of the injuries, stab wounds for two and blunt abdominal trauma for seven. Of the 21 patients 18 had severe associated trauma with injury to more than one structure of the porta hepatis or to other abdominal organs. Hepatic and pancreatic injuries comprised the predominant associated injury, occurring in 62% and 29% of patients, respectively. Concomitant aortic or vena caval injuries were found in 24% of patients. The severity of injury is underscored by the fact that among those who suffered other organ trauma, in ten of 18 (56%) three or more organs were injured. The distribution of these injuries is listed in Table 2.

Eighteen patients initially presented in shock, showing signs of an acute abdominal catastrophe. They were promptly explored after standard resuscitative measures were taken. Treatment was first directed to control exsanguinating hemorrhage from vascular injuries and then to definitive repair. This report deals exclusively with injuries of the porta hepatis; it does not discuss the techniques to treat associated injuries.

TABLE 2. *Associated Injuries*

| Organ | No. of Patients |
|---|---|
| Liver | 13 |
| Pancreas | 6 |
| Duodenum | 5 |
| Stomach | 5 |
| Kidney | 4 |
| Small bowel | 4 |
| Gall bladder | 3 |
| Vena cava | 3 |
| Aorta | 2 |
| Colon | 2 |
| Heart | 1 |

Because three patients who had sustained isolated injury to the common bile duct did not suffer with an acute abdomen, they presented five days, seven months, and one year respectively after injury with complaints of progressive jaundice.

Among the 14 patients with bile duct injury, eight were found to have complete transection, and five had suffered a tangential laceration or incomplete disruption with a portion of the duct wall remaining intact. Five of the eight patients who had complete transection underwent primary end-to-end repair with T-tube splinting while three were treated with primary Roux-en-Y choledocojejunostomy. All patients with incomplete disruption underwent primary repair with or without T-tube splinting.

Of the ten portal vein injuries, four were complete transections and six were incomplete. Direct repair of the portal vein was accomplished in eight patients; the methods used are illustrated in Figure 1.

One common hepatic artery and three right hepatic artery injuries were treated with ligation primarily or in conjunction with an hepatic resection.

## Results

Five of the 21 patients (24%) who died during the initial hospitalization are classified as operative deaths.

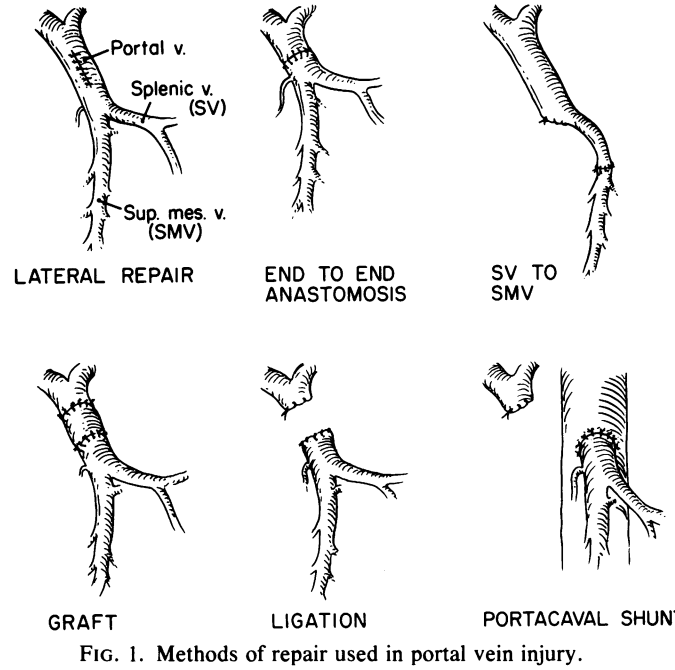


FIG. 1. Methods of repair used in portal vein injury.

Although it is difficult to precisely implicate a single injury as the primary cause of death, portal vein trauma was present in every operative death. However, massive exsanguination from the portal vein was the immediate fatal cause in only two patients; the remaining three died from intra-abdominal or pulmonary sepsis and multiple organ system failure. All of the deaths but one occurred in patients who had three or more abdominal organ injuries in addition to injury to the porta hepatis. Table 3 shows the mortality per single injury of a porta hepatis structure, again indicating the lethality of portal vein injury compared to that of the bile duct or hepatic artery. In this series, once a patient survived the initial hospitalization for traumatic injury to the porta hepatis, no late deaths attributable to this lesion occurred, although complications in those who survived the initial injury were extremely high (81%). Intra-abdominal sepsis, cholangitis, and pancreatitis or pancreatic fistula accounted for 63% of all complications.

Bile duct repair was accomplished by a variety of techniques that are depicted in Figure 2 and summarized in Table 4. Complete bile duct transection occurred in eight patients, five of whom were treated with primary end-to-end anastomosis of the bile duct in conjunction with T-tube splinting and decompression; moreover, all five required secondary biliary tract reconstruction of some type. The mean interval between the primary end-to-end repair and reoperation was six months. Three of these patients were reconstructed with a Roux-en-Y biliary enteric anastomosis and one underwent duct exploration, removal of sludge, and replacement of a T-tube. The final patient in this subset had sustained a machine gun injury to the hilus of the liver with total disruption of the hepatic duct confluence which was repaired with an end-to-end anastomosis. Ten days later, because of bile leak and intrahepatic sepsis, a right hepatectomy was performed during which the proximal end of the common duct was mobilized and anastomosed to the left hepatic duct with T-tube splinting. All of these patients subsequently have recuperated satisfactorily. Three patients who had complete transections were treated with primary Roux-en-Y hepaticojejunostomy. One of these patients had presented seven months after initial injury with a widely dilated ductal system which facilitated the operative repair (Fig. 3). None of the patients who had undergone ductal transection and were treated with primary biliary enteric anastomosis has developed recurrent stricture during the follow-up period, although one did develop a biliary cutaneous fistula that closed spontaneously.

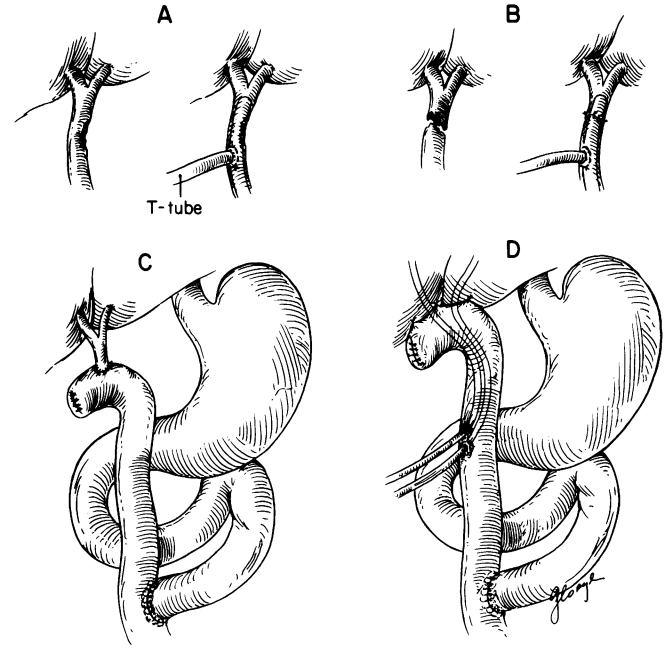


FIG. 2. Methods of repair used in bile duct injury.

Incomplete transection of the bile duct was seen in six patients, and five underwent primary suture closure of the ductal injury. In one patient in this group the cystic duct was used as a patch to repair the common duct defect. The remaining patient required concomitant hepatic resection and died of associated injuries. All five patients with incomplete ductal disruption and primary suture closure have been free of cholangitis or recurrent stricture. The fates of those with bile duct injury as determined by the first operation are summarized in Table 5.

Of the ten patients with portal vein injury, two died of massive hemorrhage before definitive repair could be accomplished. One patient who had sustained injury to the right main branch of the portal vein was treated

TABLE 3. *Mortality Per Injury to Porta Hepatis*

| Structure Injured | No. of Patients | Mortality (%) |
|---|---|---|
| Bile duct | 14 | 1 (7) |
| Portal vein | 10 | 5 (50) |
| Hepatic artery | 4 | 1 (25) |

TABLE 4. *Technique of Bile Duct Repair*

| Technique | No. of Patients |
|---|---|
| Primary end-to-end anastomosis (T-tube) | 5 |
| Primary lateral repair (T-tube) | 4 |
| Primary lateral repair (no T-tube) | 1 |
| Primary duct–enteric anastomosis | 3 |
| Liver resection | 1 |

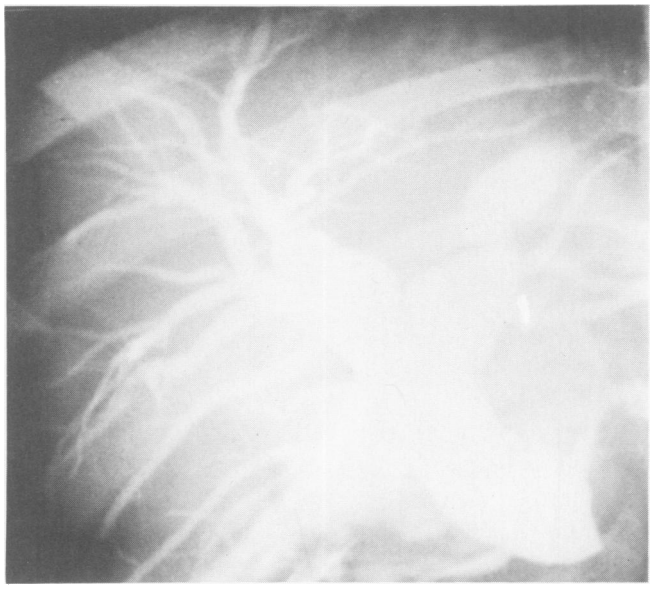

FIG. 3. Widely dilated common bile duct seven months after transection secondary to blunt abdominal trauma.

with ligation and partial hepatectomy which resulted in an operative mortality. Seven patients underwent definitive vascular repair of the portal vein; their results are listed in Table 6. No patient underwent porta-systemic shunt.

## Discussion

While it is predictable that some uncontrollable factors, such as associated injuries or degree and duration of shock, will largely influence the prognosis after porta hepatis injury, we believe that this analysis will help to select the procedure for the initial operation. Except for the unusual case of ductal injury alone with a delayed diagnosis, all of the other cases reported here and in most other literature reviews, presented in shock, requiring an emergency operation, sometimes heroic in nature. Obviously, the associated major vascular injuries may demand priority in hemostasis, and in some instances, necessitate immediate thoracotomy with cross-clamping of the descending aorta, or clamping transabdominally of the aorta at the diaphragmatic hiatus in order to gain temporary control. If, however, the associated injuries to the aorta and vena cava do not represent the major source of hemorrhage and exsanguination is suspected from an injury in the region of the porta hepatis, then a Pringle maneuver using vascular clamps will often allow the structures in the area to be dissected out in order to determine if the injury involves the portal vein, hepatic arteries, extrahepatic ducts, or a combination of structures. Depending on the findings, coupled with a knowledge of the previously reported experiences, a method of repair is then determined.

### *Extrahepatic Ductal Injuries*

The ductal injuries reported here were due equally to penetrating and nonpenetrating trauma. Those owing to penetration are usually more lethal due to the involvement of associated injuries to the liver, duodenum, pancreas, and other organs. On the other hand, injuries stemming from blunt trauma often present with a far different clinical picture, and these patients may relate a vague history of blunt trauma to the upper abdomen occurring days or weeks previously. This is followed by a variety of symptoms which may include jaundice, inanition, nausea, vomiting, abdominal distension, ascites, acholic stools, recurrent pain, and mild temperature elevation. The pathophysiology which underlies the fact that a patient can tolerate a transected bile duct with minimal symptoms despite the intra-abdominal collection of large amounts of bile is not completely clear. However, there are both anatomic and bacteriologic considerations which may explain this. First, a significant portion of the bile may remain or dissect into the retroperitoneum which could mask overt intra-abdominal pathology. With time the disruption of the duct may gradually seal over with the remaining intraperitoneal bile being resorbed. Secondly, and perhaps more importantly is the observation that intraperitoneal bile which is sterile is tolerated quite well. Cohn in 1960 showed experimentally in dogs that if the bile was infected it be-

TABLE 5. *Results per Type of Repair*

| Injury | No. | Operation | No. of Patients Requiring Secondary Repair | Mean Interval Requiring Secondary Repair (Mo) | Follow-up (Mo) |
|---|---|---|---|---|---|
| Complete transection | 5 | Primary end-to-end | 5 | 6 | |
| | 3 | Primary duct–enteric | 0 | — | 22 |
| Incomplete transection | 4 | Primary repair (T-tube) | 0 | | 56 |
| | 1 | Primary repair (No T-tube) | 0 | | 48 |

came lethal when leaked intraperitoneally, but if sterilized by antibiotics, there was a mild chemical peritonitis with low mortality.[7]

In those cases with bile duct injury due to blunt abdominal trauma the most frequent site of injury is at the pancreaticoduodenal junction and secondarily at the bifurcation of the hepatic duct or at the origin of the left hepatic duct.

The hypothesis for the mechanism of ductal injury in blunt abdominal trauma is related to three factors:[10] 1) the bile ducts are rigid and quite susceptible to a shearing force, especially if the gallbladder empties rapidly into them as a result of increased external pressure, 2) the shearing force exerts its injury at the sites of maximum fixation which are at the points where the common bile duct enters the liver or pancreas, and 3) since the bile duct is shorter and more fixed than the portal vein and hepatic artery, it receives preferential injury. Fish and Johnson[9] also postulated that the blunt trauma in the right upper quadrant compresses the costal cartilages and upper abdomen, driving the liver cephalad in relation to the fixed pancreas and duodenum, thus causing a tear at the point just above the pancreas. All of our cases of ductal injury owing to blunt trauma were located just above the pancreas as might have been predicted by these hypotheses.

In a discussion on delayed diagnosis of bile duct injury from some cases of blunt trauma, Skow and Longmire[26] postulate that either a small tear in the duct may cause inflammation, fibrosis, and eventual stricture, or that traction may cause disruption of the muscularis of the duct without mucosal rupture and eventual healing of this muscle layer could produce late stricture. Experimental work by Casey and Peacock[5] presented convincing data that wound contraction after injury, rather than collagen synthesis, was responsible for bile duct strictures. They also showed that excessive tissue damage and repair of bile ducts under tension substantially increased the likelihood of biliary tract stenosis subsequent to repair, both by increased wound contraction.

The most important factor in determining how to manage the bile duct injury depends on whether or not the duct is completely or incompletely transected. From our experience complete transection almost always ends with stricture if the duct is repaired primarily, but has a favorable outcome if some type of duct–enteric anastomosis is performed. These findings were reported by Belzer[1] some years ago when he showed that an incomplete ductal injury could be successfully repaired by duct anastomosis or patch (vein or gallbladder graft) however, a transection when mobilized for primary anastomosis or patch almost always ended in stricture. Indeed Longmire[16] in 1966 recommended duct–enteric anastomosis as the best method in the early management of injuries to the extrahepatic ducts.

TABLE 6. *Portal Vein Injury*

| Injury | No. of Patients | Repair | Mortality (%) |
|---|---|---|---|
| Incomplete transection | 4 | Lateral venorraphy | 1 (25) |
| Complete transection | 1 | End-to-end anastomosis | 1 (100) |
| | 1 | Splenic vein bypass | 1 (0) |
| | 1 | Saphenous vein interposition | 1 (0) |
| | 1 | Ligation | 1 (100) |
| | 2 | Died in operating room before repair accomplished | 2 (100) |

If the duct has been perforated or incompletely divided, primary repair can be successfully performed. There seems to be no definitive evidence that the presence or absence of a T-tube stent makes any difference in the rate of success. However, we feel that a T-tube stent should not be used if the duct is of a small caliber.

### *Hepatic Arterial Injuries*

In 1910, Burton-Opitz[4] reported that the liver could survive on portal blood alone because it supplies 80% of the total hepatic blood flow. Trygstrep and coworkers[27] showed that the hepatic artery provides 30% of the flow but 50% of the oxygen delivery.

While there have been isolated case reports[17] of liver necrosis after hepatic artery ligation with an intact portal vein, this is distinctly unusual. If the injury to the porta hepatis involves only the common hepatic artery or its primary branches, and if repair is not very straightforward then ligation should be performed. In those patients with cirrhosis or severe preexisting liver disease hepatic artery ligation should be avoided, since the livers in these patients depend on increased arterial flow. The safety of this technique has been supported in several reports on the management of liver trauma without undue side effects.[11,15] Usually it is quite predictable that if the portal vein and artery are both injured, the liver will suffer ischemia and possible subsequent necrosis.[17]

All four of the hepatic artery injuries managed by ligation in the present series resulted from gunshot wounds. The only death occurred in a patient who had transection of the common hepatic artery and incomplete disruption of the portal vein. This patient died in shock in the Emergency Room before any specific therapy could be instituted. In the patient who has a combined injury of the hepatic artery and portal vein,

TABLE 7. *Results of Collective Series of Management of Portal Vein Injuries*

| Series | Year | Method of Repair / No. Patients (Survivors) | | | | |
|---|---|---|---|---|---|---|
| | | Lateral Repair | E-E Repair | Graft or Bypass | Shunt | Ligation |
| Bostich & Stone (Emory) | 1975 | 7 (4) | 3 (1) | 0 | 1 (0) | 8 (1) |
| Graham et al. (Baylor) | 1978 | 26 (16) | 1 (1) | 0 | 1 (1) | 4 (0) |
| Peterson et al. (UCSF) | 1979 | 19 (13) | 0 | 1 (1) | 0 | 5 (3) |
| Present Series (UCLA-Tulane) | 1979 | 4 (3) | 1 (0) | 2 (2) | 0 | 1 (0) |
| Overall mortality (per cent) | | 36 | 60 | 0 | 50 | 78 |

the surgeon will usually not be afforded much choice in operative procedures. In this situation after relative hemostasis has been established by the Pringle and/or other maneuvers, it is advised that the injured artery be ligated and attempts be made at portal vein repair. This technique has been reported by Fuller.[12]

*Portal Vein Injuries*

Our experience is similar to that described by other authors,[3,13,19,22,23] in that the mortality from portal vein injuries is usually about 50% and thus this type of trauma represents the most lethal to the structures in the portal hepatis.

Although the management of portal vein injuries can be most easily handled by ligation, there is considerable controversy concerning the eventual consequences of such a procedure. Historically, in 1856, Ore[21] first observed that sudden obliteration of the portal vein resulted in the death of three dogs within an hour. The pathophysiology responsible for the death was not clear, and Claude Bernard[2] in 1859 expressed the belief that death was due to exsanguination of blood from the brain and heart to the gut. However, in 1877, Schiff[25] postulated that death resulted from toxic substances in the blood usually destroyed by the liver. Johnstone[4] demonstrated in a dog model that acute ligation of the portal vein reduces circulating blood volume by 58% of the original volume. He concluded, however, that death was not due solely to a decrease in circulating blood volume. Pilcher[24] reported that the total hepatic flow in dogs is approximately 1–2 L/minute and that the hepatic artery constitutes 20–35% of the flow. He also found that the hepatic veins contribute 1–1.5 L/minute to the total inferior vena caval flow which is 2.5–2.7 L/minute, thus up to 50% of inferior vena caval return is from the hepatic veins. This information may be helpful when managing patients after acute ligation of the portal vein in that if the patient shows a marked drop in central venous pressure after portal vein ligation then this indicates significant splanchnic pooling and the patient should be transfused to restore the central venous pressure to normal. Child[6] was the first to demonstrate that acute portal vein ligation could be tolerated in the primate when he achieved an 80% survival in monkeys. He subsequently performed portal vein ligation in six patients during pancreaticoduodenectomy.

We believe that the method of exposing the injured portal vein by medial reflection of the right colon and small bowel as described by Peterson[23] is probably the most useful. As indicated earlier, associated injuries, especially major vascular trauma, may dictate the method of emergency control, but if the vein is the principal acute injury, then packing and clamping the porta hepatis, with eventual reflection of the right colon, duodenum and pancreas, allows access to the injured vein. Sometimes it is necessary to approach the vein from the left by reflecting up the tail of the pancreas and splenic vessels as a "handle" to view this aspect of the vein.

The methods of repair used in this series are listed in Table 6, and a summary of combined experience reported in the literature is given in Table 7. From our own experience, as well as that of others, we believe that, whenever possible, portal vein injuries should be repaired either with lateral or end-to-end repair, or with a bypass or vein graft. Despite successes in selected cases[22] overall experience with acute ligation and portasystemic shunts has been very discouraging; a portasystemic shunt in a patient with previous normal hepatic flow will undoubtedly cause severe encephalopathy.[8,18] Enthusiasm for ligation has recently emerged in the belief that most patients with irreparable injury will survive only if ligation is successful. In this report[22] it is stressed that the majority will survive ligation without the necessity for a shunt in 24–48 hours. Perhaps ligation heretofore was used only in desperate situations, nevertheless these earlier reports indicate an extremely low survival rate after this procedure. We believe that until further evidence is forthcoming, every method should be employed to prevent ligation. Experience with pressure measurements after ligation as well as follow-up studies to determine vein patency after repair, and the occurrence of portal hypertension are essential before final conclusions can be drawn.

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### Discussion

Dr. William A. Neely (Jackson, Mississippi): At the University of Mississippi Medical Center we manage a substantial number of severely injured patients on our trauma service, and injuries to the liver and the porta hepatis are not uncommon. However, I will limit my remarks to a single case, though we have had an additional patient who required duodenal resection.

The patient was a 26-year-old man who walked into our emergency room after having been involved in a motor vehicle accident where his motor scooter struck the rear of a large truck. He went up under the truck, and he was struck in the abdomen. Struck in the upper abdomen, he felt severe pain, and vomited once.

Physical examination revealed generalized tenderness, with some rigidity and absence of bile sounds. At laparotomy, a long and deep laceration of the left lobe of the liver and repaired. The common duct was severed just distal to the level of the cystic duct, and the head of the pancreas and the duodenum were so macerated that the specific ductal structures could not be clearly identified.

(Slide) Accordingly, we ligated the distal common duct, and performed the resection and anastomosis shown in this slide. The duodenal stump was closed. A partial gastrectomy, a cholecystojejunostomy, a gastroenterostomy, an enteroenterostomy, and a choledochostomy with T-tube were performed as illustrated.

After a month of hospitalization complicated by staphylococcal pneumonia, the patient was discharged. I have seen this patient several times since he was discharged from the hospital.

Dr. David B. Skinner (Chicago, Illinois): I would particularly like to address the difficult problem of the occult bile duct injury. In this paper, we heard that 3 of 7 patients with blunt abdominal trauma had a delay in the diagnosis for up to one year. Five years ago, I was asked to operate on a 27-year-old man who held the same highly spiritual commitment that Dr. Griffen's patient did, who was bleeding from esophageal varices on several occasions. We had planned to do a portacaval shunt on this man.

At operation, we found a mass behind the head of the duodenum. In exploring this further, a bile lake was encountered. We opened the duodenum and explored the common duct through the ampulla, and found a transection of the duct, with a connection through a biliary fistula in the retroperitoneum, and dilatation of the proximal common duct.

Based on these findings, we changed the operative course, and did a common duct reconstruction to the duodenum. Postoperatively, close questioning revealed that this man had been involved in a motor accident two years before, had suffered blunt abdominal trauma, was observed in hospital for three days, and then discharged. He gradually then showed progressive deterioration of liver function, attributed to alcoholism.

After the common duct reconstruction, he has now gone for five years with no further bleeding, with a reversal of his portal hypertension, and with microscopic reversal of his biliary cirrhosis, confirmed by needle biopsy. The long-term consequences of a missed bile duct injury are, obviously, highly significant, but are reversible even at a later time.

Dr. H. Harlan Stone (Atlanta, Georgia): These wounds present a great challenge. (slide) On reviewing our experience with portal venous injuries, a large number were found to have associated major vascular trauma, such as wounds of the aorta and of the vena cava. These other wounds have required primary attention, so we have preferred to put a circular, noncrushing vascular clamp across the porta hepatis to control the portal venous bleeding until the injury to the aorta or cava has been corrected. As Dr. Cerise has stressed, associated injuries are often the very reason for the patient's death.

With respect to suture of the portal vein, we routinely attempt