Treating major liver injuries

Trauma is the leading cause of death in young adults, and is surpassed only by cancer and cardiovascular disease as the major cause of death in all age groups in Western countries. Twenty per cent of major injuries involve the abdomen, and failure to recognise and correctly treat serious abdominal injuries is an important cause of preventable death. The liver is the most frequently injured solid intra-abdominal organ. The spectrum of liver injuries ranges from minor lacerations with minimal bleeding that stop spontaneously and require no intervention, to major lobar crush injuries and lacerations of the retrohepatic vena cava and hepatic veins that are often lethal and tax the skill, resolve and resources of the most experienced surgical teams. Overall mortality in liver injuries ranges from 10% to 42% and is largely dependent on the type of injury and the extent of associated injuries to other organs. Less than 10% of penetrating civilian wounds are lethal, whereas overall mortality after blunt trauma exceeds 40%. The mortality in blunt hepatic injury is 10% when only the liver is injured, but if three major organs are injured, mortality approaches 70%. Bleeding causes more than half of the deaths. The magnitude of the liver injury and the complexity of the operative intervention required are determined by the cause and vector of force, the mechanism of injury, the anatomical location and the extent of parenchymal and vascular damage. Stellate, burst-type injuries, which typically occur in high-speed car accidents, tend to affect the posterior segments of the right liver lobe because of their relatively fixed position. Deceleration injuries result in shearing stresses which tear the hepatic veins between the posterior sector (segments 6 and 7) and anterior sector (segments 5 and 8) of the right lobe, producing a major retrohepatic injury in an area difficult to expose and repair surgically. High-velocity projectiles and close-range shotgun injuries cause marked fragmentation of the hepatic parenchyma with laceration of vessels and major intraperitoneal bleeding.

During the past two decades the management of liver trauma has changed substantially. Non-operative treatment in selected patients has become the standard of care. Patients who respond to fluid resuscitation and remain stable are carefully monitored for signs of bleeding or abdominal injury while the extent of liver injury is assessed by computed tomography (CT). Current guidelines indicate that after blunt liver injury, patients who remain haemodynamically stable after resuscitation without persistent or worsening abdominal pain, have no other intra-abdominal injuries that require laparotomy, require less than a 4-unit blood transfusion and have a haemoglobin level less than 500 ml on CT scan can safely be monitored with anticipated success rates up to 85% and complication rates as low as 6%. Patients with increasing abdominal distension and tenderness who remain shocked after effective resuscitation usually have continued bleeding and need an urgent laparotomy. Surgery should not be delayed to obtain the results of special investigations. The surgeon who encounters a major liver injury during a laparotomy for trauma will rapidly need to make several critical decisions. The principles of management and the surgeon’s role in achieving the objectives and priorities are self-evident and clear: stop the bleeding by the simplest means possible, remove devitalised liver tissue, and suture or repair damaged blood vessels and bile ducts. Fortunately most liver injuries are simple and can be treated without difficulty. The few complex lesions need to be diagnosed early and may require major surgery by experienced surgeons. The dilemma is that many general surgeons working outside regional trauma centres or specialist hepatobiliary units seldom deal with major liver injuries and may have limited resources. The surgeon at the coalface has a variety of choices and options at his disposal to control liver bleeding. These range from simple to increasingly demanding techniques, depending on the magnitude of the liver injury: the choices in increasing order of complexity include controlling major arterial beam coagulation, topical agents such as fibrin glue or Surgicel (for superficial injuries), temporary inflow occlusion of the portal triad (Pringle manoeuvre) using either fingers, a sling or a vascular clamp, liver packing, hepatorraphy, hepatotomy and direct suture of the bleeding vessel, or ligation and division of the hepatic artery, portal vein and bile ducts. Recently, endovascular venous stenting and embolisation have been used ad hoc in situations where conventional techniques have failed. These procedures include portal ligation, ligation of the hepatic artery, atriocaval shunting (‘more authors than survivors’) and median sternotomy and thoracotomy for proximal exposure are now obsolete. The level of intervention will ultimately be influenced by the surgeon’s experience, local resources and facilities available. The key is to use the most effective yet simplest option for each specific situation. Fortunately, most liver injuries will have stopped bleeding spontaneously by the time the operation is performed. These wounds do not require suturing but a drain should be placed to prevent bile collections.

Active liver bleeding can usually be controlled by compressing the liver wound with abdominal packs. If bleeding is massive and the patient is shocked, experienced surgical assistance and additional anaesthetic help should be summoned urgently. Initial management is critical. The greatest immediate threat to survival is exsanguination. The absolute priority is rapid control of bleeding. The injury is manually compressed with abdominal packs and tamponade maintained with packs and pressure. This may be supplemented by inflow occlusion (Pringle manoeuvre) using anatraumatic vascular clamp across the portal triad. Effective intraoperative resuscitation is crucial. Pack pressure is maintained until the anaesthetist has fully restored intravascular volume. Premature attempts to evaluate the extent of the injury by mobilising the liver before adequate intravascular volume replacement and effective resuscitation may lead to catastrophic blood loss with ensuing hypotension, coagulopathy, acidosis, hypothermia and unnecessary death. These potential consequences should be anticipated and countered by providing warm intravenous solutions, the freshest possible blood, fresh-frozen plasma, platelets, cryoprecipitate, devitalised tissue, and total hepatic vascular isolation with veno-venous bypass. Innovative methods of haemostasis including mesh wrapping, balloon tamponade, enclosure in a liver bag and endovascular venous stenting have been used ad hoc in situations where conventional techniques have failed. These procedures include hepatic artery ligation, atriocaval shunting (‘more authors than survivors’) and median sternotomy and thoracotomy for proximal exposure are now obsolete. The level of intervention will ultimately be influenced by the surgeon’s experience, local resources and facilities available. The key is to use the most effective yet simplest option for each specific situation. Fortunately, most liver injuries will have stopped bleeding spontaneously by the time the operation is performed. These wounds do not require suturing but a drain should be placed to prevent bile collections.

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bleeding, the hepatic artery and portal vein should be temporarily clamped to provide inflow occlusion and allow accurate identification of the bleeding site. If bleeding persists, adequate exposure and a clear view of the injury become essential. Intermittent inflow release and effective suction allow identification of deeper bleeding sites, which are controlled by either direct suture, ligation, parenchymal suture or a mattress liver suture. If bleeding cannot be controlled by sutures and experienced assistance is not available, the injury should be packed, the abdomen closed and definitive treatment deferred until a surgeon who can perform the required surgery is available or the patient can be transferred to a major trauma or hepatobiliary centre.

Even in referral centres under optimal conditions, if bleeding continues after attempting the above strategy, experience and mature judgement become crucial in the decision whether to advance to the next level of intervention. Extending a liver fracture to improve exposure and define the bleeding site can incur daunting haemorrhage and morbidity, which may exceed the anticipated benefit. If bleeding is not controlled by infrahepatic or portal triad occlusion, a major vena caval or hepatic venous injury or an accessory left hepatic artery is likely. Posterior right lobe injuries are relatively inaccessible and require extension of the abdominal incision, fixed subcostal retraction to aid exposure and full mobilisation of the right lobe to provide access to the IVC and right hepatic vein, which can be digitally controlled. Repair of complex venous injuries may ultimately require total hepatic vascular isolation.

Non-anatomical resection is used to remove a partially attached or ischaemic liver segment peripheral to a fracture line, which in effect results in completion of a de facto traumatic resection. Resectional debridement implies removal of non-viable liver bordering an injury. Anatomical resection performed through conventional anatomical planes unrelated to the lines of fracture is seldom necessary and is justified only in severe liver injuries in which the lobe is shattered and conservative measures fail because of parenchymal devascularisation. Such a formal anatomical lobar resection in an unstable coagulopathic patient becomes a formidable and hazardous undertaking and is neither expedient nor recommended for the occasional liver surgeon.

Juxtahepatic venous injuries involving the retrohepatic vena cava or major hepatic veins represent a small proportion of all liver injuries but constitute the most lethal category of liver trauma with a reported mortality of 50 - 80%. Technical difficulties are compounded by the inaccessible position of the major hepatic veins and retrohepatic vena cava within the thickest and least mobile portion of the liver. Most deaths result from rapid intraoperative exsanguination because of failure to control initial bleeding or severe blood loss during exposure and venous repair. Vigorous rotation during mobilisation of the liver to expose the veins aggravates the bleeding and increases the risk of fatal air embolism.

For the surgeon encountering a major liver injury in a peripheral hospital, perihepatic packing should be the first option when lesser procedures do not control bleeding. If packing controls the bleeding, no further intervention is necessary. The packs are left in place, the abdomen is closed and definitive treatment is deferred until a surgeon who can perform the required surgery is available or the patient can be transferred to a major trauma or hepatobiliary centre. Even for the experienced surgeon, if bleeding is controlled, discretion and packing may be the better part of valour. Other factors also influence the decision to pack. If the patient is acidic (pH < 7.2), hypothermic (body core temperature < 32°C) or coagulopathic or has had a massive transfusion (> 10 units of blood), the liver should be packed, the abdomen closed and the patient returned to the intensive care unit. Blood volume is restored, the patient warmed and the acidosis and coagulation defects corrected, and the packs are removed under general anaesthesia in the operating theatre 2 - 3 days later.

The original concept and technique of packing for the treatment of major liver injuries was introduced by Halsted in 1913 and became standard practice for the next three decades. A pack was thrust into a liver laceration to provide haemostasis and the end of the pack brought out through a drain site in the abdominal wall. The pack could then be removed several days later by careful withdrawal via the drain site to avoid a further laparotomy and re-exploration. This form of treatment for liver injuries remained the mainstay throughout World War II. However, recurrent haemorrhage often occurred after pack removal and infection was a frequent complication as gauze packs were ineffective drains. These complications led to the abandonment of intrahepatic packing for liver injuries. The re-introduction of perihepatic liver packing in a small series of liver injuries from England led to renewed interest and subsequent validation of the technique in larger series. The use of the modified and refined liver packing technique has now markedly simplified the treatment of major liver trauma.

The technique of perihepatic packing is important. Sufficient packs should be used to provide effective uniform pressure and generally a ‘six pack’ is adequate. An attempt should be made to restore the liver contour to normality by closing the defect and to provide uniform and consistent tamponade by external pressure with dry gauze packs placed above and below the liver. Too many packs may cause increased intra-abdominal pressure, abdominal compartment syndrome, caval compression and acute renal failure. Intra-abdominal pressure should not exceed the critical pressure limit of 25 mmHg. An important practical point is to avoid intrahepatic packing because packs forced into deep liver fractures may aggravate the injury by increasing the size of the rent and holding it open, as well as tearing small hepatic veins. Sepsis should be anticipated if there is a bile leak or bowel contamination and intravenous antibiotics should be given while packs are in situ. Packs are removed in the operating theatre after 36 - 72 hours, when metabolic and coagulation abnormalities have been corrected. The presence of packs combined with oedema of the bowel may lead to difficulties in wound closure. If this is encountered, mesh can be inserted to prevent further compromise of ventilation and bowel viability and to avoid pressure necrosis of the liver.

Rebleeding from the injury site, bile leaks, infected fluid collections and peripheral segmental liver ischaemia are the main postoperative complications associated with liver trauma. If major venous liver bleeding continues, coagulation abnormalities should be corrected and the abdomen re-explored and visible vessels sutured. The management of complex liver bleeding has been simplified by angiographic intervention. Superselective hepatic artery catheterisation and embolisation has the advantage of occluding only the source of bleeding, while preserving blood flow to normal parenchyma, in contrast to surgical ligation where technical considerations dictate more proximal ligation. As the liver has a dual blood supply, hepatic artery embolisation is well tolerated provided the portal vein is normal and there is no hepatoglufl flow as in cirrhosis. Portal vein patency must be established prior to embolisation. Selective catheterisation distal to the cystic artery avoids the risk of gallbladder infarction. Computed tomography is used to identify intra-abdominal fluid collections which are best drained by ultrasound-guided needle aspiration or a percutaneous catheter. Subhepatic sepsis develops in about a fifth of cases and is usually related to bile leaks, ischaemic tissue, undrained
collections or undetected bowel injury. The sites of bile leaks are best identified by ultrasonographic, angiographic and interventional endoscopic techniques to drain septic collections, embolise intrahepatic bleeding and stent bile leaks.

J. E. J. Krige
A. J. Nicol

Surgical Gastroenterology and Trauma Units
Department of Surgery and MRC Liver Research Centre
University of Cape Town and Groote Schuur Hospital
Cape Town

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