# Traumatic Hemobilia *

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SANDBLOM introduced the term *traumatic hemobilia* to describe hemorrhage into the biliary tract preceded by liver injury.[28] A majority of 36 reported cases have presented a common clinical picture with only minor variations, in which biliary obstruction and severe colic have been associated with obscure gastro-intestinal bleeding (Table 1). Though the hemorrhage has often been minor or occult, instances in which it has become exsanguinating have been described. Traumatic hemobilia has only occasionally been reported, and clinical experience with the condition reflects a general unawareness of its occurrence.

Recently a patient of ours presented the diagnostic problems of traumatic hemobilia in a classic form, manifesting first occult, and later severe gastro-intestinal hemorrhage four weeks following suture of a superficial laceration of the liver resulting from blunt trauma. Whereas the correct diagnosis can seldom be made prior to operation, in this case the diagnosis was established and the disorder was cured by hepatic lobectomy.

## Case Report

On 8-27-62, O. P., a 22-year-old Negro male, was admitted to Harbor General Hospital following a freeway accident in which he sustained blunt abdominal injuries. At emergency laparotomy a laceration of the left lobe of the liver was found as were several ruptures of the capsule of the spleen, which required splenectomy. Hemostasis was obtained in the liver laceration by widely placed mattress sutures which approximated the margins upon a gelfoam sponge. The patient was discharged following an uneventful recovery.

Ten days after the accident he reported gross hematuria containing strings of clotted blood and complained of episodes of right flank colic. He was re-admitted to the hospital. The hematuria cleared spontaneously while the patient was undergoing urologic evaluation and he became asymptomatic without an abnormality having been found. The symptoms and hematuria were attributed to a contusion of the right kidney incurred at the time of the accident which resulted in late hematuria and colic associated with passage of pyeloureteral blood clots. An elevated serum alkaline phosphatase determination obtained at that time was ascribed to the recent hepatic injury.

Within ten days the patient was re-admitted for further evaluation of a new complaint of attacks of epigastric colic and the finding of occult blood in the stool. At examination, moderate deep abdominal tenderness was found localized to the right subcostal and epigastric areas without a palpable mass or enlargement or tenderness of the liver. The bowel sounds were active, and the stool was strongly positive for occult blood when tested with a guaiac reagent. There was no jaundice.

Laboratory studies revealed a five point drop in hematocrit since the previous admission, 16,000 white cell count, and elevated serum alkaline phosphatase level of 49 King-Armstrong units. Direct and total serum bilirubin determinations were 2.4 mg. per cent and 2.7 mg. per cent. Subsequently, additional studies which included urinalysis, determinations of serum glutamic-oxaloacetic acid transaminase, cephalin cholesterol flocculation, thymol turbidity, serum proteins, and hematologic tests to exclude a clotting disorder were within normal limits.

The patient's hospital course was characterized by persistent occult gastro-intestinal bleeding, transient periods of chemical jaundice and a pattern of rising serum alkaline phosphatase levels which were associated with recurrent attacks of

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right upper abdominal pain resembling biliary colic (Fig. 1). On the 29th day after the accident, a typical episode of biliary-like colic was followed by hematemesis which seemed to dramatically relieve the colic. At that time only a small hematocrit reduction occurred; however, several progressively more severe episodes of hematemesis during the next six days reduced the hematocrit significantly in spite of blood transfusions.

At that point our attention was called to the similarity of this case to classic reports of traumatic hemobilia. In particular, it resembled the case of Sparkman in which primary suture of a laceration of the liver was followed by hemobilia after a latent period of two and a half weeks.[32] Therefore, following negative sigmoidoscopy and barium contrast upper and lower gastro-intestinal roentgen examinations, an oral cholecystogram was obtained. It demonstrated several filling defects within the gallbladder which suggested the possibility of blood clots rather than calculi in a healthy young adult man (Fig. 2). With the support of these diagnostic studies, the increasing severity of the hematemesis and the patient's colic were considered to be consistent with the diagnosis of hemobilia and reoperation was believed indicated.

At operation the previously sutured laceration of the liver was incised, and a 3 × 7 cm. cavity was found beneath the scar with which a single large hepatic vein and several bile ducts communicated. A zone of softened liver parenchyma surrounded the cavity which contained fresh blood clot and bile. The extent of the rupture and parenchymal contusion beneath the laceration constituted almost complete severance of the lateral segment of the left lobe of the liver, extending from its inferior surface superiorly toward the dome.

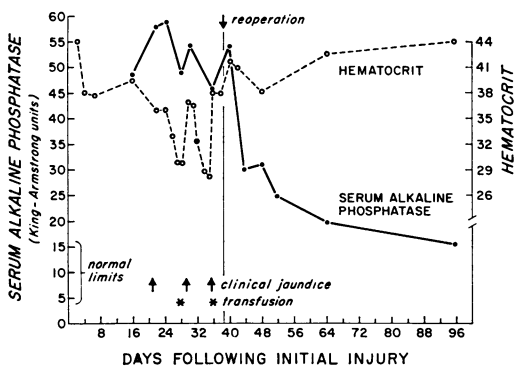


FIG. 1. Hospital course illustrating correlation of serum alkaline phosphatase levels and periods of clinical jaundice with progression of traumatic hemobilia.

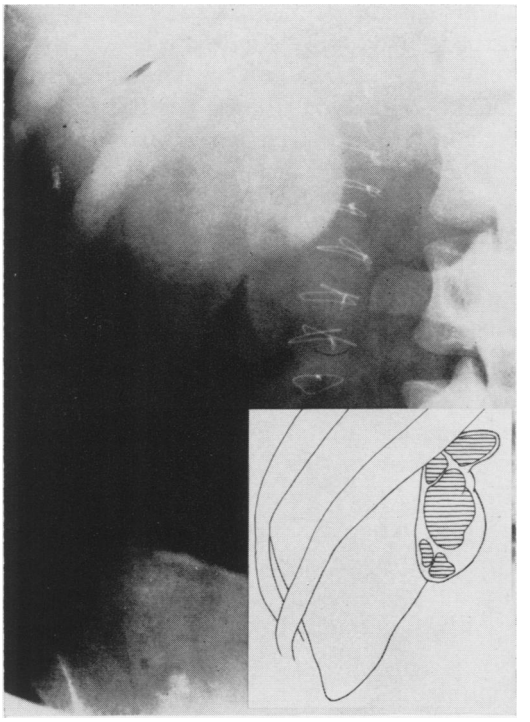

FIG. 2. Oral cholecystogram which reveals multiple noncalcific hemogenous filling defects within the gallbladder, believed to be blood clots.

A resection of the lateral segment of the left lobe containing the site of the hemorrhagic communication between the vascular system and biliary tract was accomplished without difficulty. The remainder of the liver appeared uninjured; however, the gallbladder was distended by blood clot. Examination of the intestinal tract revealed no injury or apparent source of gastro-intestinal hemorrhage.

The serum bilirubin level promptly returned to normal following the operation, and the serum alkaline phosphatase levels returned more gradually to normal. Filling defects noted in the gallbladder prior to operation were not present in an oral cholecystogram obtained 4½ months after operation. Since the operation the patient has been clinically well and has returned to work.

## Discussion

Sandblom has reviewed the mechanism by which hemobilia develops following liver trauma.[28] The disruption of relatively inelastic liver parenchyma, with rupture of bile ducts and large thin-walled vessels which have little capacity to contract, ac-

counts for the accumulation of devitalized tissue, blood and bile within a closed space following a central rupture or within the confines of an open injury that has been converted to a closed wound by suture or packing. In most instances these products are resorbed, and healing occurs by scar formation. Persistence of unresorbed material is reported to account for the occasional development of a bile cyst of the liver, or an abscess.[13] Autolysis and necrosis within a closed wound may permit these sequestered products to erupt into a large bile duct creating a hemorrhagic communication between the central injury and the biliary tract. By egress through the biliary tract, intrahepatic hemorrhage may enter the gastro-intestinal tract and become manifest.

Although traumatic hemobilia is primarily considered a complication of central rupture of the liver, it has occurred with equal frequency (16 cases) as a late complication following emergency treatment of small fractures of the liver and simple tears of Glisson's capsule (Table 1). The present case typifies this group. It should be anticipated that sheering and compression forces tending to break the liver upon the vertebral column and great midline vessels will produce more extensive injury beneath the localized wound than may be apparent at the time of emergency operation. The military experience of World War II with such injuries was large, and reviews by Madding and Burford indicate that drainage of all liver lacerations was an essential principle. The imperativeness of this principle in primary treatment of civilian injuries of the liver has become more evident as the rising incidence of blunt abdominal trauma associated with high speed travel has provided the civilian surgeon with increased experience with these injuries and their complications.

Clinically a latent interval precedes the development of hemobilia. This interval is less than four weeks in most instances, although it has ranged from 16 hours to three and one half years, and when long, hemobilia has appeared to be unrelated to an antecedent injury. Sandblom coined the term *traumatic hemobilia* by apt analogy to the more common traumatic hematuria, which is well illustrated in the present case. The passage of pyeloureteral blood clots was a latent sequella of renal contusion and was associated with several episodes of recurrent ureteral colic. The patient complained of both pain and gross hematuria containing blood clots. The patient with hemobilia complains of severe griping attacks of epigastric pain, or may present himself with hematemesis and severe gastro-intestinal hemorrhage. A tender enlargement of the liver or an upper abdominal mass may be present.

Pain resembling biliary colic has been a principal symptom of hemobilia, and it frequently heralds a hemorrhage. The pain is presumed to be due to the distension of bile ducts by blood proximal to an obstructing coagulum. The extent and duration of the biliary obstruction appear to account for the associated findings of transient jaundice and elevated serum alkaline phosphatase levels. It may be assumed that as long as the bile ducts remain obstructed by blood clot, pain will continue and gross bleeding will not be mainfest. When the clot is expelled, the pain is often dramatically relieved, and hemobilia becomes manifest as hematemesis or more commonly as copious melena from an obscure source. A pattern of recurrent obstruction and hemorrhage of this type has been common, and in some instances so consistent a sequence that patients have forewarned the physician of an impending hemorrhage. On occasion an impaction of blood clot within the gallbladder has been found associated, and we have found cases in which it has produced all the sequellae of acute calculous cholecystitis.[19, 30, 37] However, a recurring clinical course consistent with the passage of calculi through the bile duct

and transient biliary obstruction prior to hemorrhage has been more common.

The diagnosis of traumatic hemobilia is difficult and is rarely made preoperatively. Consequently, surgical intervention has often been undertaken with erroneous preoperative diagnoses, e.g., acute cholecystitis, pancreatitis, or peptic ulcer. Failure to appreciate the occurrence of this condition or difficulty in identifying a biliary source of upper gastro-intestinal bleeding have also been common at the time of exploration, and have led to *blind* resection of the stomach or cholecystectomy and numerous other procedures. These circumstances are mutely attested to by the lack of a correct diagnosis prior to most of a total of 55 operations performed in the 30 surgical cases reported. Recurrent hemobilia has commonly accounted for reoperation.

In the case presented, a preoperative diagnosis of traumatic hemobilia was suggested by the clinical course of periodic biliary pain associated with an obstructive factor manifest by jaundice and elevations of the serum alkaline phosphatase levels, prior to the episodes of gastro-intestinal hemorrhage. We believe serum alkaline phosphatase determinations were a useful and more meaningful index of the progression of hemobilia in this case than were the serum transaminase determinations, as reported by Saliba *et al.* Although not a specific test of liver function, such elevations are an accepted index of biliary obstruction if bone disease and neoplastic involvement of the liver can be excluded.[11] Oral cholecystogram was a particularly useful diagnostic adjunct in this case and demonstrated several large, homogenous filling defects which suggested blood clots within the gallbladder. We have been unable to find previous description of this observation.

Appropriate treatment of traumatic hemobilia is suggested by a review of reports of successful operations (Table 2). The variety of procedures indicates that cure of this condition depends upon accurate assessment of the injury at the time of exploration and individualized selection of an appropriate operation to control the source of hemorrhage.

Packing and drainage of the hepatic cavity have occasionally been successful.[2, 5, 6] Diversion of bile, cholecystectomy, and cholecystotomy have provided cures in seven cases.[3, 7, 14, 15, 18, 19, 30] Although neither the gallbladder nor the extrahepatic ducts were believed to be the source of bleeding, hemostasis has followed. Instances in which these procedures have eventually succeeded after repeated bouts of hemorrhage and the occasional remission which has occurred without operation [20] indicate that spontaneous resolution of the disease does occur and is not due to any special beneficial effects of the operation.

Reinhoff originally recommended ligation of the hepatic artery, and Sparkman suggested ligation of the appropriate extrahepatic branch as a means of last resort in the treatment of uncontrollable hemobilia. These measures have been employed in six cases.[6, 8, 23, 29, 31, 33] Though success has been reported following these procedures,[8, 31, 33] hepatic encephalopathy, continued hemorrhage and death,[6, 23, 29] have occurred after ligation of either the right or left branch of the hepatic artery.

It has been suggested that an anomalous accessory hepatic artery or collateral blood flow within the liver may permit back bleeding following extrahepatic arterial ligation of a single branch, and account for recurrent hemobilia. Similarly intrahepatic ligation would be required on two sides of an arterial defect to secure a predictable result.

The most consistently successful form of treatment has been resection of the portion of the liver in which the hemorrhagic injury exists. This has been achieved by hepatic lobectomy in five cases (right, 2;

TABLE 1. *Traumatic Hemobilia—Collective Review*

| Author | First Appearance of Hemobilia after Trauma | Operation | Comments |
|---|---|---|---|
| 1. Owen (1848) | Occurred 8 days after fall from carriage. Jaundice, colic, recurrent hemorrhages. Died 10th day. | None | First case report. Large central rupture. |
| 2. Hirzot (1908) | 3 months after blunt trauma.<br>5 months-<br>Hemobilia recurred, remitted, recovered. | Hepatic cavity aspirated and packed. | Long latent period. |
| 3. Siegel (1909) | 10th day after blunt trauma.<br>14th day-<br>28th day, recurred; remitted. Recovered. | Cholecystectomy. | Fibrin sealed laceration of dome; underlying central cavity. Cholecystitis mimicked by clot impacted in gallbladder. |
| 4. Strauss | 12th day after trauma, first of series of exsanguinating hemorrhages. | None | Central rupture, right lobe; large hemorrhagic cavity. |
| 5. Thorlakson (1929) | 8th day after injury. Several major hemorrhages preceeded death on the 17th day. | None | |
| 6. Wulsten (1931) | 3 weeks after blunt trauma. Four weeks of recurrent hemorrhages.<br>7 weeks, died. | None | Central rupture; large cavity within right lobe containing tissue, blood and bile. |
| 7. Hermansen & Cabitt | Liver laceration packed with gauze on days of injury. 27th day, hemobilia.<br>59th day-<br>Recurred<br>98th day-<br>Recurred, died 105th day after injury. | Gastrostomy, jejunostomy.<br>Transectomy of duodenum and posterior gastrojejunostomy. | Source of several upper gastro-intestinal hemorrhages not apparent at exploration.<br>Hemorrhage had ceased. |
| 8. Hawthorne (1941) | 9 weeks after blunt thoracoabdominal trauma.<br>17 weeks, hematemesis and melena.<br>20 weeks. Vomited branched clot.<br>Recovered. | Duodenotomy, Cholecystectomy. | At operation gallbladder and extrahepatic ducts distended by blood. After operation branched clot produced in emesis presumed to be biliary cast. First to recognize pathogenesis of hemobilia prior to necropsy. First cure. |
| 9. Ihrenias (1942) | 42nd day, hemobilia.<br>45th day-<br>Suspicion hemobilia recurred.<br>Recovered. | Cholecystectomy | Upper gastro-intestinal hemorrhages and clinical picture of cholecystitis<br>Gallbladder distended with blood. |
| 10. Westermeyer (1946) | Occurred 10 months after penetrating thoracic and blunt abdominal trauma. Enlargement of liver, colic, anemia developed.<br>12 months, melena.<br>Sepsis, hemorrhage recurred. Died. | Exploration of abdomen. Intrahepatic ligation of bleeding vessels. | Small occult gastro-intestinal hemorrahge led to anemia. |

TABLE 1—(*Continued*)

| Author | First Appearance of Hemobilia after Trauma | Operation | Comments |
|---|---|---|---|
| 11. Westermeyer (1946) | Blunt thoracoabdominal injury. Hemothorax. 40th day, biliary-like colic; 20 hours later, melena, hematemesis. Liver enlarged repeated hemorrhage. 52nd day— Recurred. 61st day– Exsanguinating hemorrhage. | Cholecystectomy. 40 cm. intestines resected, end-to-side anastomosis. | At operation emphymatic gallbladder distended by blood. At second operation, intestine distended with fresh blood, resected in belief it was hemorrahgic site. At necropsy, hematoma of liver. |
| 12. Ludwig (1947) | Melena, 6th day after trauma; hematemesis, 9th day. 17th day– Cured. | Cholecystectomy | At operation open fracture of liver with arrested hemorrhage. Gallbladder and bile ducts distended with blood. Bile in peritoneal cavity and retroperitoneal hematoma. |
| 13. Grey (1947) | Repeated gastro-intestinal hemorrhages 2nd week after injury. 6 weeks– Recurred. Died, 9th week. | Choledochostomy | Severe periodic hemorrhages, identified source of gastro-intestinal hemorrahge by distension of ducts with blood at operation. |
| 14. Sandblom | Hemorrhage began 10 days after blunt trauma. 10th day– Cure. | Cholecystectomy and drainage of central rupture of cavity. | At operation, central liver rupture cavity discovered. |
| 15. Burnet | 1st day, suture stellate laceration, dome of liver. 9th day, gastro-intestinal hemorrhage. 13th day– Hemobilia recurred. 55th day– 94th day– Recurrent hemorrhage 110th day– Recovered. Cure. | Exploratory laparotomy. Found no source or cause of gastro-intestinal hemorrhage. Re-exploration. Subphrenic clot removed. No cause for gastro-intestinal hemorrahge found. Gastrostomy, choledochostomy. Catheter drainage, packed central cavity with thrombin soaked gauze. | Biliary hemorrhage was suspected at 4th operation but site of hemorrhage not found. Successful operation when hemorrhage recurred. |
| 16. Hart (1950) | 8th day after accident liver laceration drained. Hemobilia, 8½ weeks later. 12 weeks after injury– Persistent hemobilia. 13 weeks– Recovered. Cure. | Exploration of abdomen. No source of gastro-intestinal hemorrhage found. Choledochostomy. | Though no biliary duct or gallbladder injury noted, hemobilia remitted following bile diversion. |

TABLE 1.—(*Continued*)

| Author | First Appearance of Hemobilia after Trauma | Operation | Comments |
|---|---|---|---|
| 17. Bigger (1950) | 1st day—Packed laceration of right lobe; continued hemorrhage required repacking. Recovered. 3 years and 3 months later, exsanguinating hemobilia. Died. | None | At necropsy, aneurysmal dilitation of a branch of right hepatic artery ruptured within a central cavity in right lobe. |
| 18. Epstein & Lipschultz | 1st day, laceration of liver sutured.<br>Occurred 2½ weeks later.<br>19th day–<br>Recovered. Cure. | Cholecystotomy.<br>Clot excavated from gallbladder. | Difficulty identifying source of hemobilia at operation. A normal appearing gallbladder without intrinsic injury or hemorrhage mitigated against cholecystectomy. |
| 19. Sparkman (1953) | 1st day. Sutured laceration of right lobe. Periodic hemobilia began during 2nd week.<br>6 weeks–<br>Recovered. Cure. | Cavity within right lobe packed. | At operation incised scar of previous operation revealing hemorrhagic central cavity beneath. Author presumed wound healing delayed by sequestrum. |
| 20. Baker, Hay (1955) | 1st day, laceration of right lobe packed with gelfoam and drained.<br>16 hours later hemobilia remitted.<br>Recurred on 27th day–<br>32nd day–<br>Cure. | Choledochostomy.<br>Sequestration of 30% right lobe. | Roentgen finding air fluid level in liver Encephalopathy and prolonged recovery. |
| 21. Broker (1955) | 1st day, laceration of right lobe packed with gauze, hemobilia 3 weeks later.<br>32nd day–<br>Massive hemobilia recurred.<br>7th week, exsanguinating hemorrhage, died. | Repacked, drained laceration. Colecystectomy | At necropsy, erosion of hepatic artery within substance of central rupture. |
| 22. Bailing (1956) | Laceration of liver sutured, 1st day.<br>2 weeks, hemobilia occurred<br>25th day–<br>Recurring hemorrhages.<br>6 weeks–<br>Recovered. | Exploration of abdomen.<br>Exploration of abdomen. | Spontaneous remission followed last operation. No source of hemorrhage found at 2nd abdominal exploration. Authors alert to possibility of traumatic hemobilia following 2nd operation, reintervention not required. |
| 23. Mikesky (1956) | 18 days after suture of laceration.<br>Recovered. | None | At emergency operation gallbladder and bile ducts distended with blood clots. Gastrointestinal hemorrhage, jaundice after treatment of gunshot wound of liver |

TABLE 1.—(*Continued*)

| Author | First Appearance of Hemobilia after Trauma | Operation | Comments |
|---|---|---|---|
| 24. Gombkötö (1957) | 10th day after trauma. Tampanade of bleeding space by suture of laceration.<br>8 months—hemobilia occurred.<br><br>Recurred; died. | Explorat on of abdomen, choledochostomy. | Sub capsular hematoma and hemorrhagic central cavity. Hemorrhaged through choledochostomy and into gastro-intestinal tract after its removal. |
| 25. Spector (1957) | 18th day.<br>Gastro-intestinal hemorrhage–<br>Hemorrhage recurred, 37th day.<br>Recurred.<br><br>73rd day–<br>Recurred.<br>101st day–<br><br>Recovered, cure. | Necrotic central cavity packed. Cystic central cavity repacked. Cholecystotomy.<br>Cyst repacked.<br><br>Right hepatic artery ligation. | Author cites complication of conversion of open wound into a closed rupture by packing.<br><br>Arterial ligation without serious sequellae. |
| 26. Thoromet (1957) | 1st day, laceration of liver packed.<br>Periodical hemobilia began 2 weeks later.<br>42nd day–<br>109th day–<br><br>Recovered. | Cholecystectomy.<br>Right hapetic lobectomy. | Resection of area of right lobe involved by injury procured cure. |
| 27. Manos (1958) | Hemobilia began 2 months after trauma.<br>8 months–<br><br>10 months, recurred.<br><br>Recovered, cure. | Abdominal exploration, choledochostomy.<br>Splenectomy.<br>Ligation of intrahepatic bleeding vessels. | At the last operation a scar over previous liver laceration was incised revealing central cavitary source of hemorrhage |
| 28. Goffi (1959) | Occurred on 10th day after trauma.<br>Remitted, recurred–<br><br>19th day–<br><br>Recurred.<br>85th day.–<br><br><br><br><br><br>Recovered, cure. | Exploration of abdomen.<br><br><br>Gastroduodenostomy<br><br><br>Duodenotomy, cholecystotomy, cholechostomy. Operative pancreatography, cholangiography, papillotomy. Ligation of left hapatic artery. | At initial operation, no apparent source of hemorrhage.<br><br>At 2nd, bleeding from ampula of Vater. Search revealed source within left lobe by cholangiography. |
| 29. Engler (1960) | Occurred 2 weeks after shotgun injury of right upper abdomen.<br>3rd week–<br><br>Recurred.<br>7th week–<br>Recurred<br>13th week–<br>Recovered. Cure. | Gallbladder drained of blood and clots.<br>Ligation of right branch of hepatic artery.<br>Hemorrhagic intrahepatic acvity | Recurrent hemobilia following location of appropriate extrahepatic arterial branch. At subsequent operation hemorrhagic cavity located and packed. |

TABLE 1.—(*Continued*)

| Author | First Appearance of Hemobilia after Trauma | Operation | Comments |
|---|---|---|---|
| 30. Markgraf (1960) | 2 weeks following gunshot wound, cholangiogram injection into biliary cutaneous fistula precipitated massive bleeding from fistula and into gastrointestinal tract; remitted. | | Cholangiogram revealed left hapetic duct obstruction. Diagnosis apparent prior to operation. Ascites, pleural effusion, melena following surgery. |
| | 4 weeks, recurred– | Cholecystectomy, drainage of intrahepatic cyst, extrahepatic ligation of heaptic artery. | |
| | Recurred, exsanguinating hemorrhage. Died two weeks after operation. | | At necropsy, fistula between common hepatic duct, right hepatic artery and portal vein. |
| 31. Schatzki (1961) | Occurred 14 days after trauma. | | At operation, central hemorrhagic cavity identified. Elevated serum alkaline phosphatase levels prior to recurrent hemorrhage. |
| | 26th day– Hemorrhages recurred. | Left hepatic artery ligation. | |
| | 92nd day– | Left hepatic lobectomy. | |
| | Recovered. Cure. | | |
| 32. Hutchenson (1961) | Occurred 6 weeks after suture of multiple lacerations of right lobe. | Cholecystotomy, Choledochostomy. | At operation, blood clot removed from gallbladder, common duct found distended with blood. |
| | Recovered. Cure. | | |
| 33. Saliba (9161) | Occurred 9 weeks after suture of liver lacerations produced by gunshot. | Gastrotomy, cholecystectomy, choledochostomy, and gauze packing of cavity. | Hemorrhage from central rupture apparently produced by wake of missel, followed suture of superficial lacerations. |
| | Hemorrhage recurred. 21 days later– | Intrahepatic angioplasty with arterial inflow occlusion. | |
| | Recovered. Cure. | | |
| 34. Guynn (1961) | Occurred 3 months after abdominal trauma. | | At necropsy, an intrahepatic aneurysm of right hepatic artery. At operation, impossible to ligate the artery obtain control through right hepatic duct. |
| | 4½ months– | Exploration, duodenotomy. Bleeding from ampulla of Vater noted. Cholecystectomy, choledochostomy. | |
| | 5 months– | Right hepatic bile duct divided and end oversewn. | |
| | Hemorrhagic shock, died. | | |
| 35. Souliotis (1963) | Occurred on 3rd day following suture of liver laceration. | | Diagnosis suspected prior to operation and cure obtained by extrahepatic ligation of appropriate branch of hepatic artery under hypothermia. |
| | 14th day– | Choledochostomy, drainage of post traumatic liver cyst and ligation of right hepatic artery. | |
| | Recovered. Cure. | | |
| 36. Authors (1963) | Occurred 29 days following suture of laceration. Hematemesis and copious melena, 6 days. | | Diagnosis made prior to operation and resection of hemorrhagic site produced cure. |
| | 35th day– | Left hepatic lobectomy. | |
| | Recovered. Cure. | | |

left, 3) and has provided a cure in each instance.[29, 35]

During an era of multiple blood transfusions and antibiotics, the over-all mortality rate has been reduced from 50 per cent to 36.1 per cent. Thirty cases of traumatic hemobilia have come to operation with an operative mortality rate of 26.8 per cent (Table 3).

## Summary

1. Traumatic hemobilia is an uncommon condition previously described primarily as a rare complication of central rupture of the liver. It has been found to occur as a late complication following emergency operation in which open injuries have been converted into closed wounds. The importance of drainage in the treatment of even minor lacerations of the liver which are produced by blunt trauma has been emphasized.

2. The etiologic circumstances which permit a sequestrum of devitalized hepatic parenchyma, blood and bile to erupt into a bile channel and to produce a vascular-biliary tract communication and hemobilia are obscure. A characteristic clinical course, however, in which biliary obstruction and colic precede and herald gastro-intestinal hemorrhage has become evident. It may be assumed that the bile duct becomes obstructed by blood clot and distention of ducts by blood occurs, producing pain. Pain persists as long as obstruction remains. When the clot is passed, pain dramatically remits and hemobilia becomes manifest as gastro-intestinal bleeding from an obscure source. A recurrent pattern of such pain is common. In the case reported, serum alkaline phosphatase levels were a useful index of progression of this process, and preoperative oral cholecystography was of particular diagnostic value. An unusual roentgen demonstration of blood within the gallbladder has been illustrated.

3. Whereas the diagnosis has rarely been made prior to operation, surgical experience has reflected this disadvantage and a lack of familiarity with the condition. Surgical treatment has been varied and frequently unsuccessful, and often has required reoperation for recurrent hemorrhage. Although reported experience with treatment of traumatic hemobilia is inadequate to warrant endorsement of any one

TABLE 2. *Treatment of Traumatic Hemobilia*

| Operative Procedures | No. | Cure | Recurrent Hemobilia | Spontaneous Recovery | Died |
|---|---|---|---|---|---|
| Cholecystectomy | 8 | 3 | 5 | 2 | 2 |
| Cholecystostomy | 7 | | | | |
| Choledochostomy | 9 | 1 | 12 | 2 | 2 |
| Catheter drainage of the hepatic cavity and/or packing of the cavity. | 6 | | 3 | | 1 |
| | 7 | 5 | 2 | | |
| Extrahepatic ligation of hepatic artery, | 1 | | 1 | | 1 |
| right branch– | 3 | 2 | 1 | | |
| left branch– | 2 | 1 | 1 | | |
| Intrahepatic ligation | 2 | 1 | 1 | | 1 |
| Intrahepatic angioplasty | 1 | 1 | | | |
| Right hepatic bile duct ligation | 1 | | 1 | | 1 |
| Hepatic lobectomy, right– | 2 | 2 | | | |
| Hepatic lobectomy, left– | 3 | 3 | | | |
| Miscellaneous | 17 | | | 1 | |
| No operation performed | 6 | | | 1 | 5 |

TABLE 3. *Traumatic Hemobilia*

| Cases | | Mortality % |
|---|---|---|
| Total | 36 | 36.3 |
| Operative | 30 | 26.8 |
| Non-operative | 6 | 83.5 |

procedure, it is probable that only operations which directly control the source of hemorrhage by angioplasty or ligation of the involved vessel on both sides of a defect, or those which resect the portion of the liver in which the hemorrhagic injury exists, will be consistently successful.

4. A pattern of improving surgical mortality figures has been noted which is attributed to factors other than the specific operative procedures.

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