MEDICAL PRACTICE

Hospital Topics

Injuries to the Liver: Analysis of 20 Cases

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Summary

Twenty cases of liver injury among 55 consecutive cases of abdominal injury submitted to laparotomy over a four-year period are reported. Forty-four of the cases were blunt injuries, and the cases of liver injury were in this group. Road traffic accidents accounted for 37 of the 44 cases and 17 of the 20 liver injuries. Except in two cases injury to the liver was associated with injury to other organs. Severe chest injury was found in 40% of the cases and serious skeletal injury in 45%. The overall mortality in blunt injury to the liver was 20% (4 cases) and was directly attributable to the liver injury in only one case.

Liver injuries are classified as minor or major according to the depth of the wound and the associated destruction of liver tissue. Liver resection is advocated for major injuries. Right hepatic lobectomy was performed on five occasions and three of the patients survived. Death in the other two was due to associated injuries. The remarkable regenerative capacity of the liver is emphasized.

Introduction

Road accidents account for most cases of abdominal injury in civilian practice, and the incidence of blunt injury to the liver has risen with the increase in high-speed traffic.[1] At least 60% of the victims of fatal road traffic accidents have serious liver injury.[2 3] Non-penetrating injury may extensively disrupt the liver substance and damage vascular and biliary pedicles far beyond the area of laceration.[4] Control of haemorrhage, bile leakage, difficulty in assessing the extent of the damage, and often complex postoperative problems present formidable difficulties. The decline in mortality from blunt injury to the liver from 80%[5 6] to 30% or less[1 2 7] has been attributed to better techniques of resuscitation, adequate blood transfusion, a more aggressive surgical approach, and better postoperative management.[8] We present an analysis of 20 consecutive cases of blunt injury to the liver operated on by one of us (L.H.B.) over the past four years. The indications for major hepatic resection are examined, and we suggest that resection should be resorted to more readily if haemostasis cannot be otherwise assured.

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Clinical Presentation of Abdominal Injury

From December 1966 to December 1970 55 cases of injury to the abdomen came to laparotomy. None of the penetrating wounds involved the liver. Forty-four patients had suspected blunt abdominal injuries and 35 of them were either the drivers or the passengers of a motor vehicle. Domestic, sporting, and industrial accidents accounted for only seven injuries. One man with multiple injuries, including fractured ribs and pelvis, proved to have no internal injury. The other 43 had injury to the abdominal viscera, and 20 of them had liver injuries (Table I). The frequency of injury to various organs is detailed

TABLE I—*Cause of Blunt Abdominal Injury in the 44 Cases*

	No. of Cases	No. with Liver Injury
Traffic accident	37*	17
Domestic „	4	1
Sports „	2	1
Industrial „	1	1
Total	44	20

* Thirty-six proved intra-abdominal injuries, one negative laparotomy.

TABLE II—*Distribution of Injury in 43 Cases of Proved Blunt Abdominal Injury*

	Frequency		Frequency
Liver	20	Urethra	3
Spleen	16	Gall bladder	2
Diaphragm	6	Bladder	2
Mesentery	5	Colon	2
Jejunum	3	Pancreas	1
Kidney	3	Duodenum	1

Details of chest, head, and skeletal injuries not included.

TABLE III—*Sites of Associated Injury in the 20 Cases of Liver Injury*

	Frequency		Frequency
Spleen	4	Pancreas	1
Mesentery	2	Diaphragm	1
Gall bladder	2	Chest/lung	8
Duodenum	1	Head	10
Colon	1	Skeleton	9

in Table II. Abdominal injuries were frequently associated with injury to the head (23 patients), chest (14 patients), or skeletal system (19 patients).

Diagnosis

There is probably no other field in which there is a greater justification for laparotomy on suspicion than that of abdominal injury. The mortality and morbidity are slight and must be more than offset by the serious mistake of a failed diagnosis.[9] This principle guided us in our series.

The decision whether to explore the abdomen should be made as soon as possible. The mortality of injury is increased with delay in treatment,[10] and there may be further urgency when anaesthesia is needed in the treatment of an extra-abdominal injury. We found the most reliable physical signs of intra-abdominal injury to be abdominal pain and tenderness accompanied by a rising pulse rate. Shoulder-tip pain was a good index of subdiaphragmatic irritation. The general improvement that occurs in patients with fractures of the pelvis treated with adequate blood transfusion and pelvic immobilization[11] was most useful in deciding whether they had an appreciable intra-abdominal injury. If the pulse rate continued to rise in patients so treated laparotomy was proceeded with unless the tachycardia could be accounted for by haemorrhage into the chest.

Radiology was of limited value except in cases of rupture of the diaphragm. Abdominal paracentesis was abandoned when we found a ruptured spleen at laparotomy after a negative four-quadrant tap. A positive tap is useful but a negative one means nothing.[1] Angiography or scanning techniques were not used in diagnosis.

One of the patients with extensive liver damage had a slow pulse rate for many hours after injury. This phenomenon in the presence of severe liver injury has been previously recorded,[12] and we agree with Mathieson[9] in emphasizing that a slow pulse rate should not allow a false sense of security.

Liver Injuries

The youngest of the 20 patients with liver injury was aged 8 and the oldest 43. Most were between the ages of 11 and 30. The male to female ratio was 14:6. Seventeen of the injuries were due to road accidents and three to domestic, industrial, or sporting incidents (Table I). These figures reflect the national trend of road traffic accidents.[13]

In only two cases was the liver the only organ injured. In the remaining 18 there was serious damage to at least one other organ (Tables III and IV). Though liver injuries are apt to be accompanied by other injuries in the upper abdomen

TABLE IV—*Frequency of Associated Injury in the 20 Cases with Liver Injury*

	Liver alone	+1 Organ	+2 Organs	+3 Organs	+4 Organs	+5 Organs
No. of cases	2	8	3	3	3	1

TABLE V—*Site or Nature of Chest Injury found in association with Liver Injury in 8 Cases*

	Frequency		Frequency
Flail chest	5	Azygos vein	1
Multiple rib fractures	3	Aorta	2
Lungs*	7	Heart	1

* Pneumohaemothorax.

Seven patients had tracheostomy and positive-pressure ventilation.

few were associated with rupture of the spleen, but associated injuries to the chest, head, or skeleton were found in many cases. Five of the eight patients with chest injury had a flail chest with paradoxical respiration, and three others had multiple rib fractures, underlying damage to lung tissue, and haemopneumothorax. There were two cases of severe damage to the heart or aorta (Table V). Seven patients required tracheostomy and positive-pressure ventilation either to control paradoxical respiration or for postoperative respiratory difficulty. Nine patients (45%) had serious skeletal injury involving either the long bones or the pelvis or both. One patient sustained a traumatic amputation of his left leg. Head injuries were often associated with facial lacerations and occasionally with facial fractures, but cerebral injury was limited to concussion and there were no cases of severe cerebral damage.

MINOR INJURIES

We have classified as minor those cases in which the laceration of the liver substance could be closed to its full depth by simple suture with complete control of haemorrhage and in which there was little or no associated parenchymal destruction. Fourteen of the 20 cases fell into this group, and in all of them the injury was in the right lobe or in the region of the ligamentum teres. The amount of intraperitoneal haemorrhage varied widely, and sometimes extensive haemoperitoneum accompanied a relatively minor laceration. In several cases, however, the haemorrhage was small and bleeding seemed to have stopped spontaneously.

Cases of minor lacerations were sutured with the minimum number of thick chromic catgut sutures, taking deep bites of the liver substance and using large needles. When the tears were near the ligamentum teres this structure was mobilized and folded within the laceration at the time of suture. Drainage was not carried out for this form of liver injury, as suggested by Little,[3] though an intra-abdominal drain was sometimes used for associated abdominal injury. The haemorrhage was controlled in every case. Severe associated injuries were found in several cases and accounted for the death of one patient.

MAJOR INJURIES

We have defined as major any liver laceration so extensive that the wound could not be closed by simple suture so as to ensure haemostasis and not leave "dead space" in the depths of the wound. Finger exploration may show internal disruption of the liver parenchyma[3 4] and there may also be more minor radiating tears.

The initial treatment in the first two cases of major injury was not by liver resection. One patient died of haemorrhage after his liver injury had been packed and the other subsequently required hemihepatectomy for recurrent bleeding.

The first patient, a 16-year-old boy, was deeply shocked and had signs of intraperitoneal bleeding on admission after a road accident. Laparotomy showed a 6-in (15 cm) tear of the right lobe of the liver which was bleeding profusely. Finger exploration confirmed that there was internal disruption of the liver substance. The wound was 4-5 in (10-12·5 cm) deep at its deepest point. As suture failed to control the haemorrhage the wound was firmly packed with gauze. The tail of the pack was brought out through an incision in the right hypochondrium, the subhepatic space was drained, and the abdomen closed. Though some oozing continued bleeding appeared reasonably controlled for some three to four hours, but it began again and continued for 24 hours, when the patient died after a cardiac arrest.

The second patient, a man aged 21, was admitted after a road accident with multiple fractured ribs, bilateral haemopneumothorax, a flail chest, and signs of intraperitoneal haemorrhage. At laparotomy a deep laceration 2 in (5 cm) long was found on the inferior surface of the right lobe of the liver. Digital exploration showed much internal disruption of the liver tissue. Several sutures, taking as deep a bite of liver substance as possible, seemed to secure haemostasis. Chest drains were inserted, a tracheostomy was carried out, and the patient returned to the ward on a positive-pressure ventilation. Ten hours later a further laparotomy was performed because of recurrent intra-abdominal haemorrhage. The liver capsule was found to be raised over the right lobe by a huge haemorrhage which had torn through the capsule at several places. A formal right hemihepatectomy was carried out. The patient died 10 days later from his chest injuries. At necropsy it was shown that no further bleeding had occurred from the liver and that a considerable degree of regeneration had taken place. Indeed, the liver weighed 1,700 g and was of good consistency. This case has been reported in detail elsewhere[14] and the essential features are summarized in Table VI (Case 1).

Experience in these two cases convinced us of the need to adopt a more aggressive surgical approach, and formal resection of the liver was performed as a primary procedure in the four subsequent cases of similar extensive injury.

Major Injuries Treated by Hemihepatectomy

The salient clinical features of the five cases treated by right hemihepatectomy are summarized in Table VI. All had severe damage to the right lobe of the liver with extensive disruption of the parenchyma. All but one were shocked on admission with obvious signs of intra-abdominal haemorrhage. In one patient (Case 4), though intraperitoneal bleeding was suspected clear signs of it were not manifest until 12 hours after injury. An initially slow pulse rate began to rise and increasing abdominal tenderness and shoulder-tip pain made laparotomy mandatory. Four of the five cases had associated injuries and needed 12 pints (6·8 l.) or more of transfused blood. In all cases haemostasis was complete.

Two of the patients died from their associated injuries. One died of pneumonia and empyema associated with severe chest injury (Case 1), and the other (Case 5) developed a haemorrhagic diathesis and died after an uncontrolled haemothorax originating from a small tear of the azygos vein in communication with the right pleural cavity.

OPERATIVE TECHNIQUE

Our operative technique was essentially that described by Lloyd-Davies and Angell[15] and by Quattlebaum and Quattlebaum.[16]

The abdomen is opened through a right paramedian incision, the liver wound explored with the fingers, and the damage assessed. If resection is decided on the incision is extended into the right thorax, using the seventh or eighth interspace and splitting the diaphragm down to the inferior vena cava. Haemorrhage from the liver wound is temporarily controlled by packing, and in addition the structures in the free edge of the gastrohepatic omentum may be occluded for periods of up to 20 minutes. The cystic duct and artery are then identified, ligated, and divided. A small fringe of peritoneum is raised from the structures at the porta hepatis and the structures are identified. Dissection is carried out as close to the liver substance as possible. The right branch of the hepatic artery, right hepatic duct, and right branch of the portal vein are identified serially, ligated, and divided between ligatures. The right lobe of the liver is then mobilized from the diaphragm and the right hepatic vein identified. It is large and short and its ligation and division may be difficult. In only one case (Case 5) was the right hepatic vein entirely intrahepatic, and it had to be controlled from within the liver substance at the time of resection. Further small veins draining from the right lobe of the liver into the vena cava are ligated, working from the inferior surface of the liver upwards, but in two cases where we had difficulty in doing this we secured the hepatic veins from within.

Examination of the liver at this stage shows a line of demarcation between the ischaemic and normal liver. We have found it to run medial to the gall bladder fossa. The liver substance is divided by diathermy about 0·5 cm lateral to the line. Any obvious bleeding

TABLE VI—*Summary of Cases treated by Right Hemihepatectomy*

Case No.	Age and Sex	Condition on Admission	Associated Injuries	Nature of Liver Wound	Postoperative Complications	Outcome	Remarks
1	21 M.	Concussed. Shocked. Obvious peritoneal irritation	Flail chest. Bilateral pneumohaemothorax	2-in (5-cm) tear visceral surface R. lobe with extensive deep destruction of liver tissue. Simple suture failed to arrest bleeding. R. hemi hepatectomy 10 hours after first laparotomy	Respiratory difficulty. Tracheostomy. P.P.R. Haemorrhagic diathesis. Pneumonia. Empyema	Died 10 days after operation, of pneumonia	18 units blood. Necropsy liver weight 1,700 g
2	22 M.	Concussed. Shocked. Obvious peritoneal irritation	Comminuted fracture of right olecranon	Extensive tear visceral surface R. lobe near porta hepatis extending deep into liver substance. Profuse bleeding	Respiratory difficulty. P.P.R. Haemorrhagic diathesis diagnosed preoperatively. Subphrenic abcess. Pneumonia	Survived	20 units blood
3	9 F.	Unconscious. Shocked. Obvious peritoneal irritation	Ruptured spleen (Splenectomy)	5-in (13-cm) tear diaphragmatic surface R. lobe through liver substance appearing as stellate tear near quadrate lobe. Bleeding	Transient hypoglycaemia. Haemorrhagic diathesis. Biliary leakage. Pneumonia. Pleural effusion	Survived	12 units blood
4	13 M.	Fully conscious. Initial pulse rate slow. Signs of intraperitoneal blood loss became obvious only 12 hours after injury	Nil	Stellate tear of diaphragmatic surface R. lobe almost through whole liver substance	Transient respiratory difficulty	Survived	3 units blood
5	23 M.	Unconscious. Shocked. Obvious peritoneal irritation	Multiple rib fractures. Disruption of pelvic ring. Dislocation R. sacroiliac joint and sacrococcygeal joint. Rupture R. hemidiaphragm. Tear second part duodenum. Compression necrosis ascending colon. R. hemicolectomy	Extensive stellate tear diaphragmatic surface R. lobe—through-and-through type	Haemorrhagic diathesis. Right haemothorax	Died 10 hours after operation following cardiac arrest	22 units blood. Necropsy: small rent azygos vein; liver haemostasis complete

points or biliary radicles leaking bile are individually transfixed and ligated with catgut sutures. A major branch of the left hepatic vein which is always encountered is similarly transfixed and secured. If the right hepatic vein has not been secured beforehand the line of resection is swung a little to the right posteriorly and these veins are secured from within. The edges of the raw surface are then transfixed with large catgut sutures and lightly compressed, care being taken not to produce unnecessary ischaemia of the liver substance. The common bile duct is decompressed with a T-tube, the wounds are closed, and a chest drain is left in situ. The space left by the right lobe of the liver is similarly drained with a large tube drain.

POSTOPERATIVE MANAGEMENT

After resection all patients were given ampicillin 250 mg intramuscularly six-hourly. Our reasons for this were twofold—firstly, most of the patients had associated soft tissue injury and, in addition to wound toilet, it is our practice to treat such cases with tetanus toxoid and routine antibiotic cover; secondly, liver trauma may be followed by Gram-negative infection in damaged liver substance.[17]

Daily supplements of vitamin K 10 mg intramuscularly were given. It is not known whether this is beneficial in the immediate postoperative period, but we thought it advisable since the body storage of vitamin K is reduced after the removal of a large part of the liver and prolonged bile drainage through a T-tube accompanied by the use of broad-spectrum antibiotics may interfere with the absorption of vitamin K from the gut.

Monitoring of central venous pressure was continued after operation and blood transfusion was used as required, whenever possible with fresh blood. A nasogastric tube was left in situ and intravenous fluid and electrolyte replacement continued until oral feeding became possible.

According to McDermott and his colleagues[18] the blood sugar falls after resection of 70-90% of the liver, but neither Pack and Molander[19] nor Lin and Cheng[20] found any significant change in the blood sugar level in their patients, and Aronsen and Ericsson[21] did not record hypoglycaemia. However, they administered large quantities of sugar during the postoperative period. Hypoglycaemia occurred in only one case (Case 2, Table VI), but it was life threatening (blood sugar 15 mg/100 ml despite an intravenous infusion of dextrose 5% in progress) and 50 ml of 50% glucose solution had to be given intravenously every four hours.

Many clinicians give intravenous albumin after liver resection.[7 18 22 23] Some[21] have claimed that this averts the hypoalbuminaemia known to accompany resection of large amounts of liver substance. We gave daily supplements of albumin 75 g for five days in two cases (Cases 1 and 2). It had no appreciable effect (Fig. 1) and the practice was abandoned.

The serum bilirubin rose immediately after operation (Fig. 1) and was accompanied by clinical jaundice. In the uncomplicated cases the serum bilirubin returned to normal in 10-14 days, but in two it remained raised. In Case 1 the serum bilirubin remained raised until the patient died. In Case 3 it remained raised for some five weeks, during which a biliary leak developed at the site of the choledochotomy and a bile-stained pleural effusion was aspirated before the bilirubin fell (Fig. 1).

POSTOPERATIVE JAUNDICE

There is no complete explanation for the jaundice that occurs after partial hepatectomy. It has been claimed that the level of bilirubin varies with the extent of liver resection[3 24]; however, there is little evidence that this is so. Some features are suggestive of obstruction, but even though the jaundice can be associated with retention of conjugated bilirubin (Case 1) the serum alkaline phosphatase remained within normal limits in the present cases except one (Case 3, Fig. 1). A severe reduction in liver cell mass with diversion of the portal blood flow into the small remaining segment of the liver may lead to engorgement and increased intrahepatic pressure, and consequently there may be pressure on the small intrahepatic biliary radicles.[25]

Others have suggested that the postoperative jaundice may be due to increased pressure within the bile ducts.[24] We find it difficult to accept this since T-tube drainage did not influence the development of jaundice in any of our cases. We found that the serum alanine transferase rose after hepatectomy and fell to normal levels within 10 days (Fig. 1). Others have made similar observations.[24] Hepatocellular damage however, is unlikely to be entirely responsible for the jaundice. Massive blood transfusion with haemolysis may account for some of it but, as has been pointed out,[14] there is bilirubinuria and the picture is not typically haemolytic in character. In one of the present cases (Case 3) the continuing jaundice seemed to be related to an extrahepatic biliary collection. It has been shown experimentally that the liver's capacity to excrete bilirubin is decreased after partial hepatectomy,[26] and this may also apply in man.

HAEMORRHAGIC DIATHESIS

Haemorrhagic diathesis is a well recognized problem after hemihepatectomy.[3 14 21] It occurred in four of the patients in this series and presented either as a defect in clotting or profuse oozing from the wound. In one case (Case 2) the defect in clotting was noticed preoperatively, and the low fibrinogen level, prolonged clotting time, prolonged prothrombin time, and low platelet count point to the existence of disseminated intravascular clotting, producing an acute defibrination syndrome.[27 28 29] Intravascular clotting is presumably activated by the release of tissue thromboplastin (factor III) as the result of massive crushing of liver tissue after trauma or of liver embolization, as reported by Strauss.[30] Acute defibrination

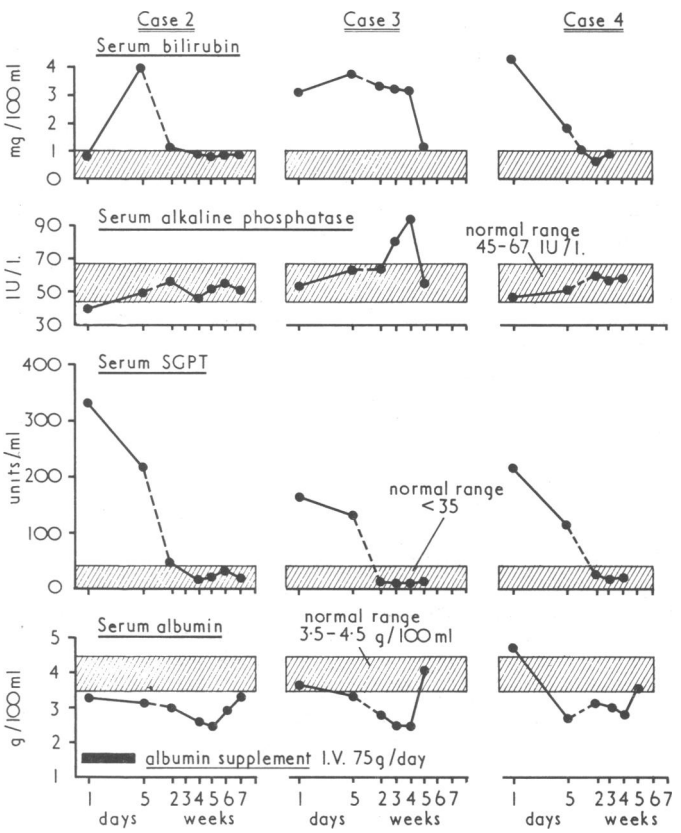

FIG. 1—Liver function in three cases surviving hemihepatectomy. Albumin supplements were used in Case 2 but not in Cases 3 or 4.

syndromes occur in liver transplantation when ischaemia of the donor liver causes cellular necrosis.[31]

The haematological evidence in the other three cases (Table VII), though suggestive of an acute defibrination syndrome, is not as strong as in Case 2, since all three had had large blood transfusions before the coagulation studies. Nevertheless, one other patient (Case 1) had a low fibrinogen level associated with a deficiency of factors V, VII, and IX. In each case the haemorrhagic tendency was controlled by the administration of fresh blood, fresh plasma, triplestrength plasma, and vitamin K.

TABLE VII—*Coagulation Studies in Cases with Haemorrhagic Diathesis*

Case No.	Fibrinogen Titre (1/32-1/128)	Thrombin Clotting Time* (min)	Prothrombin Time* (min)	Kaolin-Cephalin Clotting Time* (min)	Platelets (/mm³)
	Absolute value				
1	130 mg/100 ml	13 (5)	18 (12)		50,000
2	1/4	60 (11)	22 (11)	68 (36)	28,000
3	1/32	15 (11)	18 (14)	65 (47)	105,000
4	1/64	14 (11)	23 (14)	65 (39)	<10,000

* Normal values given in parentheses.

TABLE VIII—*Complications in the 20 Cases of Liver Injury*

	No. of Cases		No. of Cases
Haemorrhagic diathesis	4	Traumatic aneurysm of descending aorta	1
Frank haemorrhage	3*	Pancreatitis	1
Bronchopneumonia	5	Acute renal failure	1
Empyema	1	Abortion	1
Subphrenic abscess	1	Myocardial damage	1
Biliary leakage	1		
Hypoglycaemia	1		

* Two cases associated with haemorrhagic diathesis.

A summary of the complications which occurred in all the 20 cases of liver injury is given in Table VIII. Bleeding and respiratory difficulties were the major problems and have already been referred to.

LIVER REGENERATION

The liver of the patient who died 10 hours after hemihepatectomy (Case 5) showed no macroscopic evidence of increase in size. It is of interest that the line of resection was clean. Haemorrhage had stopped and there was no undue compression caused by the catgut sutures coapting the cut edges of the liver.

The liver of the patient who died 10 days after hemihepatectomy (Case 1) was of a surprisingly large size and weighed 1,700 g despite the recent removal of the whole right lobe. Liver scan was not performed in this patient and it was impossible to correlate the scan appearances with the size of the liver at necropsy.

The three surviving cases were assessed by liver scans after injection of technetium sulphur colloid (^{99m}Tc), using either a rectilinear scanner or a gamma camera. Scans were done at intervals after operation. In Case 2 scans were done from the third postoperative day up to nine months. The liver remnant had greatly increased in size by the tenth day but the further increase over the following months was small. (Fig. 2). The speed of regeneration was approximately that recorded at necropsy in Case 1. In the other two cases the scans increased little after the first postoperative month.

Results

Out of the total of 20 patients with liver injury (14 minor and 6 major lacerations) 4 (20%) died—two from haemorrhage, in one case associated with a haemorrhagic diathesis; one from bronchopneumonia; and one from renal failure associated with pancreatitis and myocardial damage. The mortality rate is analysed in Table IX. Only one of the patients with minor liver lacerations died. Death was due to renal failure, and pancreatitis and myocardial damage were found at necropsy. The other three deaths occurred in patients with major damage to the liver. One died as a result of continuing haemorrhage

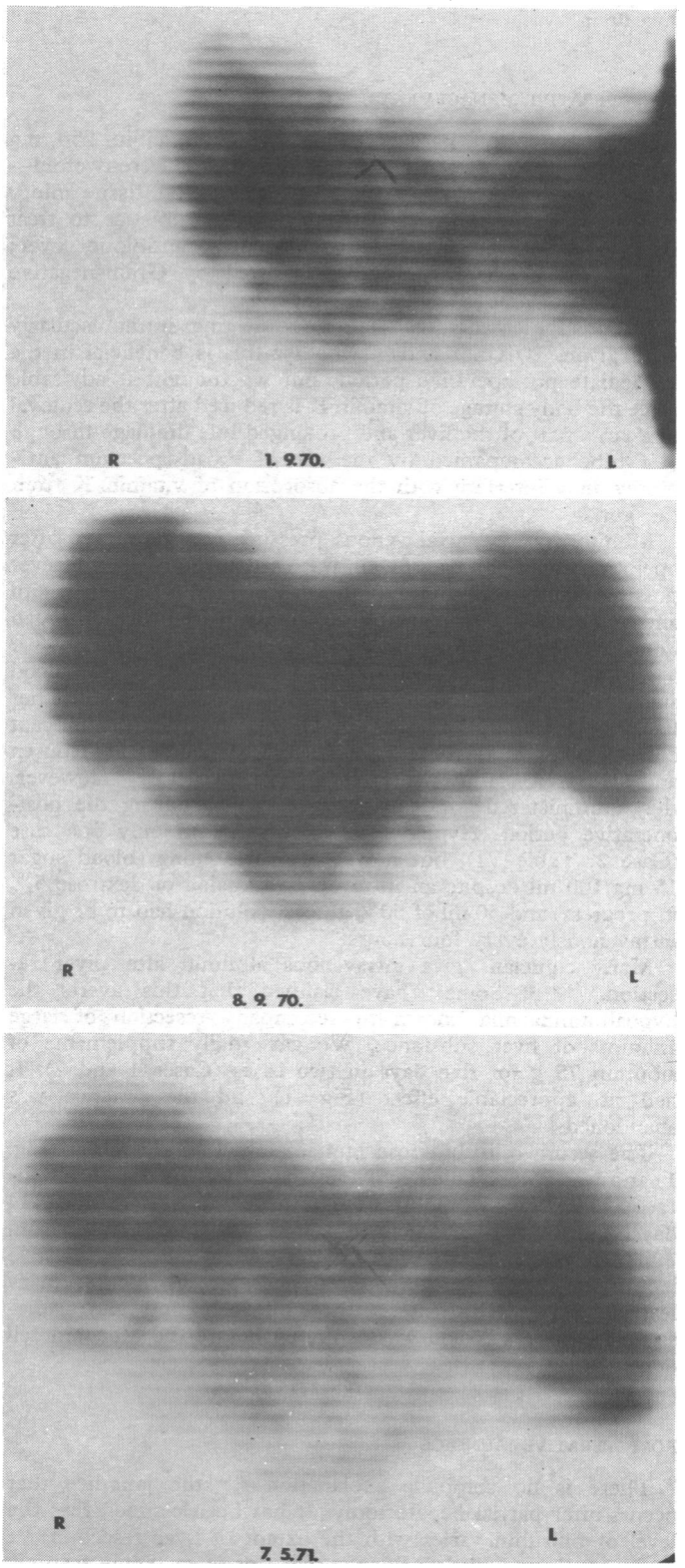

FIG. 2—Rectilinear scans obtained three days, 10 days, and nine months after right hemihepatectomy (Case 2.) Note the rapid increase in size of the liver remnant within the first 10 days postoperatively with little increase thereafter.

TABLE IX—*Mortality Rate in Patients with Liver Injury*

Type of Injury	No. of Cases	No. of Deaths	Mortality (%)
Minor	14	1	7
Major	6	3	50
Total	20	4	20

after packing. Two of the five patients submitted to hemihepatectomy died, one from haemorrhage originating in the azygos vein and the other of respiratory insufficiency accompanied by pneumonia. Only one death was attributable to the liver injury alone.

Discussion

The high incidence (20 cases) of damage to the liver in the 44 cases of blunt injury to the abdomen in this series is similar to the experience of others,[1 2] as is our finding that nearly all such injuries are the result of road accidents.[1 9]

It is striking that liver damage alone occurred in only two cases. This is somewhat different from previous experience in the United Kingdom[9] and may be a reflection of the severity of the injuries in our series, most of which were associated with high-speed accidents. The finding that the right lobe of the liver or the region of the ligamentum teres was injured in all these cases but that the left lobe was never damaged is similar to the findings of others.[1 3 9] The overall mortality of 20% is comparable to the 24% recorded by Little and Williams.[1] That all but one of the deaths occurred in patients with severe multiple injuries is in keeping with the observation that multiple injury can raise the mortality to as high as 67%.[5]

Most liver injuries can be dealt with by simple suturing, and 14 of the 20 cases in our series were satisfactorily treated in this manner. This has been the experience of many others.[1 3 32-34] Such wounds are not usually deep or associated with pulping of adjacent liver parenchyma. We did not leave any wound unsutured, as advocated by Little,[3] nor did we find it necessary to drain any of the wounds.

A difficult decision has to be taken, however, when extensive damage to the liver is found. We treated our first two cases of major liver laceration by methods that were subsequently recognized as inadequate. After that we thought it necessary to define a policy on which a reasonable decision to proceed with liver resection could be taken. We classified as major all wounds whose depth was such that they could not be completely closed, haemostasis secured, and dead space obliterated by simple suture. Wounds in which there was severe associated disruption of the liver substance were also classified as major. Simple suture of such wounds is inadequate and carries a high risk of recurrent bleeding, and our experience after packing a deep wound of the liver has been that of others.[34-36] Not only may there be secondary haemorrhage but when the liver parenchyma is pulped dead or severely devitalized tissue may be left behind. Damage to the liver tissue is aggravated by ischaemia from compression by the pack, and necrotic liver tissue is particularly sensitive to Gram-negative infection.[17] In common with others,[3 6 7 18 32] we now advocate liver resection for extensive liver lacerations.

Haemostasis was achieved in all cases of right hepatic lobectomy. The insertion of a T-tube into the common bile duct to decompress the biliary tree was originally suggested by Merendino and his colleagues[37] and has been adopted by others.[1 3 24] Some have debated whether the procedure prevents biliary leakage,[7] and in our Case 2 the leakage seemed to arise at the site of the choledochotomy. Nevertheless, our impression is that the T-Tube is a useful means of decompression after hemihepatectomy and no appreciable leakage of bile occurred from the raw surface of the liver in any of these patients. Further, as emphasized by Little and Williams,[1] a T-tube serves as a useful avenue of investigation.

LIVER REGENERATION

Recovery of liver function was shown within two weeks of injury and was well advanced by the fifth or sixth week after operation in the present series. This confirms the findings of others.[18 21]

Though it has been estimated that replacement of liver mass may take as long as four to six months after partial hepatectomy[18 38] recent reports suggest that the process may be much more rapid, and especially so in the first 7 to 14 days after operation.[14 39] Exact measurements of the rate of regeneration of the liver in man are difficult to obtain. Pack *et al.*[22] suggested that growth may be as rapid as 50 g or more a day. It is of interest that the postmortem evidence in one of our patients suggests that regeneration begins shortly after injury and is well advanced within 10 days. An estimate of the rate of regeneration during this period would be close to 100 g a day. The postoperative liver scans in the other cases tend to confirm this and show little change in the size of the liver remnant after the first postoperative month.

Complete regeneration of the liver in the rat occurs within three weeks after removal of 67% of the liver mass. This immense reparative reserve is set in motion very soon after hepatectomy, with a peak rate of mitosis occurring one day after operation.[40 41] It may well be that a not dissimilar time sequence occurs in man.

The cases reported in this series were operated on by one of us (L.H.B.) at Cardiff Royal Infirmary, the Royal Infirmary, Sheffield, and Nottingham General Hospital. We record our thanks to the following surgeons under whose care some of the cases were admitted: Mr. C. D. P. Jones, Mr. J. F. Sheehan, Mr. T. Field, Mr. J. Swan, Mr. H. H. Renyard, and Mr. D. L. Crosby. We also acknowledge the help of Drs. A. S. Bligh and K. G. Leach, of the departments of diagnostic radiology and medical physics at Cardiff Royal Infirmary, who were responsible for the liver scans.

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Clinical Problems

Torsion of the Testis and its Appendages

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Summary

In a survey of 101 cases with an index diagnosis of torsion of the testis or its appendage, there were 86 instances of torsion of the testis and 14 of torsion of the hydatid of Morgagni. In 38 of the patients with testicular torsion there were one or more "warning" attacks of pain and swelling, due to torsion which became spontaneously untwisted. In 19 cases operative fixation was performed because of this warning history, and all the testes were preserved. In the other 19 the testes were not explored until the patient came in with a severe "classical" episode of torsion: seven of these testicles were lost from gangrene or delayed atrophy.

In 31 cases torsion resulted in failure to save the testis. Of these, 20 were misdiagnosed and treated initially as epididymo-orchitis, despite there being no evidence of a urinary tract infection. There is no excuse for not exploring any actuely inflammed testicle which is unaccompanied by definite evidence of urinary or urethral infection.

Introduction

Though torsion of the testis was described by Delasiauve as long ago as 1840[1] it has taken an unconscionable time to get into the general awareness of doctors and to become accepted as a relatively common and serious surgical emergency. At the London Hospital neither Curling[2] nor Hutchinson[3] were aware of this condition when writing about the testicle, and it was not until 1907, when Rigby and Russell Howard[4] wrote their classical paper on torsion, that the condition was widely recognized. Since then there has been no want of interest in torsion or lack of awareness about it in this hospital.[5-9] In spite of this cases are still explored at too late a stage for the testicle to be saved. We recently asked ourselves why this disaster is not prevented more often than it is. Where does the fault lie? Can anything be done to improve matters? In an attempt to answer these questions we have reviewed the experience at this hospital over the past 23 years.

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Methods and Materials

We have examined the records of 101 cases with an index diagnosis of torsion of the testis or its appendages referred to the London Hospital during 1948 to 1971 or treated by one of us (A.J.W.) in either of two regional hospitals during the same period. The 91 London Hospital cases follow on the series described by Kennedy.[5]

On scrutiny of the case records of these 101 cases the index diagnosis of torsion of the testis was accepted in 85 instances, of torsion of the hydatid of Morgagni in 14, and of torsion of a lipoma of the scrotum in 1 case. One case was rejected; in retrospect it was clearly an example of idiopathic oedema of the scrotum.[10]

Operative Technique.—Our policy is to explore the affected testicle through a transverse scrotal incision. The tunica vaginalis is opened longitudinally and the diagnosis confirmed. The twist is undone and the viability of the testicle assessed. If it seems viable the tunica vaginalis is everted, as in a Jaboulay procedure, and three chromic catgut sutures are passed through the dartos and tunica albuginea of the testicle to prevent the possibility of further torsion. The other testicle is explored and fixed in a similar manner at the same session. The skin is closed with catgut sutures without drainage. The reason for fixing the other testicle at the same time is because of the possibility of torsion in this testicle. In most of the cases where the other side was explored there was an abnormality in the anatomy similar to that on the side in which the torsion occurred.

Torsion of Testis

Torsion of the testis occurred in 85 patients, in one of whom it was bilateral.

Aetiology.—The left testis was affected nearly twice as often as the right (54:30). The ages of the patients ranged between 5 weeks and 54 years, and there was the usual preponderance of adolescent cases (Table I). Maldescent was associated with