A Study of the Postoperative Course After Hepatic Lobectomy

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RESECTION of an anatomic lobe of the liver has become standard operative treatment for extensive hepatic trauma and primary hepatic neoplasm. Although the indications and technic for hepatic lobectomy are well-established, the postoperative management remains a formidable challenge to both the patient and the surgeon. The postoperative course is frequently complicated by fever, infection, gastrointestinal hemorrhage, and malnutrition. Previous reports [1, 4, 12, 15, 16] have considered various aspects of the course and management of these patients. This report evaluates *in detail* the postoperative course of patients who have undergone hepatic lobectomy.

Clinical Material

The hospital records from January 1956 through June 1970 were reviewed for patients who underwent hepatic resection at the Vanderbilt University Medical Center. The study includes 31 patients who underwent complete or extended hepatic lobectomy (resection in excess of an anatomic lobe). Patients in whom less than complete hepatic lobectomy was done are excluded. Patients who died in the operating room or very shortly thereafter were also excluded. There were 19 males and 12 females. The age range varied from 8 months to 77 years. Nineteen had resections for trauma and 12 were performed for other indications, usually neoplasms. One patient in the trauma group underwent right hepatic lobectomy for rupture of primary hepatic carcinoma after minor trauma. Indications for the resections are shown in Table 1.

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Preoperative Status

The preoperative status of the nontrauma group generally showed no significant abnormality. The trauma group included seven patients in whom there was more than 12 hours delay from injury to treatment. Three of these patients had been explored elsewhere and transferred to Vanderbilt 2½, 9, and 25 days after injury because the initial suture of the hepatic laceration failed to control bleeding. Preoperative hemodialysis for acute renal failure was necessary in one of the three patients. Two other patients were transferred from local hospitals without prior operation, two and 10 days after injury.

Fifteen patients in the trauma group had associated injury of other organ systems. These included skeletal fractures in 12, pulmonary contusion in six, and pneumothorax in three. Associated abdominal injuries included a ruptured spleen in two, a ruptured adrenal gland in one, and renal injury in four.

Details of Operation

A thoracoabdominal incision was used in 25 patients and an abdominal incision in six. The duration of operation varied from 2 to 9 hours with most operations requiring from 4 to 7 hours. Initial, transient hypotension was recorded in nine patients and

TABLE 1. *Indications for Hepatic Resection*

Hepatic neoplasm		12
Primary carcinoma Liver	7	
Benign tumors	5	
Hepatic trauma		19

TABLE 2. *Extent of Hepatic Resection*

	Trauma	Neoplasm	Total
Right Lobectomy	16	3	19
Extended Right Lobectomy	2	2	4
Left lobectomy	1	6	7
Extended left lobectomy	0	1	1
Total	19	12	31

TABLE 3. *Major Post-operative Complications*

	Trauma Group	Neoplasm Group	Total
Infection	7	2	9
Pulmonary	6	2	8
GI tract	6	2	8
Hepatobiliary	1	6	7
Urinary tract	0	1	1
Sudden unexplained death	0	1	1
Complications in 16 patients			34

a prolonged period of hypotension in six patients. Cardiac arrest and successful resuscitation occurred in three patients. The amount of blood given in the pre- and perioperative periods ranged up to 22,500 cc. with an average of 3,800 cc. Injury to the inferior vena cava and/or the hepatic veins complicated the resection and led to massive blood loss in four patients. One patient had an embolus of hepatic tissue in the left lung. The hepatic lobe resected is shown in Table 2. Only one left hepatic lobectomy was performed for trauma, but there was a more even distribution of resected lobes in the non-trauma group. The technic of formal dissection of the hilar structures with ligature of the lobar ducts and vessels was used in 10 of 12 patients in the non-trauma group and in only 11 of 19 patients in the trauma group.

Biliary decompression via T-tube choledochostomy was used in 19 patients, catheter choledochostomy via the cystic duct in one patient, and Roux-en-Y choledochojejunostomy in a patient with common bile duct dilatation. Biliary decompression was not employed in 10 patients. This latter group was younger (average age 8 years). Operative cholangiography was used in five of the 31 patients. Drainage of the right upper abdominal quadrant was performed in all patients.

Postoperative Management

Nasogastric suction was maintained for an average of 4 days and oral intake was usually initiated on the fifth postoperative day. Adequate caloric intake and positive nitrogen balance was rarely obtained before the tenth postoperative day. Intravenous human serum albumin, vitamin K, and antibiotics were routinely administered in the postoperative period. Penicillin and Kantrex were the most commonly employed antibiotics. Twenty-one patients required postoperative blood transfusions, averaging 2,400 cc. per patient. Postoperative hospitalization averaged 30 days in surviving patients.

Liver Function Tests

Serial liver function tests were determined postoperatively in the majority of the patients. Evidence of abnormal liver

FIG. 1. Graphic representation of postoperative changes in serum albumin in 31 patients after hepatic lobectomy.

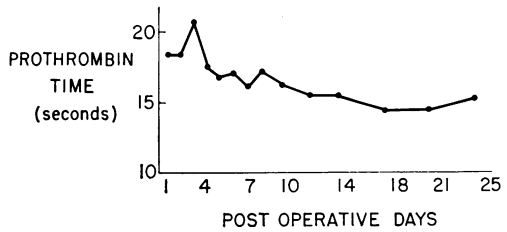

FIG. 2. Graphic representation of prothrombin time after hepatic lobectomy.

function was reflected in the level of serum albumin, prothrombin time, bilirubin, alkaline phosphatase, SGOT, and LDH. The majority of the patients received supplemental human serum albumin. The group as a whole fell to an average low of 2.7 Gm./100 ml. serum albumin on the eighth postoperative day (Fig. 1). The lowest value in a single patient was 0.7 Gm./100 ml. There was a progressive rise in serum albumin over the second and third postoperative weeks. The prothrombin time was prolonged to a maximum average of 20.8 sec. on the third postoperative day (Fig. 2). The maximum prolongation of prothrombin time was 29 sec. There were transient elevations of bilirubin, alkaline phosphatase, SGOT, and LDH in all patients (Fig. 3). There was a more marked and prolonged elevation of these values in those patients with the more complicated postoperative courses.

Postoperative Complications

Sixteen patients had one or more major postoperative complications (Table 3). Thirty-four major complications occurred in those 16 patients. Fifteen patients had no major complications, seven of whom had no complications at all. In addition to complications which can be specifically tabulated there was the perplexing problem of fever in excess of 38.8° C. in all but three patients (Table 4). These patients were carefully evaluated with all conceivable cultures and radiographic studies in an attempt to elucidate the etiology of the fever. Of 14 patients five had subphrenic abscesses; one patient was operated on for subphrenic abscess but none was found. Another patient had radiologic evidence but no clinical signs of subphrenic abscess and subsequently did well. Other documented infections were bacteremia proven by blood culture, pelvic abscess, empyema, wound infection, and pneumonia. In one patient a positive drain culture and radiographic evidence that the T-tube was not in the common bile duct appeared to explain the fever. The above infections were established clinically, proven by culture or both.

There were 14 patients with fevers of 38.8–39.4° in the first 5 postoperative days in whom no clinical explanation could be found. Two patients in the group died, death in one was sudden and unexplained. The other patient was hypotensive and febrile and may have had gram negative sepsis which was never proven. Neither patient underwent autopsy. The other 12 patients had uneventful recoveries. In these 12 patients the temperature returned to normal by the 7–10th postoperative day.

Five patients had massive upper gastrointestinal hemorrhages which required transfusion of whole blood. Three patients had stress ulceration of the stomach at autopsy. The fourth patient had aspiration pneumonia also, he died and there was no autopsy. The final patient survived; gastrointestinal x-rays failed to demonstrate a stress ulcer which was strongly suspected clinically. Five other patients had evidence of blood in the nasogastric aspirate or had hematemesis on at least one occasion but clinically they did not have significant upper gastrointestinal bleeding. One of the five patients who bled on the 15th postoperative day died of hepatic insufficiency. In the other four patients the bleeding occurred between the second and fifth postoperative days.

The other gastrointestinal tract complications included a colonic fistula which closed spontaneously and obstruction of the

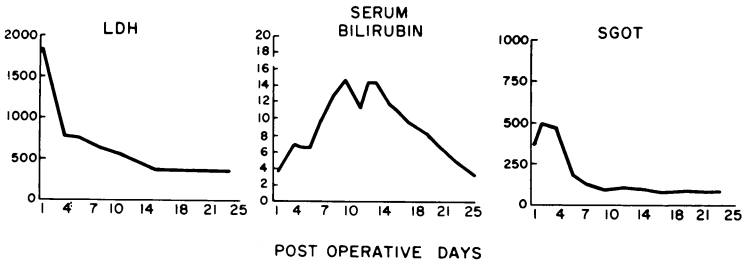

FIG. 3. Graphic representation of changes in serum levels of lactic dehydrogenase (LDH), bilirubin, and glutamic oxaloacetic transaminase (SGOT).

GI tract in two patients. One patient had small bowel obstruction and the other patient had gastric obstruction (Fig. 4). At reoperation the cause of antral obstruction was found to be the omentum which had been pulled across the anterior wall of the stomach to cover the cut surface of the liver. The obstruction was relieved and the patient subsequently did well. One patient had elevation of serum amylase postoperatively. This rise was felt to be secondary to the position of the T-tube and returned to normal subsequent to removal of the T-tube.

Pulmonary complications occurred more frequently in patients with thoracoabdominal incisions. Of eight patients with pneumonia, four died secondary to this complication. Tracheostomies were performed in four of these eight patients. Of much less consequence was the frequent appearance of right effusion, basal atelectasis or both, on thoracic x-rays. These were attributable to thoracoabdominal incision and proved to be of little consequence. One patient had pleural empyema which responded to drainage and antibiotic agents. Infection, pulmonary and GI tract complications were more common in the trauma group while those related to the biliary tract, liver and portal system were more common in the elective, non-trauma group. Two patients had leakage of bile into the peritoneal cavity secondary to dislodgment of the T-tube from the common bile duct. Another patient in whom a T-tube was not used developed progressive jaundice and underwent re-operation on the 21st postoperative day. The distal common duct was compressed by a mass of inflammatory lymph nodes. Choledochojejunostomy was performed and the patient is alive and well 4 years later. Massive ascites developed in two patients who had extensive resections for hepatic carcinomas. Both patients died. At autopsy in one thrombosis of the portal vein was found. The death in the other patients was attributed to hepatic insufficiency.

In 21 of 31 patients the biliary tract was decompressed with a T-tube or catheter. The average age in these 21 patients was 32 years whereas in the other 10 patients the average age was 8 years (Table 5). Major complications were more frequent in the group with decompression. Average hospital stay was shorter in the group without decompression. There were no deaths in the group without decompression. All of the gastric stress ulcerations occurred in the group with biliary tract decompression.

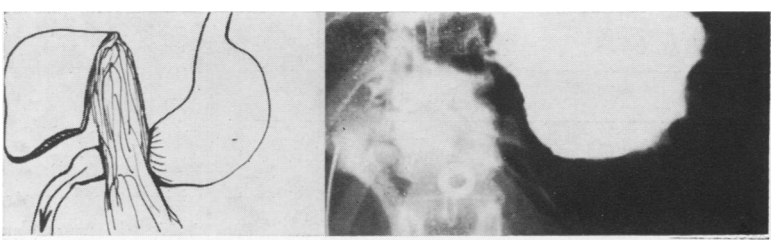

FIG. 4. Thirty-five-year-old woman developed gastric outlet obstruction (x-ray) 4 weeks after extended left hepatic lobectomy. Omentum used to cover the raw liver surface was obstructing the antrum (sketch). Division of omentum and mobilization of stomach relieved the obstruction.

TABLE 4. *Postoperative Fever*

	No. Patients
No significant post-operative fever	3
No clinical explanation for postoperative fever	14
Clinical explanation for postoperative fever	14

TABLE 5. *Comparison of Patients With and Without Biliary Decompression*

	Biliary Decompression	No Biliary Decompression
Number of pts.	21	10
Average age	32 yr.	8 yr.
Trauma group	16	3
Neoplasm group	5	7
No. of major p.o. complications	13	3
Subphrenic abscess	3	2
Post-op. GI hemorrhage	10	0
Average days hospitalization	33.3	24.5
Death	7	0

TABLE 6. *Causes of Death*

Trauma Group

1. Stress ulcers, bilateral pneumonia, renal artery thrombosis
2. Gram negative sepsis
3. Stress ulcers
4. Massive upper GI hemorrhage (? stress ulcers) and aspiration pneumonia

Neoplasm Group

5. Stress ulcers, renal failure, portal vein thrombosis
6. Hepatic insufficiency
7. Azotemia, sudden unexplained death

TABLE 7. *Delay from Injury to Operation*

	Patients	Mortality
Delay of 12 hours or more	7	43%
Initial suture of lacerations requiring reoperation and resection	4	50%

Renal complications included urinary tract infection in three patients, acute renal failure and death in one patient and the last patient had azotemia (BUN 102 mg. /100 ml.) during an otherwise uncomplicated course and died suddenly. There was no apparent cause for the death and autopsy was not done.

Deaths

There were seven deaths, an overall mortality rate of 23% (Table 6). There were three deaths (25%) in the non-trauma group and four deaths (21%) in the trauma group. In the 21 patients less than 30 years of age, there were two deaths (9.5%). In the 10 patients over 30 years of age there were five deaths (50%). There were seven patients in whom there was a delay of 12 hours or more from the time of injury to definitive treatment (Table 7). There was no delay in only one of the four deaths in the trauma group. Two of the four patients who died had undergone previous suture for hepatic lacerations. Recurrent hemorrhage led to referral, reoperation and hepatic resection in those two patients.

Discussion

The postoperative course after hepatic lobectomy may be smooth and uncomplicated. However, many patients have very complex clinical courses with concomitant major complications. The preoperative condition is usually stable in patients undergoing elective resection although resections for hepatic trauma are usually performed under precarious circumstances with massive blood loss and associated severe injuries. Preoperative evaluation of trauma patients with various diagnostic methods is often possible and occasionally indicated; however, unnecessary delay in operative control and management of severe hepatic injury may jeopardize the chances of survival (Table 7). The patient who is significantly hypotensive with suspected hepatic injury should undergo exploratory

surgery promptly with volume replacement instituted enroute to the operating room.

Only a small percentage of hepatic injuries require resection.[19, 21, 30, 35] A very conservative approach to the use of lobectomy or hepatic trauma is advocated by some. Our experience suggests that lobectomy in extensive hepatic injury is preferable to awaiting complications such as hemobilia, re-bleeding or infection.[19, 35] Half of the deaths in the trauma group might have been avoided if initial hepatic resection had been done instead of treating extensive lacerations by means of suturing and packing with gel foam.

The technic for hepatic lobectomy has been clearly detailed [2, 30] and should be carefully followed in all elective instances and in resections for trauma in which bleeding can be temporarily controlled. The use of the Pringle [29] maneuver (digital occlusion of hilar hepatic vessels) and temporary packing of the hepatic wound may be helpful in this respect. In some instances formal hilar dissection may not be possible or wise; bleeding from the liver may be so profuse that it is necessary to transect the liver and ligate individual vessels as they are encountered. This was done in eight of the 19 resections for trauma in this report. An extremely difficult and often lethal injury involves the hepatic veins and/or the inferior vena cava.[3, 27, 31] This injury causes massive blood loss which is difficult to control, and increases the potential for embolism of air [6, 27] or hepatic tissue.[33] Various technics to control the massive bleeding associated with this injury have been suggested.[3, 6, 31]

The immediate operative concern associated with hepatic lobectomy is hemostasis. Control of bile leakage from the cut surface of the liver has received less attention. The propensity for bile to lyse clots, its chemical irritant effects and the incidence of infection secondary to bile leakage can create a dreaded complication. The elementary principles of controlling bile leakage are (1) adequate resection and debridement of injured and devitalized hepatic tissue, (2) meticulous control of all ducts which are transected and, (3) adequate external drainage. Retrograde injection of methylene blue into the biliary system may help to identify transected ducts. A cholangiogram should be routinely performed, primarily to evaluate the integrity of the remaining biliary tree and also to demonstrate any leakage of bile from the hepatic surface. Merendino [24] has suggested the use of biliary decompression, via T-tube drainage of the common duct, to lower pressure within the bile ducts and thereby minimize leakage. The logic behind this proposal seems sound and it has been widely used, even by some in relatively minor hepatic injuries. Convincing proof of the value of biliary decompression in this situation is lacking. In this series the overall mortality and morbidity was greater in the group with T-tube drainage. However, these patients (Table 5) were appreciably older than those in whom decompression was not employed. Recent reports have questioned the wisdom of the use of biliary decompression. Lucas [18] could demonstrate no reduction in intra-hepatic bile duct pressure with cholecystostomy or choledochostomy in the dog. In a prospective study of biliary decompression in hepatic injuries of all degrees, Lucas and Walt [19] reported a lower percentage of complication in those patients in whom biliary decompression was not used. This was consistent with their findings in a retrospective study of an earlier group of hepatic injuries.[18, 19] The hazard of choledochostomy is greater in infants and children and has resulted in the omission of biliary decompression by many who do hepatic resection in children.[11, 25, 34] Clatworthy [10] advocates cholecystostomy as a means of biliary decompression and as a site for injection of methylene blue to search for open ducts in the raw liver surface. Dow [13] has used catheter drainage of the stump of the transected lobar duct in

infants undergoing hepatic lobectomy. Experience in this study would indicate that biliary decompression could safely be omitted in lobectomy in childhood. The role that bile diversion may play in the alarming incidence of stress ulceration and upper GI bleeding is a factor which may temper the widely accepted use of this procedure.

Hepatic insufficiency following lobectomy does not occur provided the remaining liver is normal and the vessels and ducts of the remaining lobe are intact. One of the patients in the series developed ascites with progressive deterioration of liver function and clinical status. Thrombosis of the portal vein secondary to tumor invasion was found at autopsy. While the threat of hepatic insufficiency is of little concern there are consistent changes in the biochemical parameters of hepatic function following lobectomy.[4, 7, 8, 9, 16, 17, 22, 26, 36] The two most significant changes are decrease of the blood sugar and serum albumin. The hypoglycemia is almost never documented because of the constant infusion of glucose solutions.[9, 22] The hypoproteinemia, manifested primarily by hypoalbuminemia, should be treated vigorously by the administration of serum albumin.[7, 22] The predictable fall in serum albumin, which may show little response to intravenous administration of albumin, is further compounded by the anorexia and resultant limited intake noted when oral nutrition is first begun. Biliary diversion has a marked anorectic effect on the patient which leads to a prolonged period of catabolism and negative nitrogen balance. We believe that the currently available total parenteral alimentation preparations (TPA) should be administered in the immediate postoperative period and continued until the patient can maintain adequate oral nutrition. It is our opinion that the use of TPA will substantially decrease morbidity and perhaps mortality following major hepatic resection. Hepatic dysfunction is further manifested by elevation of SGOT, LDH, SGPT, alkaline phosphatase, bilirubin, cephalin flocculation, thymol turbidity, serum ornithine carbamoyl transferase and a decrease in serum cholesterol, triglycerides, nonesterified fatty acids and cholesterol esters.[22] The reason for these changes is related to a combination of factors including net loss of liver mass and hepatic congestion secondary to the entire hepatic inflow traversing one lobe of the liver. These changes are transient and gradually return to normal over a variable period of time.

Following hepatic lobectomy measurable decreases in fibrinogen, prothrombin, factor V, factor VII, factor IX, factor X and plasminogen have been reported. In this series prothrombin time, the only parameter routinely measured, was invariably prolonged following hepatic lobectomy. Whether the vitamin K which was routinely administered had a beneficial effect is uncertain. The prothrombin time returned to normal levels in 7–10 days. Postoperative hemorrhagic diathesis was not encountered in this series of hepatic resections. While bleeding diathesis has been uncommon after hepatic resection it has been noted in a few cases,[19, 21] and when it occurs its etiology will most likely be a problem with fibrinolysis or consumptive coagulopathy rather than a deficiency in coagulation factors. The use of epsilon aminocaproic acid may be of benefit in the management of fibrinolysin but may also introduce the problem of intravascular coagulation.

Temperature elevation above 38.3–38.8° C. following hepatic resection is very common during the first postoperative week and presents a genuine clinical challenge. In this study lower lobe atelectasis with pleural effusion following hepatic resection through a thoracoabdominal incision was sometimes the source of fever but could not explain the majority of the fever problems. Obvious infections such as pneumonia, empyema, wound infection, urinary tract infection and sepsis with positive

blood culture allow specific therapeutic efforts. Subphrenic abscess and other intraperitoneal infection are often more difficult to recognize. Overt infections have been a source of difficulty in the postoperative period following hepatic resection in most reports in the literature.[1, 12, 21, 27, 35] This study revealed a group of patients with perplexing temperature curves but with no detectable source for the fever. There was concern that these patients might have any of the many infectious complications which can occur following hepatic resection. The temperature elevation occurred in the first 3 to 5 days postoperatively. The early onset of high fever with recurring elevations abve 38.8° was in sharp contrast to that group in whom the temperature elevation was delayed until after 5 days and corresponded to the appearance of an overt clinical infection. The etiology or origin of this fever following hepatic lobectomy usually falls into one of three categories: pulmonary complications, perihepatic infection and fever of unknown origin. We suspect that the latter group represent the response to necrosis of devitalized hepatic tissue in suture ligatures on the cut surface of the liver. Re-exploration of these latter patients has little to offer provided adequate drainage has been initiated at the primary operation.

In an earlier report [15] we reported a very significant incidence of stress ulcer in a large group of patients undergoing major hepatic resection. Prior to that report there had been little mention of this problem after hepatic resection. Since that time other reports [12, 19, 21, 27, 28, 35] indicate that stress ulcerations should be a major concern following hepatic lobectomy. There were five patients in the present series who had either proven stress ulcers or massive upper GI bleeding with a mortality rate of 80%. Another five patients who showed some signs of bleeding from the upper GI tract did not require transfusions. All five of the patients with massive bleeding had biliary decompression. The association of stress ulcer and biliary decompression was also noted by Lucas.[19] Prophylactic measures such as strict avoidance of ulcerogenic drugs and early instillation of antacids in the stomach are strongly recommended. While the specific etiology of stress ulceration remains obscure, there are certain features of the postoperative status of a patient following hepatic lobectomy which lead to speculation regarding etiology. The biochemical evidence of altered hepatic functions raised the possibility of a "physiologic shunting" of a gastric secretagogue normally present in portal venous blood. Unpublished experimental canine studies from our laboratory have failed to demonstrate any increase in gastric acid secretions after 60% hepatic resection. There was instead a fall in gastric acid secretion. Earlier experimental work by Dragstedt [32] and Menguy [23] clearly demonstrated hypersecretion following exclusion of bile and pancreatic juice from the small intestine. This operation causes a high incidence of gastric ulcerations in the dog as originally described by Exalto [14] and later by Mann and Williamson.[20] While T-tube choledochostomy is unlikely to completely exclude all bile and pancreatic juice, the high incidence of "stress ulcer" in these patients certainly raises the question of whether biliary decompression is responsible. The patients in this report all had associated injuries or other major complications both of which made their course difficult to interpret. Nonetheless the possibility remains that biliary diversion plays a distinct role in the genesis of stress ulceration and raises doubt regarding the general acceptance of biliary decompression as an essential step in the management of major hepatic injuries and resections.

Attention to adequate ventilation and pulmonary toilet is essential in the postoperative care of these patients. Pulmonary complications are common as others have noted.[5, 21, 35] Many patients had associated

pulmonary contusions as did six in this series; the frequent use of thoracoabdominal incision further complicates pulmonary function. Walt [35] has noted progressive pulmonary deterioration associated with hypoxia secondary to "physiological shunts." Such changes should be anticipated in this group of patients who are frequently subjected to pulmonary contusion, shock, massive transfusions, embolism of air, thrombi, fat and liver tissue, and massive intra-operative administration of fluids. Ventilatory assistance and tracheostomy may be necessary to manage these patients. The prophylactic approach to pulmonary complications is essential. There were eight patients with either pneumonia or aspiration pneumonitis, four of whom died. The use of bronchoscopy and tracheostomy may be life saving in such patients.

The mortality rate of patients treated surgically for hepatic injuries usually has a direct relation to the number of associated injuries of other organ systems.[5, 19, 21, 35] The mortality rate in our patients was more closely related to the presence or absence of postoperative complications. There are several factors which are prominent, though possibly not significant, as regards mortality in this group of patients. All had biliary decompression. Stress ulcer or massive bleeding occurred in four of the seven patients who died. Pneumonia or aspiration pneumonitis was present in four of the seven. In the trauma group the number of associated injuries was not significantly higher in those who died than in the survivors. There were four deaths in the trauma group. In three of these four patients there had been a delay of 12 hours or more from injury to the time of definitive operation. In two of these latter three deaths there had been initial suture of the laceration with subsequent bleeding requiring re-operation and resection. It is felt that the mortality can be lowered if deep and extensive hepatic injuries are treated with hepatic resection rather than attempting to control bleeding by suture of the lacerations. Stress ulceration and infection, especially subphrenic abscess and pulmonary infections, were major problems in our patients who died.

This study of the postoperative course after hepatic lobectomy leads us to make the following recommendations: (1) early operation, (2) resection for extensive lacerations, (3) hilar dissection when time permits, (4) meticulous control of bile leakage by suture, (5) adequate drainage by sump tube and posterior dependent drains, (6) total parenteral hyperalimentation with the addition of vitamin K, (7) antacids and avoidance of ulcerogenic drugs, and (8) avoidance of *routine* biliary tract decompression.

Summary

A review of the postoperative course after hepatic lobectomy has been presented. Although emphasis has been placed on complications there were 15 patients (49%) who had no major complications. The overall mortality rate was 23%. Those patients with complicated postoperative courses were analyzed for contributing factors in the preoperative status, operative procedures and postoperative management. The advisability of biliary decompression has been discussed. Postoperative problems of unexplainable fever and stress ulcers have been reviewed.

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Discussion

Dr. Julian K. Quattlebaum (Savannah): Dr. Sawyers kindly permitted me to read his manuscript before the meeting, and I wish to congratulate him on a most comprehensive and detailed study of the postoperative course of a most impressive series of hepatic resections.

I think, in general they illustrate what might be expected following operation of this magnitude on patients who, for one reason or another, are far from acceptable risks.

Over the years we have removed a significant portion of the liver, either as a primary procedure or as part of an even more extensive operation, on 18 patients. None of these patients developed in their postoperative course any type of postoperative bleeding, such as Dr. Sawyers', and none developed intestinal obstruction. Perhaps if some of them had lived a little longer they would have developed these complications. The common bile duct was routinely opened as the first step in the dissection of the hilus in all major resections in order to locate and protect the hepatic ducts, the duct being closed over a "T" tube.

Our troubles usually have occurred promptly in the immediate postoperative course. They have