

Treatment of Traumatic Hemobilia*

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ALTHOUGH Owen²⁵ first described a patient who probably had traumatic hemobilia, he did not recognize that the blood from the hematoma in the liver probably entered the gastrointestinal tract via the biliary tract. He postulated that the gastrointestinal bleeding was from a "more or less congested state of the mucous membrane throughout." This original case and those of Siegel in 1909²⁹ and Sandblom in 1948²⁷ are not proved to be cases of hemobilia. The bleeding in Siegel's and Sandblom's patients was secondary to blunt trauma and both patients had distended gallbladders which, at operation, were filled with clotted blood. In Siegel's case the gallbladder had become gangrenous and was removed. There were liver lacerations in this case but liver lesions are not described in Sandblom's case. These cases are not proved to be hemobilia secondary to a hepatic cavity with hematoma or arteriobiliary or venobiliary fistulae and should not be considered in attempts to analyze modes of treatment. Only cases with central rupture or laceration of the liver with blood in the biliary tract or in the gastrointestinal tract

are pertinent. In 1919 Hitzrot¹⁹ described such a case, as did Strauss³⁴ and Thorlaxson and Hay³⁷ in 1929. Sandblom²⁷ in 1948 first gave this condition its name.

Hemobilia may result from foreign bodies, such as T-tubes and calculi, neoplasms, and hepatic arterial aneurysms. Trauma, however, is the greatest factor. Thirty-five cases of traumatic hemobilia from the world literature, including two of our own (one previously reported by Shatzki²⁸), are analyzed (Table 1) in an attempt to assess the optimal mode of surgical therapy. In the past treatment of traumatic hemobilia consisted of a multiplicity of operations which circumvented direct attack upon the lesion. Nondefinitive operations such as cholecystostomy, cholecystectomy, choledochostomy and gastroduodenal procedures have been employed, mostly because of lack of understanding of the pathogenesis.

After blunt trauma to the upper abdomen, development of intermittent, severe, colicky, abdominal episodic pain after a latent period, followed by melena or hematemesis or both, sometimes mild transient obstructive jaundice, lead to the *diagnosis*. The problem is preoperative or operative *localization of the source of bleeding* and direct attack upon this lesion. The plan of attack should include general hypothermia and hepatic inflow occlusion^{4, 38} to allow approach to the hepatic lesion with control of bleeding from liver surfaces. The direct approach includes resection of the involved

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TABLE 1. Summary of 35 Cases of Traumatic Hemobilia

Author Year	Age Sex	Type Trauma	Type of Liver Pathology	Interval Between Injury and Hemobilia	Location of Hepatic Cavity	Surgical Treatment	Result
Hitzrot, 1917	32 M	Blunt	Central rupture	3-4 mo.	Not described	Opening of cavity and gauze packing	Cured
Strauss, 1929	19 M	Blunt	Central rupture	12 da.	Not described	None	Died. At autopsy, intrahepatic hematoma communicating with hepatic ducts.
Thorlakson & Hay, 1929	45 M	Blunt	Lacerated liver	8 da.	Right lobe	None	Died
Wulsten, 1931	11 F	Blunt	Central rupture	4 wk.	Right lobe	None	Died
Hermanson & Cabitt, 1934	16 M	Blunt	Lacerated liver	4 wk.	Right lobe	Three operations: 1) gauze packing of liver laceration, 2) feeding jejunostomy, 3) duodenal exclusion and gastrojejunostomy	Died
Hawthorne, Oaks & Neese, 1941	Adult M	Blunt	Lacerated liver	7-11 wk.	Not described	Cholecystectomy and exploration of common duct	Recovered
Ireneus, 1942	14 F	Blunt	Central rupture	6 wk.	? Right lobe	Cholecystostomy	Recovered
deMoraes Grey, 1947	10 M	Blunt	Central rupture	2 wk.	Not described	Choledochostomy	Died
Burnett, Rosemond, Caswell & Hall, 1949	7 M	Blunt	Lacerated liver	9 da.	Dome of liver	Six operations: 1) suture of liver laceration, 2) exploratory laparotomy, 3) evacuation of blood clots from subphrenic space, 4) choledochostomy, 5) cavity opened, vessels sutured, cavity drained, 6) cavity re-opened and packed with gauze, 7) packing removed.	Cured

TABLE 1 (Continued)

Author Year	Age Sex	Type Trauma	Type of Liver Pathology	Interval Between Injury and Hemobilia	Location of Hepatic Cavity	Surgical Treatment	Result
Hart, 1950	15 M	Blunt	Lacerated liver	5 wk.	Not described	Three operations: 1) liver lacerations drained, 2) exploratory laparotomy, 3) choledochostomy.	Recovered
Bigger, 1950	35 M	Blunt	Lacerated liver	6 wk.	Right lobe	Two operations: 1) liver laceration packed, 2) evacuation of subphrenic clots and packing.	Died 3½ yr. after injury of recurrent hemobilia
Epstein & Lipschutz, 1952	9 M	Blunt	Lacerated liver	2 wk.	? Right lobe	Two operations: 1) liver laceration sutured, 2) choledochostomy.	Recovered
Sparkman, 1953	3½ M	Blunt	Lacerated liver	2 wk.	Right lobe	Two operations: 1) liver laceration sutured and drained, 2) cavity opened and packed.	Cured
Francioli, Jost & Saegesser, 1954	9 M	Blunt	Lacerated liver	5 wk.	Right lobe	Three operations: 1) liver lacerations sutured, 2) side-to-side portocaval shunt, 3) cavity opened and vessel sutured, then packed and liver laceration re-sutured.	Cured
Broker & Hay, 1955	20 F	Blunt	Lacerated liver	3 wk.	Right lobe	Two operations: 1) liver packed, 2) choledochostomy and liver repacked.	Died
Bariatti, 1955	21 M	Blunt	Central rupture	6 da.	Left lobe	Splenectomy; no operation on liver or biliary tract.	Died
Gombkötö, 1957	26 M	Blunt	Lacerated liver	24 da.	? Right lobe	Three operations: 1) liver laceration sutured, 2) packing and drainage of right subphrenic space, 3) choledochostomy & choledochostomy.	Recovered
Thomeret, Dubost, Dubray-Vautrin & Cabrol, 1957	25 M	Blunt	Lacerated liver	2 wk.	Right lobe	Three operations: 1) liver laceration packed, 2) choledochostomy, 3) right hepatic lobectomy.	Cured

TABLE 1 (Continued)

Author Year	Age Sex	Type Trauma	Type of Liver Pathology	Interval Between Injury and Hemobilia	Location of Hepatic Cavity	Surgical Treatment	Result
Spector, 1957	43 M	Blunt	Lacerated liver	2½ wk.	Right lobe	Four operations: 1) liver laceration packed, 2) same plus cholecystostomy, 3) liver laceration packed, 4) ligation, right hepatic artery.	Cured
Mörl, 1958	6	Blunt	Lacerated liver	3 wk.	Both lobes	Five operations: 1) liver laceration sutured and packed, 2) drainage of subphrenic abscess, 3) drainage of pelvic abscess, 4) liver laceration packed, 5) splenectomy & liver laceration packed.	Died
Goffi, Fanganiello, Borges & Bastos, 1959	22 M	Blunt	Central rupture	9 da.	Left lobe	Two operations: 1) exploratory laparotomy, exploratory gastro-duodenotomy, 2) left hepatic artery ligation.	Cured but necrosis, left lobe of liver
Sworn, 1959-1960	34 M	Blunt	Central rupture	24 da.	Right lobe	Two operations: 1) cholecystectomy & choledochostomy, 2) exploratory laparotomy & cholangiogram.	Died
Ingelrans, Lacheretz & Saint- Aubert 1960	10 M	Blunt	Lacerated liver	12 da.	? Right lobe	Three operations: 1) ligation, vessels in liver laceration, liver laceration packed and drained, 2) cholecystostomy, 3) exploratory laparotomy.	Recovered
Saliba, Sawyer & Sawyer, 1960	14 M	Penetrating	Lacerated liver	9 wk.	Right lobe	Three operations: 1) liver sutured, 2) choledochostomy, liver laceration packed, 3) suture, branch of hepatic artery in cavity with drainage.	Cured
Markgraf, 1960	42 M	Penetrating	Lacerated liver	13 da.	Both lobes	Cholecystectomy	Died

TABLE 1 (Continued)

Author Year	Age Sex	Type Trauma	Type of Liver Pathology	Interval Between Injury and Hemobilia	Location of Hepatic Cavity	Surgical Treatment	Result
Steichen, 1960	39 M	Blunt	Central rupture	10 da.	Both lobes	Left hepatic lobectomy	Died
Schatzki,* 1961	4½ F	Blunt	Central rupture	2 wk.	Left lobe	Two operations: 1) left hepatic artery ligation, 2) left hepatic lobectomy.	Cured
Gynn & Reynolds, 1961	74 M	Blunt	Hepatic artery aneurysm	3 mo.	Left hepatic artery—right hepatic duct fistula	Two operations: 1) cholecystectomy, choledochostomy, 2) right hepatic duct transected and ligated.	Died
Moretz, 1961	31 M	Penetrating	Lacerated liver	2 wk.	Right lobe	Three operations: 1) cholecystectomy, 2) right hepatic artery ligation, 3) hepatic cavity opened and packed.	Cured
Fallis & Stephens, 1961	13 M	Blunt	Lacerated liver	16 da.	Right lobe	Right hepatic artery ligation.	Cured
Detrie, 1962	31 M	Blunt	Lacerated liver	5½ mo.	Both lobes	Four operations: 1) cholecystectomy, 2) ligation of common hepatic artery, 3) drainage of subhepatic hematoma, 4) resection of biliary-bronchial fistula with drainage.	Cured
Souliotis, Pettigrew & Chamberlain, 1963	6½ F	Blunt	Lacerated liver	17 da.	Right lobe	Two operations: 1) liver laceration sutured and drained, 2) cholecystectomy, choledochostomy, opening of cavity and drainage, ligation of right hepatic artery (hypothermia).	Cured

TABLE 1 (Continued)

Author Year	Age Sex	Type Trauma	Type of Liver Pathology	Interval Between Injury and Hemobilia	Location of Hepatic Cavity	Surgical Treatment	Result
Amerson & Ferguson, 1963	11 M	Blunt	Lacerated liver, right lobe; central rupture, left lobe.	18 da.	Left lobe	Three operations: 1) suture of liver laceration, 2) choledochos- tomy, 3) left hepatic lobectomy.	Died
Amerson & Ferguson, 1963	39 F	Penetrating	Lacerated liver	52 da.	Left lobe and quadrate lobe	Three operations: 1) liver laceration sutured and drained, chole- cystectomy, 2) lysis of adhesion for bowel obstruction, 3) liver cavity opened and left hepatic vessels and junction of hepatic duct sutured, liver tissue re- sutured, quadrate lobe resected.	Cured
Wheelan & Gillespie, 1964	20 M	Blunt	Lacerated liver	4 da.	Right lobe	Four operations: 1) liver laceration sutured and drained, 2) sub- phrenic abscess drained, 3) he- patic cavity packed, 4) right he- patic lobectomy.	Died

* This case, reported by Schatzki, is Case 1 in this report and was treated on the General Surgery Service, Walter Reed General Hospital.



FIG. 1. Case 1. Posterior displacement of stomach is noted on lateral view of gastrointestinal x-ray series.

liver, opening of the cavity with suture of vessels and bile ducts from within and right or left hepatic artery ligation in certain instances.

Two patients treated at Walter Reed General Hospital in the past 4 years illustrate that direct surgical attack on the hepatic lesion is most likely to yield good results.

Case Report

Case 1. A 4½-year-old white girl was thrown against the dashboard in an automobile accident in Germany on June 2, 1960. A minor scalp laceration was sutured at a local dispensary and the patient was sent home. During the following week she complained of vague abdominal pain and listlessness. Two weeks after the accident hematemesis and melena suddenly developed following severe, colicky, upper abdominal pain and she was admitted to a local hospital. Blood transfusion was given. Jaundice and hepatomegaly without abnormal liver function tests were found. Recurrent gastrointestinal hemorrhages occurred at approximately 10-day intervals. The patient was transferred to a larger U. S. Army Hospital in Germany.

The upper gastrointestinal bleeding continued and on July 3, 1960 the abdomen was explored. Blood was found in the lumen of the entire large and small bowel and there was a mass in the left lobe of the liver. The surgeon considered the

diagnosis of hemobilia due to a left hepatic hematoma and ligated the left hepatic artery. Six days later, however, crampy abdominal pain, hematemesis and melena recurred and again became intermittent at 10-day intervals. The patient was then transferred to Walter Reed General Hospital, arriving on September 2, 1960. During the 3 months prior to admission to Walter Reed General Hospital, she received 18 units of whole blood and had three serious episodes of shock secondary to gastrointestinal bleeding.

On admission, physical examination was essentially normal. The abdomen was soft, flat and nontender, and the liver was palpable two fingerbreadths below the right costal margin. Laboratory data showed hemoglobin 12.5 Gm./100 ml. and hematocrit 37%. Urine and liver function were normal except for prothrombin activity of 47%, which returned to normal following Vitamin K administration, and serum alkaline phosphatase level of 23 King-Armstrong units. Upper gastrointestinal x-ray series showed posterior displacement of the body of the stomach by a mass thought to be the left lobe of the liver (Fig. 1). Esophagoscopy was normal. On September 9 the patient was re-explored. A 5-cm. indurated mass within the left lobe of the liver, projecting toward the posterior surface, was found. Operative cholangiogram revealed a large cavity in the left hepatic lobe in direct communication with a large radical of the left hepatic duct (Fig. 2). Aspiration of the mass in the left lobe of the liver yielded old blood and substantiated a diagnosis of a central hematoma cavity in the left hepatic lobe. Left hepatic lobectomy was performed, during which a fragment of sequestered liver tissue was found in the left hepatic duct. The postoperative course was uncomplicated. There was no further bleeding. Postoperative liver function tests remained

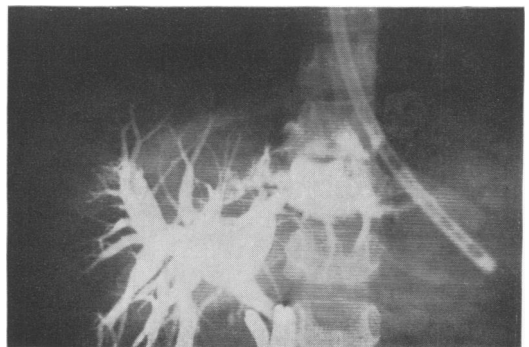


FIG. 2. Case 1. Operative cholangiogram reveals communication from left hepatic duct to a cavity in left hepatic lobe. Bulldog vascular clamp on distal common duct allows for filling of the proximal biliary tract.

normal. She was discharged during the third postoperative week. She remains asymptomatic and in good health 3½ years later.

Comment. Central rupture of the left lobe of the liver occurred following blunt trauma in this child. She developed a classical picture of hemobilia beginning 2 weeks after injury. Left hepatic artery ligation failed to control bleeding into the biliary tract and left hepatic lobectomy became necessary, resulting in cure.

Case 2. A 20-year-old white Air Force enlisted man was admitted to Walter Reed General Hospital on October 28, 1963 from an Air Force Hospital in Plattsburg, New York. The patient had been involved in an automobile accident at midnight on October 25, 1963. He sustained multiple fractures of the right femur, distal right fibula and right medial malleolus; multiple lacerations of the right leg, scalp, wrist and chin; cerebral concussion; and severe closed injury to the liver. Resuscitation from profound shock with dextran and aramine was in part successful and exploratory laparotomy was performed 3½ hours after the accident. Two thousand cubic centimeters of blood was found in the peritoneal cavity. There were four separate lacerations of the right lobe of the liver which were sutured without debridement or exploration of the depths of the wounds. The deepest of these lacerations was drained with a No. 16 French catheter. Three Penrose drains were used for drainage of the perihepatic spaces. The patient received 3,000 cc. of blood for an estimated 3,500 cc. blood loss. Vital signs were normal at the conclusion of the procedure. Within 24 hours, however, acute renal failure developed. There was no response to mannitol administration. Hematocrit decreased from 45 to 34% by October 27. Transfer to Walter Reed General Hospital was effected for treatment of renal failure.

On admission blood pressure was 135/85, pulse 86 per minute, temperature 37.7° C. A nasogastric tube was draining gross blood and later gastric fluid with guaiac positive coffee-ground sediment. The patient was lethargic but oriented. The abdomen was moderately distended and drains were present as noted above. A right-sided Kehr incision appeared to be healing well. Bowel sounds were hypoactive. There was poor expansion of the lung bases.

The liver drains were advanced beginning on October 29 and were completely removed by November 2. BUN which on admission was 70 mg./100 ml. steadily rose to 166 mg./100 ml. on October 31, and peritoneal dialysis was begun on

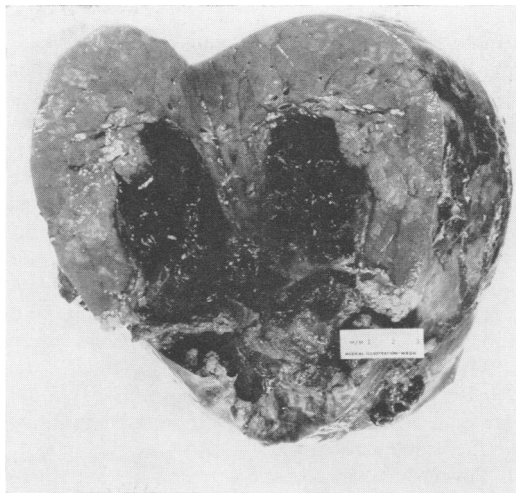


FIG. 3. Case 2. Transected gross specimen of right hepatic lobe. Large hematoma cavity filled with clots communicating with torn liver capsule is noted. Lighter zone around hematoma represents softened necrotic liver tissue.

the following day. Within 24 hours BUN was 100 mg./100 ml. and the serum potassium level was 4.5 mEq./L. Urinary output continued in oliguric ranges—about 100 cc. per day. On both October 30 and 31 there were varying amounts of blood draining from the stomach. Hematocrit dropped to 28%.

On November 3 he became febrile and signs of a right subphrenic abscess prompted incision through the bed of the eleventh rib and drainage of a large collection of thin pus and blood-stained bile on November 4. *Klebsiella aerobacter* was cultured from this material. The postoperative course was complicated by secondary hemorrhage from the liver through the drainage wound on November 6. This prompted temporary Gelfoam and gauze packing of a large cavity in the right lobe of the liver. This cavity represented the depths of at least one of the large lacerations extending from the superolateral aspect of the right lobe into and through about two thirds of the thickness of the liver at this point. Again blood was noted coming from the gastric tube. Because external bleeding continued despite the packing, right hepatic lobectomy was performed on November 7 under hypothermia and general anesthesia. This was accomplished with control of the bleeding. The left lobe of the liver appeared normal. Twenty-two units of blood were required during the operation. The specimen of the right hepatic lobe showed diffuse softening and autolysis with a large clot-filled cavity occupying most of the lobe (Fig. 3).

His postoperative course was encouraging dur-

ing the first 34 hours. Vital signs were stable; hematocrit was between 44 and 49%, and blood volume was 4.9 to 5.9 L. There was no further bleeding from the wound or from the gastric tube. Urine output rose to 500 cc. on November 8, the first postoperative day.

At 8:30 a.m., November 9, gram-negative septicemia supervened with profound shock, metabolic acidosis and death within 10 hours. Antibiotic agents administered during hospitalization were Streptomycin 0.25 Gm. every third day. Chloramphenicol 1.0 Gm. every six hours, and Albamycin 0.5 Gm. every six hours. On November 9 Colymycin 0.2 Gm. was given as an initial dose. Despite the usual supportive measures death occurred at 6:35 p.m. Blood cultures were positive for *Klebsiella aerobacter*.

Postmortem examination revealed that there had been no further bleeding into either the peritoneum or the biliary tract and that death had been caused by septicemia. *Klebsiella aerobacter* was grown from right heart blood, the right pleural cavity and the peritoneal cavity.

Comment. Hemorrhage from the liver had been controlled with resectional therapy. The outcome might have been favorable had it not been necessary to perform the resection across an already infected field.

Pathogenesis

Hemobilia results from trauma which causes 1) central liver rupture while the capsule remains intact or 2) laceration of the liver including the capsule when the capsule either is sutured or heals spontaneously, producing in effect a subcapsular lesion. Hepatic tissue is disrupted by the blow and blood and bile accumulate in an intrahepatic cavity. Autolysis and further pressure necrosis of liver tissue enlarges this cavity. Since there can be no decompression of this blood-bile mixture through the liver capsule, decompression occurs through the biliary tract. From the periodicity of bleeding, in many reports, it appears that the cavity decompresses into the bile ducts after blood, bile and clots accumulate under pressure, then a period of time elapses during which the cavity fills again.

Ten of 35 cases resulted from central liver

rupture whereas 24 resulted from liver lacerations which had sealed spontaneously or been sutured, leaving a cavity in the depths of the liver. The clinical course varies in the two entities. With central liver rupture there was injury to the abdomen or lower chest. Vague abdominal pain is usually present but is not severe enough to suggest intra-abdominal catastrophe. Early operation is not indicated and the first suggestion of trouble is the initial episode of gastrointestinal bleeding following a bout of upper abdominal, colicky pain. The average interval between injury and initial bleeding is 4 weeks, the earliest is 6 days and the longest, 3 to 4 months.

In contrast to the insidious onset of hemobilia in central liver rupture, the course in laceration of the capsule is more dramatic. Here there is the need for early surgical intervention to control hemorrhage into the peritoneal cavity. If this laceration of the capsule is repaired without approximation of deep tissues, a potential cavity from which hemobilia may arise is created. If approximation of deep hepatic tissues is impossible because of loss or friability of disrupted tissue, major resection must be considered or, if not feasible, the depths of the laceration should be drained to the outside. Spontaneous sealing of a liver laceration may sometimes be brought about by abutment against an adjacent organ or the diaphragm. The interval between injury and hemobilia is the same as in central liver rupture, averaging 4 weeks, with the earliest 4 days and the longest 5¼ months.

Localization of the Hepatic Lesion

Preoperative demonstration of the location of the hepatic cavity or the arterio-biliary fistula allows for planning the surgical approach. A cavity in either lobe suggests the possibility of resection of that lobe, whereas a centrally-located cavity suggests opening of the cavity and suture control of bleeding vessels within it.

There are, at present, three means by which preoperative localization can be attained.

The first is a *radioisotope scanning* of the liver. Just as tumors may be demonstrated by this technic, so can blood-filled cavities within normal liver substance.

Hepatic angiography has been utilized at the time of operation^{9, 35} but not preoperatively. At the time of operation the possibility of damage to the artery has been raised.¹⁵ Increasing use of selective angiography with visualization of the celiac artery and its branches makes this examination a potentially important preoperative study. Hepatic artery aneurysm has been identified as has leakage of dye from a bleeding artery in a cavity wall. In one case¹⁵ the lesion was a fistula between a branch of the left hepatic artery and the right hepatic duct. If extrahepatic hepatic artery ligation should become necessary, accurate information regarding the involved branch would be very helpful.

Splenoportography is the third preoperative study which might help to localize the lesion. This should be done only if celiac angiography is normal. Most bleeding vessels in hemobilia have been arterial although venobiliary fistulae have been reported^{18, 22} and splenoportography can localize some of these lesions.

If the foregoing studies do not demonstrate the lesion, then the hematoma cavity must be delineated at operation by palpation, needle aspiration and operative cholangiogram. To insure adequate filling of all branches of the hepatic ducts by cholangiography, a bulldog vascular clamp is placed across the distal common duct and 10 to 20 cc. of radiopaque material is injected. In our first case, palpation of a mass in the left lobe, needle aspiration of old blood from a cavity in this lobe and roentgenographic demonstration of contrast medium in a cavity indicated the site of hematoma for which left hepatic lobectomy was done. When there is active bleeding from the

TABLE 2. *Results of Nondefinitive Procedures**
(*Influence of Age*)

	Died	Recovered
Children (under 16 years)	4	4
Adults (over 16 years)	8	2
Totals	12	6

* Biliary tract drainage was unsuccessful in 10 of 14 such procedures and was followed by another operation or death.

common duct at operation, selective occlusion of the left or right hepatic artery with the common duct open may aid in demonstrating which artery is bleeding. Active arterial bleeding from the common duct at operation is rare and cannot be relied upon to localize the bleeding lobe.

Results of Treatment

Of the 35 cases reviewed, 18 had non-definitive procedures performed such as cholecystectomy, cholecystostomy or choledochostomy. Of these 18, 12 patients succumbed while six recovered some time after further episodes of hemobilia. These must be considered spontaneous arrests of bleeding with little effect from operative intervention. Of the six recoveries four were in children below the age of 16 and only two in patients over 16. Of 12 deaths four occurred in patients under 16 years and eight patients were older (Table 2). Spontaneous arrest of bleeding, therefore, seems more likely to occur in children than in adults. Detrie,⁹ in his analysis of 28 cases, reported similar observations.

Seventeen patients had definitive surgical procedures (Table 3). In seven the liver cavity was directly attacked by opening it, packing it or suturing vessels in the wall. The cavity was then drained. No patient in this group died. Five had hepatic lobectomies, of whom three died and two survived. Causes of these deaths are discussed under "Surgical Approach." Ligation of the right or left hepatic artery was performed

TABLE 3. Results of Definitive Surgical Treatment

	Died	Recov- ered
Opening of hepatic cavity		
With packing	0	5
With suturing	0	4*
Resection of liver tissue	3	2
Ligation, right or left hepatic artery	0	4**
Ligation, common hepatic artery	0	1
Total	3	16

* Two cases had both suturing and packing.

** Two additional ligations were performed but did not control hemobilia; required another definitive procedure.

on six occasions with no deaths and four cures. Two patients required further surgical procedures for recurrent bleeding.²³ ²⁸ In another patient the common hepatic artery was ligated and this patient survived but with a complicated postoperative course including recurrent hematemeses following heparin therapy for pulmonary embolus, subhepatic hematoma requiring drainage and thoracotomy for a resulting broncho-biliary fistula.⁹

Surgical Approach

As mentioned previously, 18 nondefinitive surgical procedures were performed. In 14 the biliary tract was drained either by cholecystostomy or choledochostomy. Only four of the 14 cases recovered. In the remaining ten, the drainage procedure was unsuccessful, and was followed by operation or resulted in death.

Direct operation on the liver is best performed under hypothermia and total hepatic inflow occlusion.^{4, 38} This renders the organ almost bloodless. Since air embolism may occur through hepatic veins,¹ a thoracic component to the incision will break the negative intrathoracic pressure seal. Incisions then may be made through rela-

tively bloodless liver tissue, either for resection or control of bleeding from the inner wall of the cavity. When resection is carried out there is no need to transect the organ entirely through normal tissue unless the laceration is fresh and the wall of the cavity is friable. When the wall of the cavity is fibrotic, the line of transection may enter the cavity at its medial wall, removing the cavity but leaving a portion of the firm medial wall as raw liver surface (Fig. 4). This maneuver preserves normal liver tissue and requires less cutting across normal liver tissue. Hemostasis in the medial wall of the cavity and in the remainder of the cut surface of the liver is by individual transfixing ligatures to vessels and ducts and large mattress sutures approximating liver surfaces. When necrosis and softening of the wall of the cavity is marked, sutures may tear out, in which case the line of hepatic transection must be through normal tissue. Resection should be performed if the cavity is confined to right or left hepatic lobes. That three deaths occurred in five patients having hepatic lobectomies requires explanation. In our second case resection had to be performed as a life-saving maneuver across a subphrenic space already infected with *Klebsiella aerobacter*. The patient tolerated the operation and was improved but then deteriorated after 34 hours from an overwhelming septicemia. Amerson and Ferguson's¹ patient died as the left lobe was being dissected due to air embolus through the left hepatic vein. The third death occurred in a patient who probably had a lethal wound with large necrotic cavities in both lobes. Left hepatic lobectomy was performed without awareness of the magnitude of the lesion in the right lobe—only appreciated at autopsy 3½ weeks later.³³

If the cavity is centrally located, resection is not possible. Again under hypothermia and hepatic inflow occlusion, normal liver over the cavity is incised, the interior of the cavity evacuated and its walls carefully

inspected. Release of the clamp on the portal triad permits identification of bleeding vessels in the wall of the cavity and control by transfixing sutures. Packing of the cavity also may be necessary. Such a cavity then should be drained to the outside until healing occurs from the depths. If filling with further clots is prevented the cavity will collapse and close within a few weeks. Drains should be shortened and removed over a 2 to 3 week period.

Because of poor condition of the patient, left or right hepatic artery ligation—depending upon the site of the lesion—may be performed. Two factors should be remembered: 1) two of six patients in whom this procedure was employed continued to have hemobilia and required further operation and 2) a stormy convalescence should be anticipated with possible liver slough through drainage sites, jaundice and fever. None of these patients died but three had difficult postoperative courses.^{13, 30, 32} In one other case the common hepatic artery was ligated without permanent sequelae⁹ and hemobilia was controlled.

Conclusions

Surgical procedures which do not attack the hepatic lesion directly should be abandoned. These include operations on the gallbladder and common bile duct. At the same time procedures which can control hepatic hematomas should be standardized.

From experiences of others and from our own, the following recommendations are made:

1) If the cavity is localized in either the left or right lobe: resection of that lobe under hypothermia and hepatic inflow occlusion.

2) If the cavity transcends the anatomic lobes in the central zone of the liver: incision into the cavity with evacuation of its contents and control of bleeding points with suture or by packing followed by external drainage, under hypothermia and hepatic inflow occlusion.

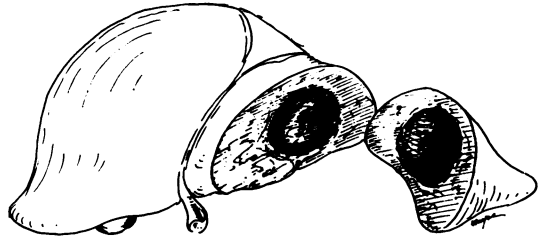


FIG. 4. Diagram of type liver resection described in text. Medial wall of cavity remains *in situ*; remainder of cavity is included in resected specimen.

3) If either of the aforementioned methods are not possible because of the general condition of the patient: hepatic branch artery ligation.

Summary

Thirty-five cases of traumatic hemobilia, including one not previously reported, are reviewed.

The importance of preoperative or operative localization of the hepatic lesion and the need for standardization of operative procedures for this lesion are emphasized.

Three operative approaches are recommended, all directed at the lesion within the liver. Indications for each operation are discussed.

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