THE SURGICAL MANAGEMENT OF HEPATIC TRAUMA AND ITS COMPLICATIONS

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by

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When the liver is wounded, much blood commeth out at the wound . . . the blood that falleth from thence downe into the intestines doth oftentimes inferre most maligne accidents, yea and sometimes death.—PARÉ.

STABBINGS, KICKS AND clubbings have long been part of the human condition. More recently, injuries from gunshots and automobiles have been added. Over many centuries, little could be done for the victim as noted by Hippocrates when he wrote that 'a severe wound of the liver is deadly'. For the next 2,000 years hepatic wounds remained essentially in the province of the military surgeon. Yet John Hunter, in dedicating his treatise on 'Blood, Inflammation and Gunshot Wounds' to King George III in 1793, in which he notes that he had been given 'extensive opportunities of attending to gunshot wounds, of seeing the errors and defects in that branch of (military) surgery, and of studying to remove them', makes only two brief mentions of wounds of the liver. A practical surgical approach to the problem had to wait on the discovery of general anaesthesia. It was 77 years after Hunter's death that Bruns performed the first successful resection of a portion of the liver in 1870. Subsequent experiences in a succession of wars increased the familiarity of surgeons with serious hepatic injury and established principles which finally reduced mortality rates to 27 per cent in World War II.

While military surgeons wrestled with the problems of penetrating trauma with relative success, far less progress was made with the lesions associated with blunt trauma. Observations on closed injuries of the liver were first limited to case reports and related post-mortem findings. Writing from Cape Town in 1843, Abercrombie recorded the history of a patient with two small lacerations following abdominal compression 'by a silk handkerchief' and noted that 'comparatively slight external injuries have ruptured this organ, and caused fatal haemorrhage into the abdomen'. Owen (1848) gave a graphic description of an intoxicated young man who, thrown from his light chaise, suffered a closed abdominal injury and hypovolaemic shock. Over the next 10 days before his death, he became dehydrated, septic, jaundiced, uraemic and finally developed a haemorrhagic tendency as evidenced by epistaxis and melaena. At post-mortem, he was shown to have a large laceration of the right lobe of the liver and an intra-hepatic collection of purulent blood clot. Owen reflected on 'how little sign existed of extravascated blood and peritoneal inflammation succeeding so severe an injury'. It has, in fact, remained

for the automobile to bring our understanding to fuller flower (Mills, 1961; Baker *et al.* 1966; DiVincenti *et al.* 1968). And so, perhaps, it is not inappropriate that this study, which concentrates on the management of this modern lesion, should emanate from Detroit.

There is little doubt that the incidence will continue to rise in areas where new highways are devoured by increasingly powerful automobiles or where civil strife reaches uncivil proportions. Until recently 30 to 60 per cent of patients with closed liver injuries have failed to reach the hospital alive (Hellstrom, 1961). Now, as helicopters and high-speed vehicles staffed by trained personnel are used to transport civilian casualties without delay directly to well-equipped emergency rooms, the victim who would previously have been exsanguinated may be saved if certain principles are clearly understood and energetically followed. It is only 20 years since appreciation of the segmental nature of the hepatic anatomy encouraged the same precise surgical approach which had been brought to bear on the lung 15 years earlier (Healey, 1954; Goldsmith and Woodburne, 1957). The purpose of this lecture is to review the many varieties of hepatic injury, the ubiquity of the clinical picture, some important technical aspects of the surgical approach, the spectrum of complications which may ensue and the metabolic sequelae.

CLINICAL SURVEY

The observations which follow are based on 500 consecutive patients with hepatic injury treated surgically on the Wayne State University service at Detroit General Hospital between 1st July 1961 and 7th January 1969 (Table I). Two hundred and seventy of these patients suffered stab

TABLE I

AETIOLOGY OF LIVER TRAUMA

Penetrating wounds	443 (8.1%)		
Stab wounds		270 (1.9%)	
Gunshot wounds		173 (17.9%)	
Blunt trauma	57 (30.0%)		
Automobile accidents		44 (31.8%)	
Drivers			20 (30.0%)
Pedestrians			13 (23.4%)
Passengers			9 (55.5%)
Unstated			2 (0.0%)
Other blunt injury		13 (23.0%)	
Total	500 (10.6%)		

Numbers in parentheses represent mortality rate.

wounds, 173 gunshot wounds and 57 closed abdominal injury. While the mortality rate of those stabbed was only 1.9 per cent and of those shot 17.9 per cent, no fewer than 31.0 per cent (18/57) with blunt trauma died. The great majority with blunt injury were between the ages of 10 and 40, and this group, still in the prime of life, presents the greatest challenge to our surgical skills.

Mechanisms of injury

The liver is the largest of the intra-abdominal organs and is frequently injured despite the protection it receives from the thoracic cage. Of 307

patients with blunt abdominal trauma seen at Detroit General Hospital, 57 (18.6 per cent) sustained hepatic injury (Walt and Grifka, 1969). While the pattern of injury is obvious in penetrating trauma, there are several variations in cases of blunt trauma. The net results are more easily understood if the liver is viewed as a syncytium of fluid channels containing blood, lymph and bile, buffering sheets of soft hepatic cells (Fig. 1). It is a heavy mobile organ, suspended from the diaphragm by the coronary and triangular ligaments and maintaining essential con-

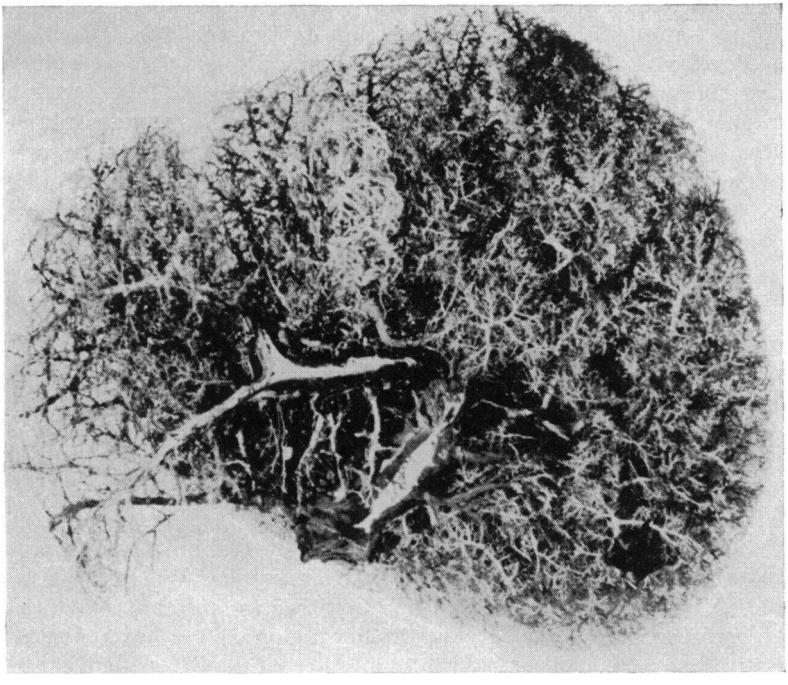


Fig. 1. A cast of the liver which demonstrates the syncytium of veins, arteries and bile ducts.

nections through the portal triad and delicate hepatic veins. Most injuries can be classified as either shearing or fracturing.

Sudden shearing forces directed to this large inert organ may result in parenchymal tears close to the attachment of the suspensory ligaments. Far less commonly, transection of the hepatic veins or damage to elements of the portal triad may occur. The lacerations are usually single, up to 15 cm. in length, and fairly shallow, but these may on occasion extend for 6 cm. or more into the liver substance with an increased danger of serious bleeding. Characteristically, but not exclusively, these lesions tend to occur in occupants of the automobile and are thought to reflect

the sudden deceleration force which causes the liver to oscillate and twist rapidly.

In contrast, direct blows to a liver entrapped in the right upper quadrant may produce deep jagged multipronged fractures, usually in the posterior and superior aspects. These occur mainly in pedestrians and in the course of violent assaults. In a few patients, however, direct blows may generate sudden violent internal pressures which have an explosive effect. In these, deep disruption of liver tissue may result without being proportionately reflected on the capsular surface which may show little more than a few stellate cracks or subtle bruises. Where this intra-parenchymal damage is not recognized, delayed rupture or subsequent haemobilia may develop as the ischaemic tissue gradually breaks down and bile and blood collects under increasing pressure. Subjecting cadaver livers to a variety of graded impacts, Mays (1966) demonstrated progressively more severe lesions ranging from superficial tears at 30 foot-pounds to wide internal destruction at 360 foot-pounds. Rarely, a subcapsular haematoma may develop which, growing larger with time, ruptures within a few days, producing serious haemorrhage.

Stab wounds

Unless a large vessel is transected or an adjacent organ such as the lung, pancreas, or colon perforated, stab wounds present few difficulties. The great majority have ceased to bleed by the time of laparotomy, which consequently serves mainly to confirm this fortunate fact. In view of this, some investigators (Stein and Lissoos, 1968) have recommended a policy of close observation without operation, reserving laparotomy for those patients with an unstable clinical picture. If there is still active bleeding, haemostasis is secured with a few interrupted chromic catgut sutures which may be passed through pledgets of gelfoam or omentum to prevent the sutures from cutting through the liver tissue. Where there is no bleeding, sutures are unnecessary. All these wounds are well drained with Penrose drains placed close to the region of the lesion for a few days in case a biliary leak occurs. Only five of the 270 patients (1.9 per cent) died, four of haemorrhage or damage to adjacent organs and one of unexplained cardiac arrest due probably to aspiration at the end of the operation. The complication rate, mainly wound infection and atelectasis, was 12 per cent.

Gunshot wounds

Gunshot wounds are much more serious. The mortality rate of our 173 patients was 17.9 per cent and the outcome in the individual patient was related to the associated damage to adjacent organs and to the aorta, portal vein or inferior vena cava as much as to the liver damage itself. In the last 74 patients with gunshot wounds, 23 (31.0 per cent) had an isolated hepatic injury. The remaining 51 patients had concomitant injury in descending order of frequency to the diaphragm (20),

chest (12), kidney (8), extremities (7), colon (7), pancreas (6) and duodenum (3).

The problem of the extent of optimal debridement to adjacent parenchyma often presents itself when there is a clean through-and-through wound. We have chosen for the most part not to open tracks caused by low-velocity missiles. Where there is extensive damage by large calibre, high-velocity bullets, the damaged liver is unroofed and all devitalized tissue removed. With haemostasis achieved, the area is well drained, and choledochostomy or cholecystotomy is usually established although the rationale for these procedures remains to be proved. Occasionally, hepatic lobectomy is necessary. Thirteen of the 173 patients required resections of at least one segment of the liver. Of the last seven performed, only one patient died.

Blunt injuries

Forty-four of our 57 patients with blunt trauma were injured by automobiles, 20 as drivers, 13 as pedestrians and 9 as passengers. In two, it is

TABLE II

ASSOCIATED INJURIES IN BLUNT LIVER TRAUMA

Associated injury in 57 patients	*Number of patients*	*Mortality rate per cent*
None	6	0
Head	23	22
Chest	18	33
Extremities	13	56
Spleen	13	70
Pelvis	9	50
Pancreas	9	25
Duodenum	4	25
Kidney	5	75
Colon	2	0

not known whether they were inside or outside the car. The remaining 11 patients suffered a variety of injuries including falls from heights, kicks and beatings with objects such as baseball bats. Four patients with abdominal pain were admitted to the gynaecological service, two with vaginal bleeding and a diagnosis of ectopic pregnancy. No history of trauma was immediately obtainable and consequently undesirable delays occurred while gynaecological investigation and subsequent observations took place.

Rupture of the liver occurs as an isolated lesion in only about 10 per cent of victims of blunt abdominal trauma. In most cases, therefore, the picture tends to be dominated by the associated injuries, most often of the head, thorax and extremities, and it is these injuries which usually determine the mortality rate (Table II). Seventeen of our 57 patients died (31.0 per cent.). Of these, a number were virtually moribund on admission and some died many weeks after operation. Six patients of the 57 had a total or segmental lobectomy; four survived.

Clinical picture

The clinical picture may be most deceptive and only unfold gradually. Classically, the patient is hypotensive with a rigid abdomen, absent or hypotensive bowel sounds, and in hypovolaemic shock. As noted by John Hunter, about 20 per cent will complain of pain referred to the right or left shoulder. Surprisingly, many patients who subsequently prove to have substantial hepatic damage are remarkably well when first admitted. Up to 30 per cent show no immediate hypotension, and "initial splinting" may actually diminish for a few hours as the abdomen distends with blood and the patient becomes hypovolaemic. This paradox constitutes a potential danger as catastrophic haemorrhage may occur once the tamponading effect of the intra-peritoneal blood is lost when the abdomen is opened. It is for this reason that adequate intravenous catheters, monitoring devices and plentiful blood should be available before laparotomy is undertaken. Head injury is never acceptable as an explanation of shock and abdominal injury is always suspected in the unconscious patient, especially when signs of hypovolaemia are present. The presence of fractured ribs on the right is ominous and any combination of head injury, tyre marks on chest and injury to the legs suggests that structures between these points may well have been damaged too. In addition, other clues such as ecchymoses or skin burns of the abdominal wall are also sought as these are sometimes the only clues in the comatose patients.

DISCUSSION

The fundamental principles governing safe hepatic surgery which have evolved over the past 20 years are based on new anatomical and physiological concepts and an appreciation that adequate operative exposure frequently demands concomitant thoracotomy. In addition, advances in blood banking, respiratory care and restoration of homeostasis have been crucial in improving results. This is best seen in our own hospital, where the mortality rate was 90 per cent in 10 patients with blunt hepatic trauma treated between 1927 and 1934; 35 per cent in 20 patients treated between 1954 and 1959; and 31.8 per cent in the 57 patients treated between 1961 and 1969. The improvement is more striking than is immediately apparent as the most recent gains are obscured by the fact that, with improved ambulance facilities, patients who would have previously died *en route* to the hospital now arrive moribund with major injuries and enter the series. As high-speed transport and helicopters are introduced to emergency services, more such victims will be encountered.

The main factors determining the ultimate mortality rate are haemorrhage from the liver and the number and severity of associated injuries. In this group of 57 with blunt injury, five of six patients survived in whom the liver was the only organ injured, but the mortality rate increases to approximately 17.0 per cent with two associated injuries and is more

than 50 per cent with four associated injuries (Kindling *et al.* 1969). Extra-abdominal lesions are treated on their own merits but must not be permitted to distract the surgeon from the possibility of concomitant hepatic injury. This error is most often encountered when the patient is excessively drunk, unconscious or grossly confused due to a head injury, or hypoxic from respiratory obstruction. In cases of doubt, we have preferred in these circumstances to make a small vertical incision in the mldline above the umbilicus. Through this, the sterile gloved finger or a suction catheter can be inserted into the peritoneal cavity. While the use of the four-quadrant tap with needle or catheter has its proponents, we have found it less reliable. If the patient is conscious, it is the clinical picture which determines the need for laparotomy; if he is comatose, exploration through a small incision is more accurate. The cause of any

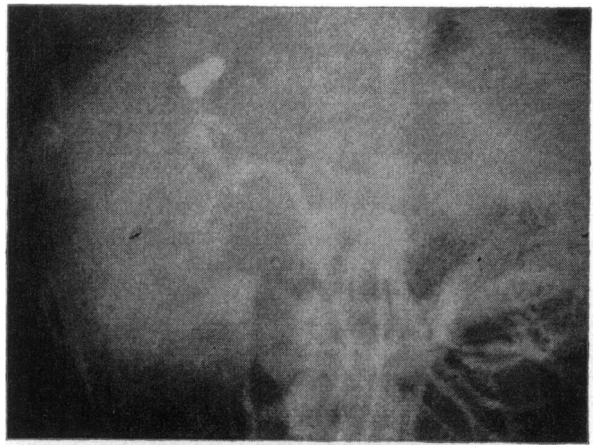


Fig. 2. Hepatic angiogram which demonstrates absence of vessels in the superior aspect of the right lobe which has been struck by a bullet.

shock must be relentlessly pursued, remembering always that there may be more than one source. A blow over the lower chest, for example, may produce hepatic fracture, myocardial contusion, pneumothorax and a ruptured abdominal viscus. The potential of pre-operative aortography and selective coeliac angiography has been explored (Freeark, 1969), but these are not reliable and, when positive, serve only to confirm the clinical indications for operation. Intra-operative hepatic angiography may be of assistance in the uncommon 'bursting' injury in which it may be difficult to delineate the extent of intraparenchymal destruction. The value of liver scanning using Au^{198} has also been investigated but appears to add little in the acute stages (Little *et al.* 1967). Both angiography and scanning techniques, however, may be decisive in the diagnosis of intra-hepatic necrosis, haematomas and abscesses (Figs. 2 and 3).

Major injury to the liver demands urgent surgical management. The

rapidity of the continuing blood loss will frequently exceed the surgeon's ability to replace it and the administration of excessive quantities of banked blood tends to disrupt homeostatic mechanisms, especially in the hypoxic patient. These patients are further threatened by the presence of hepatic parenchymal destruction as the liver plays a central rôle in

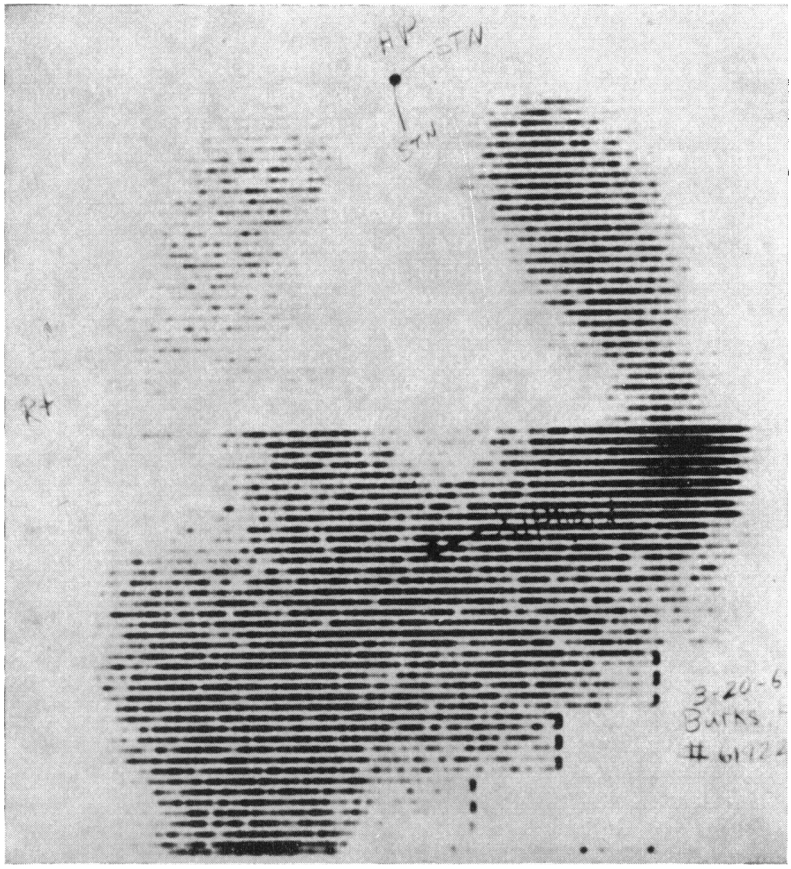


Fig. 3. Combined lung and liver scan which delineates a large filling defect. At operation, a large intrahepatic abscess in the posterior segment of the right lobe was successfully drained.

the coagulation mechanism, in the metabolism of citrate and perhaps in the production, under these circumstances, of harmful vaso-active substances. Early clinical diagnosis is therefore paramount, but it may not be easy. As haemorrhage is directly responsible for death in more than half the cases, operation is designed to control this as rapidly and

expeditiously as possible. In preparation for this, a No. 15 polyethylene intravenous catheter is inserted into each arm to deliver the large quantities of blood and Ringer's solution that may be rapidly needed. One of these catheters is placed in the superior vena cava to monitor the central venous pressure closely.

General principles of the operative management

The abdomen is opened through a midline or paramedian incision with preparations having been previously made to extend this into the 8th intercostal space without hesitation, if necessary. In most patients, the damaged segment will be easily accessible and repair can be accomplished without the necessity of opening the thoracic cavity. Linear tears in relation to the suspensory ligaments are treated much like stab wounds. Individual bleeders, if any are active, are suture-ligated and devitalized tissue is removed. The hepatic wound is then closed with deep catgut sutures in such a way as to obliterate any potential dead space which could otherwise serve as the site for an expanding haematoma and subsequent haemobilia.

In the presence of more severe trauma, surgical decisions are more difficult and the problems of the surgeon tend to be proportional to the severity of the parenchymal damage. Grossly disrupted tissue is easily recognized and is removed together with the less obvious adjacent ischaemic tissue which tends to be cyanotic or tan in colour. This is done in piecemeal manner if necessary until some degree of bleeding is encountered. Unfortunately, clear definition of the extent of residual ischaemic tissue may still be uncertain after wide debridement. We have attempted to delineate this area by the injection at operation of 5 c.c. fluorescein into the hepatic artery and the subsequent application of ultraviolet light to the liver. With the field kept dry to avoid spurious staining from open vessels, the vascularized areas will fluoresce rapidly while the ischaemic areas do not. As yet we do not have sufficient experience to offer a considered opinion of the value of this manoeuvre.

Complete saucerization of the hepatic wound, sometimes referred to as resectional-debridement, may remove 300 G. or more of tissue, creating a large defect. Closure of this hepatic cavity should obey the usual principles of wound care. The configuration of the injury after wide debridement and suture-ligation of individual vessels and ducts determines whether the surfaces can be approximated by a series of interrupted sutures which obliterate the dead space. This is done wherever possible and a small suction catheter may then be led from the depths of the hepatic wound to remove collections of blood and bile during the early post-operative days. In contrast, where the hepatic defect is broad and relatively shallow rather than wedge-shaped, the walls of the saucerized cavity cannot be brought together easily. Under these circumstances, multiple drains, including a sump, are inserted down to the raw area which is covered by omentum. Occasionally, direct blows to the liver

and bursting injuries provide special pitfalls. Although damage may in fact be extensive, the capsular surface may show no more than a few minor stellate cracks and a dusky discoloration, disguising the disrupted and infarcted tissue in the centre of the lobe. This is more likely to be found on the right side and it may be very difficult to identify the true extent of the injury. In these uncommon circumstances, the affected liver deep to the capsular lesions is unnaturally soft. With this clue, the use of intra-operative hepatic angiography may be of valuable assistance in defining the need to remove or resect the pulped liver tissue.

In some patients the problem may be still more complex, when, on release of the abdominal tamponade, the collection of pooled blood is augmented by a rush of fresh and sometimes catastrophic haemorrhage. Under these conditions, the hepatic wound is immediately packed tightly with gauze and the portal triad is occluded digitally (Pringle's manoeuvre) or with a soft rubber-shod clamp. If this manoeuvre achieves a respite from bleeding and the damage is localized to a lobe, the clamp may be adjusted and the artery supplying the affected area may be selectively occluded, permitting oxygenation of the uninvolved portion of the liver. This may be vital as the response of hepatic cells to anoxia is not accurately predictable. It is generally agreed that the portal triad can be safely occluded for up to 15 minutes under normothermic conditions and that this can be extended to 50 minutes under planned hypothermia. Sometimes ignored in these critical decisions is the duration and extent of hepatic hypoxia that has unavoidably existed prior to any deliberate occlusion. Nonetheless, in this desperate circumstance, we have successfully extended the period of 15 minutes to about 20 to 24 minutes in a few patients who were incidentally hypothermic from the administration of large quantities of stored blood, with a core temperature of about 32° C. Special attention is directed to combating the predictable acidosis which occurs on release of the occluding clamp.

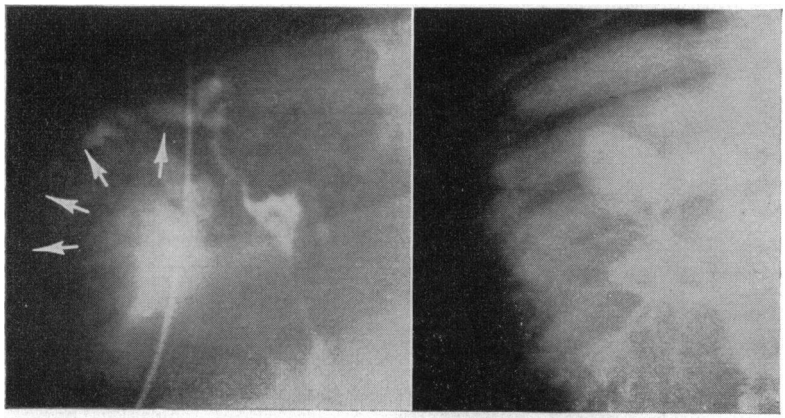
If haemorrhage continues despite Pringle's manoeuvre, and the fracture is situated over the dome or posteriorly, it is probable that one or more hepatic veins or the inferior vena cava have been torn or that the liver is partially supplied by an anomalous branch of the hepatic artery. Attempts to approach this area by downward traction on the liver will often serve to extend the laceration and increase the blood loss. In these circumstances the incision should be immediately extended into the right chest and the diaphragm opened. This gives ready exposure to this otherwise inaccessible area and prevents the occurrence of air embolism through the open subdiaphragmatic veins which is a major hazard until the negative intrathoracic pressure seal is broken. This latter complication is believed to be a potent cause of death (Aronsen *et al.* 1968) in this type of injury. With the peritoneal suspensory ligaments of the liver transected and increased mobility and improved exposure gained, conscious efforts must be taken to avoid occlusion of the inferior vena cava as this will

reduce the return of blood to the heart and lead to sudden cardiac arrest. Any lacerated hepatic vein is suture-ligated under direct vision where possible. Where this is not feasible, partial occlusion digitally or by a Satinsky clamp may be of great assistance, but every precaution must be taken not to tear the fragile inferior vena cava. Management of an extensively lacerated inferior vena cava or multiple torn hepatic venous tributaries in this region remains one of the great unsolved problems of emergency surgery. The approach is difficult and total occlusion of the inferior vena cava for more than a few minutes is lethal. Techniques to obviate this have been suggested; Shrock *et al.* (1968) have tried unsuccessfully to insert an intra-caval shunt through the right atrium. Others have reduced the splanchnic inflow by clamping the supra-coeliac aorta (under hypothermia) to permit occlusion of the inferior vena cava for a few minutes during which the repair is attempted. Exposure of the supra-diaphragmatic inferior vena cava in these circumstances may be best achieved by opening the pericardium. Whatever manoeuvre is adopted, the prognosis in this type of injury remains very poor.

Once the immediate life-threatening haemorrhage is temporarily controlled, the degree and anatomical distribution of the damage may be carefully assessed. In a minority of cases, there is no reasonable alternative to hepatic lobectomy because of massive destruction of tissue or the inability of the surgeon to control arterial or venous bleeding by lesser means. There is virtually no place in modern surgery for gauze packing of the liver as sepsis and recurrent bleeding are almost inevitable sequelae. In considering surgical results, the differences between emergency and elective hepatic resection must be borne in mind. The margin of reserve is greatly reduced in the former as about 75 per cent of these patients have suffered hypotension, severe shock, hepatic hypoxia, parenchymal disruption, acidosis, the effect of a massive blood transfusion, and the presence of extrahepatic injuries. Detailed description of the operation may be found in accounts by Quattlebaum *et al.* (1959) and Donovan *et al.* (1968).

Whether debridement or resection is performed, leakage of bile in the post-operative phase is a potentially serious complication, leading in some patients to infection, lysis of thrombi with consequent secondary haemorrhage, and/or bile peritonitis. Identification and ligation of small biliary radicals may be made easier at operation by the instillation through a previously inserted T-tube of a dilute solution of methylene blue while the distal common bile duct is temporarily occluded. As the most meticulous suturing of visible intra-hepatic ductules and extensive drainage does not insure safety, Merendino *et al.* (1963) proposed routine drainage of the extra-hepatic biliary tree by T-tube choledochostomy or cholecystostomy with a view to reducing the pressure in the intra-hepatic biliary channels. While this has gained widespread acceptance, there is as yet little experimental or clinical proof of its efficacy. We have not

discerned any clinical improvement in the end-results of 18 patients who had biliary drainage compared with 76 who did not. Furthermore, among these 18, we have not been able to distinguish any substantial functional difference between cholecystostomy and choledochostomy. It may be significant that in a study presently being conducted in our experimental laboratories, we have not been able to demonstrate reduction of intra-hepatic pressure by controlled external biliary drainage (unpublished). It is a matter of practical importance that, in a number of our patients with shattered livers, the common bile duct has been so narrow at operation that it has been deemed meddlesome and even dangerous to try to insert a **T**-tube. In others, the **T**-tube—with patency checked by cholangiography—has drained less than the sump drainage leading from the region of resection. Finally, in some of our more seriously injured



(*a*) (*b*)

Fig. 4. (*a*) Catheter cholangiogram on the 14th post-operative day following right hepatectomy showing a leak from the biliary duct in the liver. (*b*) Hepatic arteriogram in a patient with haemobilia demonstrating the cavity. Fatal haemorrhage occurred a few hours later while preparations for operation were being made.

patients (again with the **T**-tube demonstrated to be patent) severe jaundice and serum bilirubin levels up to 25 mg. per cent have occurred within a week of operation in the absence of obvious sepsis and have only returned to normal after several weeks. A **T**-tube may have other value, however, serving as a conduit for the injection of an appropriate radiopaque material to demonstrate leakage from intra-hepatic biliary radicles or the presence of an intra-hepatic haematoma or abscess (Fig. 4*a*).

Inasmuch as some leakage of bile is inevitable and the degree not always predictable, we believe that reliable drainage of the peri-hepatic area is vital following resectional debridement or lobectomy, although Perry *et al.* (1967) feel this to be unnecessary. We invariably lead sump drains and numerous soft rubber drains through separate incisions wide enough to allow free egress of bile. Stressing this need, McClelland *et al.* (1964)

advocate the removal of the 12th rib to provide efficient dependent posterior drainage. Drains should be left in place for at least ten days but may need to remain in place for a few weeks where there is substantial drainage.

Complications

The main complications encountered are listed in Table III. While many are non-specific in that they may follow any major injury, some are closely associated with hepatic trauma and merit special consideration.

TABLE III

MAJOR COMPLICATIONS OF LIVER INJURY

- *I. Haemorrhage*
  - A. Pre-operative
  - B. Intra-operative
  - C. Post-operative
    - (*a*) from liver—haemobilia
    - (*b*) stomach—stress ulcer
    - (*c*) haemorrhagic diathesis
- *II. Sepsis*
  - A. Intrahepatic—infarcted residua
  - B. Peri-hepatic
  - C. Pulmonary
  - D. Wound
  - E. Septicaemia
- *III. Pulmonary*
  - A. Ventilation—perfusion defects
  - B. Pneumonia
  - C. Emboli—vascular, fat, hepatic
  - D. Pleural effusions—blood, bile, pus
- *IV. Renal*
  - A. Oliguric renal failure
  - B. Non-oliguric renal failure
- *V. Metabolic*
  - A. Hypo-albuminaemia
  - B. Hypoglycaemia
  - C. Transient jaundice

(*a*) *Haemobilia:* Although haemobilia had been recognized many decades earlier, Sandblom clarified and named the entity in 1948. Since then, at least 31 cases treated by definitive surgery have been described and the incidence appears to be increasing. The passage of blood from the liver to the intestinal tract via the extra-hepatic ducts—often associated with colic, haematemesis or melaena and mild jaundice—may occur within days or be delayed for years following hepatic injury. Unrecognized intra-parenchymal necrosis or haematoma, the suturing of the surface of a bursting injury without adequate deep debridement and obliteration of dead space, or the formation of an intra-hepatic abscess set the stage at the time of injury. While the first episode of haemobilia may be torrential, more often warning is given by previous sporadic appearances of small to moderate quantities of blood in the T-tube or stool. The severity of individual episodes varies. The clinical diagnosis may be confirmed by cholangiography (where a T-tube is in place), by hepatic scan using an appropriate isotope, by splenoportography, by hepatography via the umbilical vein or, more effectively, by selective hepatic

angiography. Even though a number of apparently spontaneous cures are on record, radiographic demonstration of the lesion demands immediate action as massive fatal bleeding may occur suddenly. The surgical procedure adopted will depend on the site and anatomical situation of the source of bleeding. Three alternatives are now available—hepatic lobectomy (partial or total), unroofing and meticulous haemostasis of the walls of the cavity, or ligation of the hepatic artery, proximal or distal to the gastroduodenal artery.

Wilkinson *et al.* (1968) have recently reviewed the 31 cases reported as having surgical treatment. Many more, including one of our own (Fig. 4*b*), have died without operation. While major hepatic resection is sometimes advocated as the standard treatment, the mortality rate with this procedure has been relatively high (4 out of 13), especially in the presence of sepsis. The simplest procedure, especially where sepsis is established, is ligation of the hepatic artery, but surgeons have been deterred from this for fear of inducing hepatic ischaemia and necrosis. It is increasingly apparent from work of Mays (1967) and Andreassen *et al.* (1962) that the normal liver can tolerate ligation of the hepatic artery at any level in most cases and that our fears based on extrapolation from experimental work on dogs and the early work of Graham and Connell (1933) are largely unfounded. We have recently deliberately ligated the right hepatic artery in two patients; in one for a damaged liver to stop bleeding when a formal resection was thought to be unfeasible, and in one for haemobilia due to an intrahepatic abscess. Where anatomically feasible, the safest treatment for haemobilia will often be a combination of hepatic artery ligation, thereby reducing arterial pressure, and the unroofing of the lesion with direct suture of the intramural vessels, followed by efficient drainage. The right or left hepatic artery may be ligated if the lesion is situated in an unequivocally definable anatomical division, but ligation of the proper hepatic artery or common hepatic artery is also acceptably safe. Occasionally, it may be necessary to add a cholecystectomy where the blood supply of the gall bladder is jeopardized. Where ligation of the hepatic artery is adopted, hypotension and shock, with consequent hepatic hypoxia, must be avoided and the oxygen content of the portal blood should be augmented for the first few post-operative days by increasing the blood oxygen tension (Tygstrup *et al.* 1962). In addition, high antibiotic levels are maintained and a cooling blanket is used in an attempt to reduce hepatic metabolism if the patient develops a fever of more than 101° F. Hepatic resection is reserved for those patients who re-bleed.

(*b*) *Pulmonary:* Pulmonary complications frequently occur and are best treated by anticipating their development. Clinical recognition of incipient respiratory insufficiency cannot be relied upon. The early signs, hyperventilation and restlessness, may be extremely subtle and develop into the more florid symptom-complex of pulmonary failure very rapidly,

especially in older patients. In many cases, these are predictable when associated with rib fractures, and haemothorax and pulmonary contusions are present. The problem is compounded by traumatic rupture or surgical incision of the diaphragm as these limit movement in the post-operative period, further predisposing to 'congestive atelectasis'. Finally, embolization of hepatic tissue (Marshall, 1874), fat or free fatty acids, venous thrombi or micro-emboli from the administered blood may seriously disturb pulmonary function. We have studied in detail 21 patients with a combination of hepatic and pulmonary trauma and have noted marked changes in many parameters. These are obviously affected by a great number of variables such as the presence and duration of shock, the quantity of blood transfused and development of sepsis (Table IV).

TABLE IV
ACID-BASE AND BLOOD GAS STUDIES IN 21 SEVERELY ILL PATIENTS WITH LIVER TRAUMA

	Range
pH	7.07–7.56
PO_2 RA	54–67 mm. Hg
PO_2 70% O_2	82–265 mm. Hg
PCO_2 Total	32–53 mm. Hg
A–a D RA	32–62 mm. Hg
' Physiologic ' shunts ..	32–66%

Unless the severe impairment of respiratory function is recognized early, subsequent attempts to maintain adequate oxygenation are often doomed to failure (Wilson *et al.* 1969). For these reasons, assisted ventilation is desirable and there are few patients who do not require tracheostomy at the end of a major hepatic resection or debridement, especially where there is associated lung trauma, prolonged shock or pre-existing pulmonary disease. It is true that some patients will survive without this, but their margin of safety is considerably reduced.

(*c*) *Stress ulceration:* As noted by Foster *et al.* (1968), little attention has been given to this problem. Foster, observing eight patients with massive gastro-intestinal haemorrhage from ' stress ' ulceration in a series of 50 major resections, speculated about the relationship of biliary diversion, fatty metamorphosis of the liver, sepsis and uraemia as aetiological factors. Our experience parallels this. Seven of about 30 patients developed substantial haematemesis following major hepatic debridement or resection, despite continuous gastric suction and the administration of antacids at regular intervals. While ' high-risk ' patients can be defined, we have been unable to predict which will actually bleed. Sporadic measurements of gastric acidity have failed to reveal any secretory abnormality. In five of the seven patients, attempts at non-surgical management such as lavage with ice-water and gastric hypothermia were unsuccessful, but two patients with moderate bleeding subsequently recovered on non-operative treatment. In those who underwent operation, widely scattered multiple superficial erosions were present. As so often

occurs, these were not actively bleeding at the time of gastrotomy. Nevertheless, vagotomy and pyloroplasty has been our treatment of choice. This has been successful in two, but fatal re-bleeding occurred in the three other patients. In one, who did not have a vagotomy, a generalized haemorrhagic diathesis developed (Table V), and in the other two the bleeding was associated with deepening jaundice, sepsis and renal failure. These three patients died on the 15th, 60th and 92nd post-operative days, having received 63, 72 and 140 units of blood, respectively.

(*d*) *Metabolic:* While patients following trauma may survive after

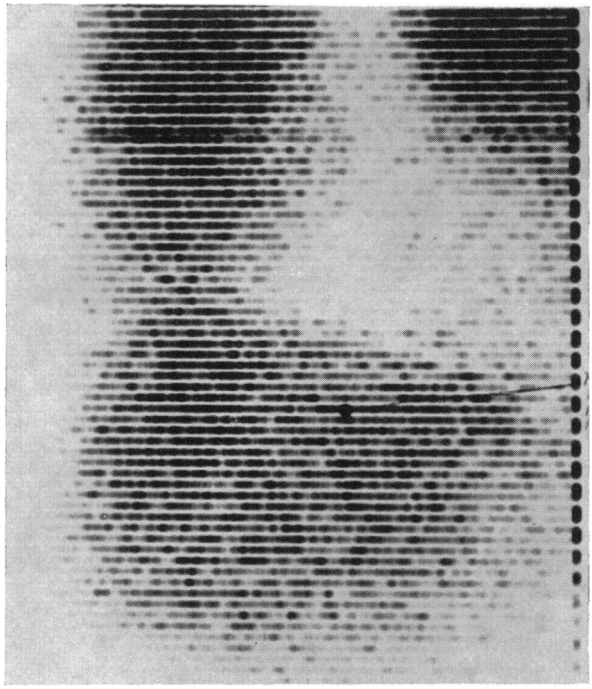


Fig. 5. Liver-lung scan eight weeks after resection of most of the right lobe of the liver, demonstrating its Prometheus-like capacity for regeneration (same patient as in Figure 4*a*).

80 per cent resections and the liver will regenerate completely in about six months, close attention must be given to metabolic support over the first week or two (Fig. 5). Ten per cent glucose is given intravenously during the phase of hypoglycaemia which persists until the patient begins oral feeding. Hypo-albuminaemia is the most persistent abnormality as the liver is the sole site of production of albumin. Many patients require 200 G. of albumin or more over the first week to prevent the development of generalized oedema. Four to six months may elapse before the serum albumin passes into the normal range, reflecting the regenerative capacity

TABLE V

BLOOD CHANGES FOLLOWING EXTENDED RIGHT HEPATECTOMY FOR TRAUMA

Post-operative days	3	4	5	6	7	8	9	10	11	12	13	14	15	
Platelets per cm. × 10^3	163	40	0	20	93	29	15	88	74	0	11	0	5	
Prothrombin U/ml.	46	47	35	36	43	46	51	53	59	45	60	36	48	
Fibrinogen mg. per cent	97	20	13	0	97	103	115	78	130	137	124	100	133	
Factor V per cent of control	43	11	11	0	45	82	52	43	60	56	61	72	42	
Profibrinolysin per cent of control ..	42	48	27	14	82	97	71	102	105	44	44	44	62	
														*Total**
Fresh blood						2	2	6		1		6	2	19
Banked blood (23 units plus)				3	1			5		4	1	7		44
Fresh frozen plasma		2		2	2		2	6	2	2	2	3	1	24
Fibrinogen					1	1	1	4	2	2	1		1	13
Albumin	4	4	3		1	2	3	9	2	3	8	6		45
Platelets					2			2				1	3	8

* Units

of the liver. Serum alkaline phosphatase, transaminase, and often bilirubin levels may be high for two or three weeks in the absence of overt sepsis and may reflect the swelling of the regenerating hepatic cells.

There are also falls in the prothrombin, fibrinogen, and Factor V levels reflecting in part the reduction in hepatic manufacture of these but also the composition of the large quantities of banked blood which these patients almost invariably receive. The normal liver has the function of clearing the products of platelet and fibrin breakdown, neutralizing circulating plasminogen activators and filtering pro-coagulants from the portal system. We have encountered three patients with a generalized bleeding diathesis after partial hepatectomy; in two, the haemorrhage appeared to be due to a 'consumption coagulopathy' rather than a 'wash-out' effect or inadequate production of blood factors. Pure fibrinolysis was not a feature of any of our patients. A comparison of the coagulation changes in six patients who had major liver resections and massive transfusions of banked blood with the changes in the group of 108 patients receiving massive transfusions for other reasons reveals no significant differences (Table VI). The third of these patients, a 19-year-old girl, who had 1,100 G. of liver resected for an isolated injury, died of generalized bleeding on the 15th post-operative day after receiving 19 units fresh blood, 44 units banked blood, 24 units fresh frozen plasma, 13 units of fibrinogen, 45 units of albumin, and 8 units of platelet concentrates. She was studied exhaustively and appeared to have developed an anti-platelet antibody which caused a persistent thrombocytopaenia (Table V). At autopsy, the remaining liver showed considerable fatty metamorphosis, most marked in the central zones, prominent evidence of regenerative activity, and obvious signs of biliary stasis. The entire gastro-intestinal mucosa was friable and haemorrhagic with definite ulcerations confined to the terminal ileum, caecum and ascending colon. The kidneys showed the microscopic appearance of acute tubular necrosis.

SUMMARY

Five hundred consecutive patients surgically treated for hepatic injury have been reported. The mortality rate was 1.9 per cent for 270 stab wounds, 17.9 per cent for 173 gunshot wounds and 31.8 per cent for 57 closed injuries. The clinical picture may be deceptive, especially in the presence of associated injuries which occur in the majority of these patients.

The general principles of management are modified by basic anatomical and physiological factors unique to the liver. Certain specific technical problems relating to haemorrhage and biliary drainage can be anticipated and require predetermined alternative plans of action. The commoner complications of haemorrhage, sepsis, respiratory failure, acute tubular necrosis, stress ulceration and haemobilia can be reduced by attention to detail at the time of operation and in the immediate post-operative

TABLE VI

COMPARISON OF COAGULATION CHANGES BETWEEN PATIENTS RECEIVING MASSIVE TRANSFUSION

FACTORS		PTT	PT	PU	V	VII	FIB	AT	TT
Units of Blood									
8	Hepatectomy	80.4	+ 4.3	100	- 15.6	56.5	115.5	38	+ 0.3
	Non-hepatectomy	105.8	+ 6.6	123.4	- 7.3	68.1	152.3	23.1	+ 3.4
12	Hepatectomy	85.8	+ 3.5	102	- 16.6	81.5	114	39	+ 3.8
	Non-hepatectomy	114.9	+ 5.7	113.4	- 7.6	64.7	149.7	23.5	+ 8.3
16	Hepatectomy	82	+14.5	121.2	- 13.6	60.6	143.2	23.5	+ 0.9
	Non-hepatectomy	147.2	+ 6.8	114.9	- 9.5	76.7	166.5	20.3	+ 5.6
20	Hepatectomy	63	+ 1.8	119.5	- 14.2	83	176.5	37.5	+ 7.5
	Non-hepatectomy	166.4	+ 6.7	102.4	- 9.0	53.1	131.7	22.6	+10.9
>20	Hepatectomy	88.9	+ 7.7	121	- 9.6	94	189.5	25	+ 8.9
	Non-hepatectomy	152.8	+ 6.3	106.1	- 9.0	53.1	192.8	22.3	+ 5.6
		NV 50–70 secs. C H=13.3 N/H=13.5		NV 250±50 μ/ml.	C H=18.8 N/H=16.2	NV 60–200%	NV 200–300 mg.%	NV 40–60%	C H=16.4 N/H=15.7

Key NV = Normal values
C = Control
H = Hepatectomy
N/H = Non-hepatectomy

phase. Many victims are in the prime of life and can be saved if the details of treatment are understood and vigorously applied.

ACKNOWLEDGEMENTS

Acute trauma to the liver demands emergency treatment often at the most inconvenient times. A great many people have contributed to the care of these patients and to the shaping of our present procedures. First among these over the years have been the Wayne State University surgical residents at Detroit Receiving Hospital. Among others, I would like to express special thanks to my colleagues, Drs. Robert Wilson, Thomas Grifka, and Charles Lucas and Miss Betty Gutman of the Department of Surgery; Dr. Eberhard Mammen and Dr. Marta Ross for assistance with the coagulation data; and Miss Lucy Brand, R.N., and her nurses in the intensive care unit, whose efforts have so improved the prognosis for many of our patients.

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PROCEEDINGS OF THE COUNCIL IN NOVEMBER

AT A MEETING of the Council on 13th November 1969, with Sir Thomas Holmes Sellors, President, in the Chair, the appointment of Professor Sir Hedley Atkins, K.B.E., F.R.C.S., as the Hunterian Orator for 1971 was reported.

Dr. O. T. Clagett was admitted to the Honorary Fellowship of the College.

Mr. Norman Barrett, C.B.E., F.R.C.S., was appointed the Thomas Vicary Lecturer for 1970. The appointment of Dr. C. F. Scurr, M.V.O., F.F.A.R.C.S., as Frederic Hewitt Lecturer for 1971 was reported.

Mr. I. A. Tumarkin, F.R.C.S.Ed., of Liverpool, was appointed the Joseph Toynbee Memorial Lecturer for 1970.

Mr. S. H. Harrison, F.R.C.S.Ed., was admitted to the Fellowship *ad eundem.*

The award of the Gilbert Blane Medal for 1969, jointly with the Royal College of Physicians, to Surgeon Commander Norman James Blacklock, F.R.C.S., of Gosport, Hants., was reported.

The Begley Prize was awarded to Gerald Courtenay Davies of Westminster Medical School.

The McNeil Love Medal was presented to Mr. Edward John Noon (Department of Anatomy).

The death of Mr. T. G. Wilson (Honorary Fellow) was reported with deep regret.

A Diploma of Fellowship was granted to W. B. Coman.

Diplomas of Membership were granted to 145 candidates.

Licences in Dental Surgery were granted to 80 candidates.

Diplomas in Orthodontics were granted to 21 candidates.

A Diploma in Dental Public Health was granted to J. D. Leadbeater.

The following Diplomas were granted, jointly with the Royal College of Physicians:

Child Health (108), *Medical Radio-Diagnosis* (56).