Eur J Trauma Emerg Surg (2015) 41:229–237
DOI 10.1007/s00068-015-0521-0

REVIEW ARTICLE

Management of blunt liver injury: what is new?

J. Ward[1] · L. Alarcon[1] · A. B. Peitzman[1]

Received: 29 October 2014 / Accepted: 11 March 2015 / Published online: 23 April 2015

Abstract Nonoperative management has become the surgical treatment of choice in the hemodynamically stable patient with blunt hepatic trauma. The increased use and success of nonoperative management have been facilitated by the development of increasingly higher resolution computed tomography imaging, improved management of physiology and resuscitation (damage control), and routine availability of interventional procedures such as angiography and embolization, image-guided percutaneous drainage, and endoscopy. On the other hand, recognition of the patient who should proceed to immediate laparotomy is of utmost importance. A systematic and logical approach to the control of hemorrhage is required in the operating room. Thorough knowledge of the anatomy and surgical techniques, such as perihepatic packing, effective Pringle maneuver, hepatic mobilization, infrahepatic and suprahepatic control of the IVC, and stapled hepatectomy, is essential.

Keywords Blunt hepatic injury · Nonoperative management · Liver resection · Angiography/embolization

Introduction

The liver is the most commonly injured organ in patients suffering blunt abdominal trauma [1]. Over the past three decades, nonoperative management has become the primary treatment modality for the vast majority of patients, with significant improvements in outcome [2–4]. Patients with hepatic injury who are hemodynamically unstable at presentation require immediate operative control of major bleeding. Approximately a quarter of patients with blunt hepatic injury managed nonoperatively will manifest complications of major liver injury mandating intervention, infrequently operative [5–7]. The focus of this paper is review of current management of blunt hepatic injury, focusing on the past 10 years, as well as key references from earlier literature.

Initial assessment

As with any victim of blunt trauma, the patient with peritonitis or hemodynamic instability and signs of abdominal trauma should undergo prompt laparotomy. For hemodynamically stable patients, computed tomography (CT) of the abdomen and pelvis is the diagnostic test of choice [8–12]. This is a critical decision point in the patient with blunt hepatic injury. In general, any patient with blunt hepatic injury stable enough for CT (assuming appropriate patient selection) will have the liver injury managed nonoperatively [13]. CT allows rapid evaluation of the hepatic injury, assessment of the extent of injury, grading of the injury, determination of the volume of hemoperitoneum, and detection of active hemorrhage (Table 1). The successful nonoperative management of blunt liver injury is highly dependent upon this diagnostic modality [14–16]. CT is also useful to detect associated injuries. In patients with liver injury, concurrent injuries affect the spleen in 21 % of patients, kidney in 9 %, and bowel in 4 % [17]. Additionally, in patients with blunt abdominal injury, the incidence of hollow viscus injury increases as the number of solid organ injuries increases [18]. Finally, the initial

✉ A. B. Peitzman
peitzmanab@upmc.edu

[1] Department of Surgery, University of Pittsburgh, F-1281, UPMC-Presbyterian, 200 Lothrop Street, Pittsburgh, PA 15213, USA

Table 1 American Association for the Surgery of Trauma Organ Injury Scale, liver [14]

Grade	Type of injury	Description of injury
I	Hematoma	Subcapsular, <10 % surface area
	Laceration	Capsular tear; <1 cm parenchymal depth
II	Hematoma	Subcapsular, 10–50 % surface area Intraparenchymal, <10 cm diameter
	Laceration	Capsular tear, 1–3 cm parenchymal depth, <10 cm length
III	Hematoma	Subcapsular, >50 % surface area Intraparenchymal, >10 cm diameter or expanding Ruptured subcapsular or intraparenchymal hematoma
	Laceration	>3 cm parenchymal depth
IV	Laceration	Parenchymal disruption involving 25–75 % hepatic lobe 1–3 Couinaud's segments within a single lobe
V	Laceration	Parenchymal disruption involving >75 % of hepatic lobe >3 Couinaud's segments within a single lobe
	Vascular	Juxtahepatic venous injury
V	Vascular	Hepatic avulsion

Advance one grade for multiple liver injuries, up to grade III

laboratory studies should include serum aminotransferases. Elevated serum ALT, AST, LDH and GGT have each been associated with liver injury, and the degree of elevation of ALT, AST and LDH have been shown to be associated with the grade of injury, and ALT as the best laboratory screening test [19–21]. While most patients with blunt abdominal trauma will be evaluated with CT, serum aminotransferase levels may be useful screening tests for those who do not undergo immediate exploration or imaging.

Nonoperative management

Nonoperative management of hepatic injury is preferred for hemodynamically stable patients without peritonitis (Fig. 1) [22–24]. The reported success rates for nonoperative management of liver injury are generally greater than 85 % [22, 25–31]. Approximately 70–80 % of liver injuries can be safely managed nonoperatively; even in most of the severe injuries, the nonoperative management rate approaches 50 % [32–34]. The corollary is that 80–85 % of blunt liver injuries are grades I–III and only 15 % are high-grade injury (grade IV–V). Furthermore, the patient requiring immediate laparotomy for hemodynamic generally has a grade IV or V liver injury. Thus, trauma surgeons operate on blunt liver injury infrequently, but in such cases, the operations are often technically challenging. A recent review of the National Trauma Databank (NTDB) found that only 13.7 % of liver injuries are managed operatively [35]. Another review focused on 3627 grade IV and V blunt liver injuries from the NTDB and found that 7 % failed nonoperative management, with higher associated mortality. Predictors of failed nonoperative management of high-grade liver injury included age (OR 1.02), male gender (OR 1.73), higher ISS (OR 1.02), lower GCS, and hypotension (OR 2.07) [36]. Successful nonoperative management requires careful patient selection based on the hemodynamic status of the patient, absence of other signs of visceral injury that require surgery, good-quality CT imaging, and the availability of an effective multidisciplinary team with intensive care physicians, experienced surgeons, and interventional radiologists at the ready. While many factors help predict nonoperative management failure, it is now generally accepted that the most important factor determining successful nonoperative management is the hemodynamic stability of the patient, irrespective of the grade of injury or the volume of hemoperitoneum [3, 8, 37]. Approximately one quarter of patients with hepatic injury managed nonoperatively will require an intervention to manage a complication [29, 38]. Patients with higher grade injuries are at risk of complications, and higher grade injury alone has been shown to independently predict the need for surgical intervention [5, 22]. Isolated low-grade injuries will uncommonly require surgical intervention for the management of the liver injury itself and may not require ICU admission. Additional risk factors for nonoperative failure or the need for surgical or other invasive interventions have been identified, including active extravasation of contrast on CT, intraperitoneal extravasation of contrast, hemoperitoneum in six compartments, transfusion requirements, volume of resuscitation, and concomitant injuries [5, 30, 33, 39, 40]. As such, patients with these risk factors should generally be observed in the intensive care unit, and those with active extravasation should undergo early hepatic angiography and embolization. Additionally, while hepatic hemorrhage is the major concern for nonoperative failure and need for intervention, surgical intervention is infrequently needed. Indeed, for most patients with liver injury, the most common indication for surgical intervention is the presence of associated injury to the spleen or kidney [34]. Furthermore, for most liver injuries requiring surgery, the indication is related to the development of a complication, such as abscess or bile peritonitis, rather than bleeding [5, 33]. As bleeding complications generally present early in the course, and inflammatory and biliary complications present late, prolonged surveillance in the ICU for hemorrhage is unnecessary [5, 28, 33]. Observation should include physical examination, hemodynamic monitoring, serial hemoglobin measurements, and serial liver function tests. For patients who have evidence of ongoing bleeding from the hepatic injury, either angiography or surgery is warranted, depending on the patient's hemodynamic status,

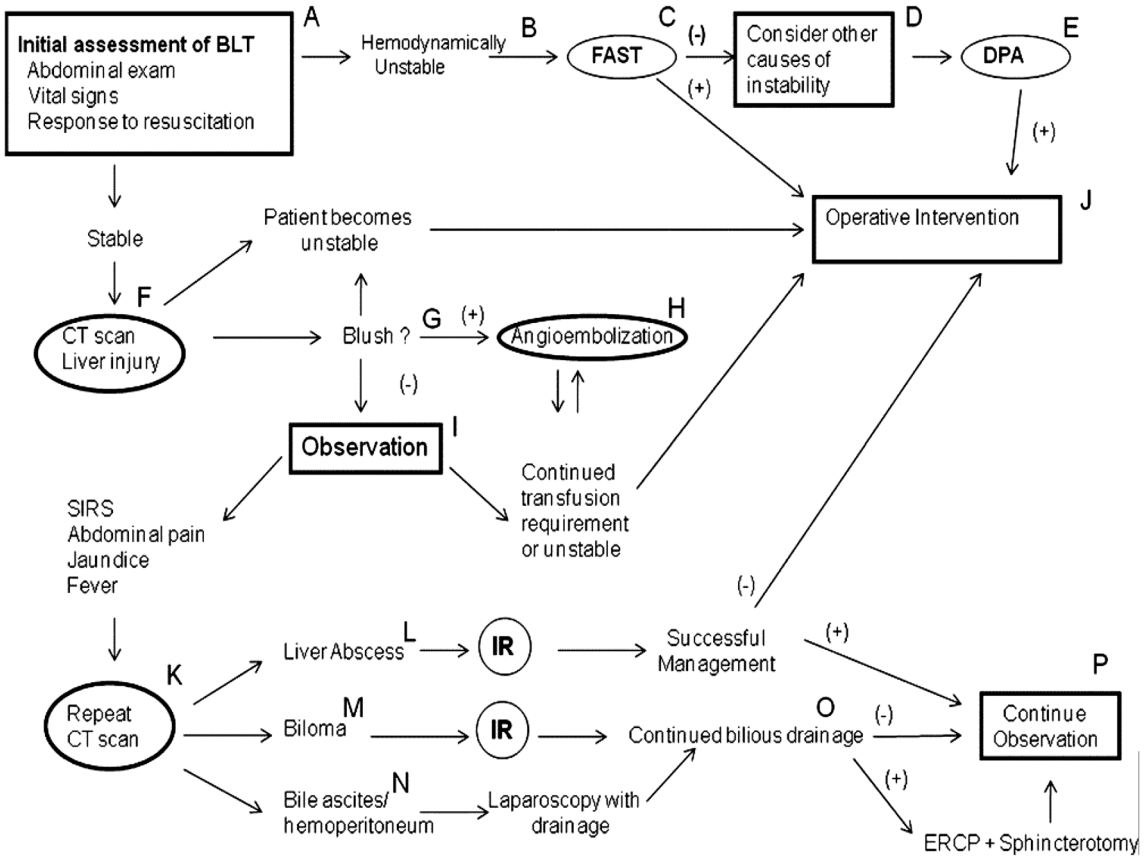

Fig. 1 Algorithm for the nonoperative management of blunt abdominal injury (from [13], Figure 1). © Wolters Kluwer Health, Inc

magnitude of liver injury, and associated intra-abdominal injuries. Those patients who develop delayed complications may require multimodality therapy. With appropriate patient selection, nonoperative management is shown to be associated with high success rates, reduction in blood transfusion requirements, and reduced length of stay [34, 41].

Angiography

Angiography/embolization is a useful adjunct and enhances the success of nonoperative management [37, 42]. Patients who are hemodynamically stable and have active extravasation of intravenous contrast on CT are appropriate candidates for angiography. While contrast extravasation into the peritoneal cavity has been shown to be predictive of nonoperative failure, early and aggressive use of angiography/embolization may reduce the need for early operative intervention in the hemodynamically stable patient [40, 43]. Active extravasation of contrast on CT predicts the need for embolization, with embolization rates of 60–80 % at angiography [44, 45]. In comparison with those without extravasation undergoing hepatic angiography for other indications, such as grade of injury, patients with active extravasation were 20 times more likely to have a positive finding during hepatic angiography [45]. In addition, angioembolization is a useful adjunct in the management of the patient undergoing damage control for liver injury. On the other hand, angioembolization has its own risks. Hepatic necrosis, gallbladder necrosis, bile leak, and abscess can occur after embolization, with complication rates ranging from 29 to 80 % [44–46]. While nonoperative management of isolated hepatic necrosis after embolization has been described, 26 % of patients who develop complications after embolization will require surgical intervention [44–49]. Furthermore, hepatic lobectomy for the management of hepatic necrosis after angioembolization may result in reduced morbidity and fewer procedures when compared to multimodality therapy (Fig. 2) [48].

Complications of nonoperative management

While nonoperative management has resulted in improved mortality, complications of liver injury are increasingly recognized, with complication rates ranging from 12 to 24 %

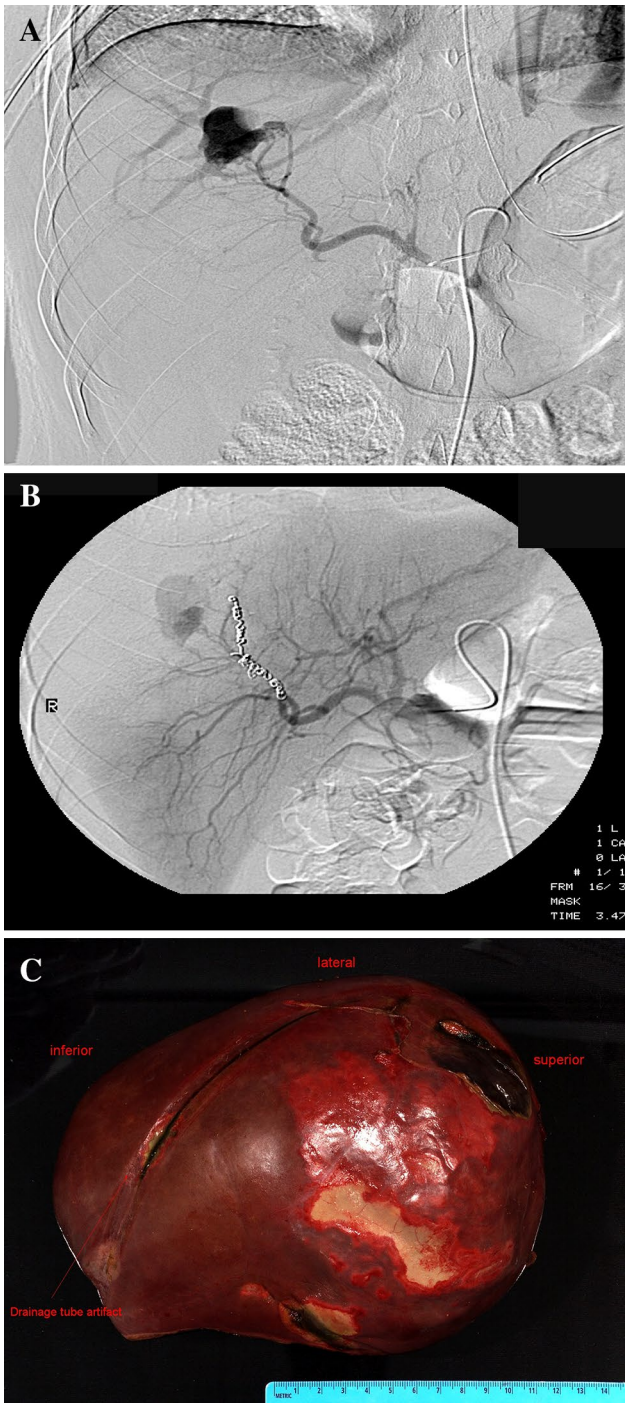

Fig. 2 **a** Angiogram following hepatic injury showing active extravasation from the right hepatic artery. **b** Two days postinjury and embolization with multiple coils, a recurrent bleed is seen. **c** Right hepatic lobectomy was performed for the recurrent bleeding. Hepatic necrosis is apparent from the previous embolization

[5, 37, 38]. These include bleeding, bile leak, hepatic necrosis, gallbladder necrosis, abscess, fistula, or thrombosis hepatic vasculature [5, 46]. The development of a complication can often be anticipated and recognized based upon changes in vital signs, physical examination, and laboratory studies. Imaging is critical in the diagnosis given the multiple therapeutic modalities available to aid in management. CT is highly effective in identifying hepatic complications and is indicated for patients who develop tachycardia, fever, leukocytosis, or changes in liver function tests, while HIDA is a useful adjunct for the diagnosis of bile leak [16, 50]. Higher grade injury and transfusion requirements have been identified as risk factors for subsequent complications, while others have shown that central injury to the liver and the use of hepatic angioembolization to be independent risk factors for bile leak in patients managed nonoperatively, and angioembolization and high-grade injury are associated with hepatic necrosis [5, 50, 51]. A small biloma may resolve without any intervention, while percutaneous drainage is often effective for a larger biloma, and endoscopic retrograde cholangiopancreatography (ERCP) and stenting is an effective primary management strategy or adjunct for major bile leaks [33, 52, 53]. Surgical management is generally reserved for patients with bile peritonitis, those who fail endoscopic therapy, or patients with bile leaks in the presence of other complications, such as persistent inflammation or abscess [5, 33, 52]. Increasingly, laparoscopy is utilized in the management of bile peritonitis [5, 54–56]. Drains may be placed, and ERCP may be used as secondary therapy to promote internal biliary decompression, while patients with extensive injury and associated necrosis or abscess may require resection. Hepatic necrosis may occur from devascularizing injuries, as sequelae from angioembolization, or as a result of large subcapsular hematoma. As previously discussed, nonoperative management of hepatic necrosis has been described; however, many patients will require multiple procedures or surgical intervention, especially when the necrosis is complicated by concomitant bile leak or abscess. In the case of large subcapsular hematoma, laparoscopic decompression may improve portal flow, reverse ischemia, and avoid thrombotic complications [57]. Delayed bleeding is a recognized but uncommon complication and can occur as intra-abdominal or gastrointestinal hemorrhage (Fig. 3). The former is often related to inflammatory or infectious hepatic complications and the development of pseudoaneurysm. Angioembolization may be useful to control delayed or recurrent hemorrhage; however, definitive surgical intervention may be required [5, 33]. With delayed presentation of gastrointestinal hemorrhage after liver injury, hemobilia should be suspected. Symptoms of hemobilia include abdominal pain, melena or hematemesis, and jaundice. However, the classic Quincke's triad is present in fewer than 35 % of cases [58, 59]. While contrast-enhanced CT may be useful, it is not always diagnostic. Angiography remains the best diagnostic test, and super-selective angioembolization is highly successful, with few patients requiring surgery for embolization failure or hemodynamic instability [58, 60].

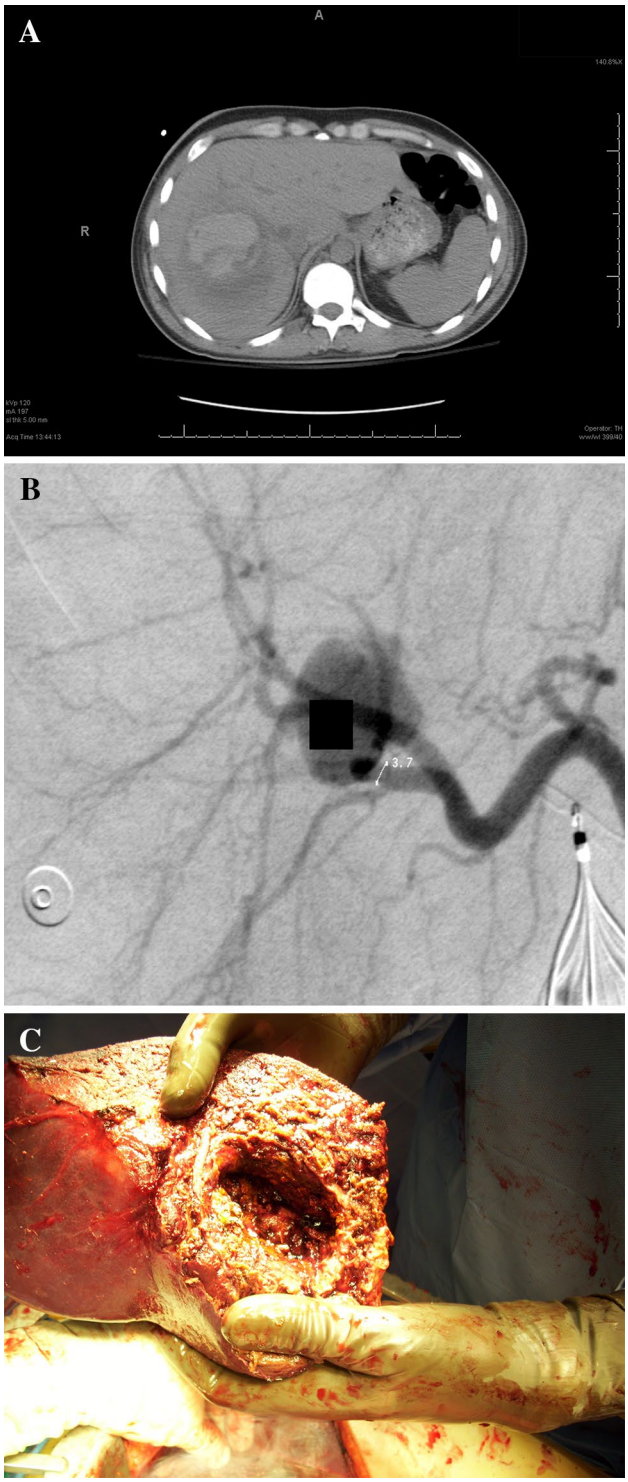

Fig. 3 **a** 18 days after a major blunt right lobe injury and initially negative arteriogram, the patient develops acute abdominal pain and hypotension. A large hematoma is seen within the liver injury. **b** Despite the initially normal hepatic angiogram, a large pseudoaneurysm is now seen. Cultures from this grew fungus. **c** Right hepatic lobectomy was performed. The large infected pseudoaneurysm is evident

Operative management

While nonoperative management of liver injury offers benefits of low mortality, reduced transfusion requirement, and reduced length of stay, and the vast majority of patients with liver injury can be managed nonoperatively, up to 35 % of patients with complex liver injury will require early operation [5, 32, 33]. Operative management of hepatic injury is utilized as primary therapy in patients who present with peritonitis or hemodynamic instability and signs of abdominal trauma or hemoperitoneum. In these patients, the liver injury is often diagnosed intraoperatively. Minor hepatic injuries will often require little, if any, surgical intervention. Should bleeding from these injuries be present, control the hemorrhage with perihepatic packing and allow exploration to continue. The technique of packing is critically important. Initially compress both lobes of the liver back to normal anatomy and push the liver posteriorly to tamponade any hepatic vein bleeding. This maneuver will generally slow bleeding enough that anesthesia can catch up with resuscitation. If packing controls bleeding in the unstable patient, truncate the procedure and complete damage control. Do not place packs into the liver injury as this will widen the injury and worsen the bleeding. Packs (dry, folded laparotomy pads) should be placed over the anterior surface of the liver, posterior–inferior surface of the liver, and the lateral side of the liver. Although debatable, we always take down falciform ligament but generally do not divide coronary or triangular ligaments to pack the liver. The concern is that tamponade may be lost with retrohepatic injury. Avoid packing so tightly that the inferior vena cava is compressed and venous return is impaired. In those patients managed with damage control surgery, perihepatic packing should be left in place and removed at the subsequent surgery. For hemorrhage from the exposed parenchyma or in patients for whom a definitive operation is pursued, electrocautery, argon beam coagulation, and bipolar hemostatic sealers are useful adjuncts to control minor bleeding. In practice, bipolar hemostatic sealers have replaced the use of argon beam coagulation in hepatic surgery and are more effective in the emergency setting as well. Major bleeding and complex hepatic injury identified at laparotomy presents a significant surgical challenge. A logical thought process, thorough understanding of anatomy, and experienced assistance are paramount. The liver consists of a right and left hemiliver, subdivided into the eight segments of the liver. The portal triad, which includes hepatic artery, portal vein, and bile duct branches, is enclosed within an extension of Glissen's capsule which is resistant to injury. The portal triad branches course within the segments of the liver. The major hepatic veins run between the segments of the liver, are not covered

by Glissen's capsule, and are therefore more vulnerable to injury. In addition, the three major hepatic veins have long intrahepatic segments and short (1–2 cm) extrahepatic segments. Injury to the intrahepatic branches of the major hepatic veins is more common than extrahepatic vein injury [61]. Furthermore, 7–11 short hepatic veins drain the liver and may be a source of blood loss. Massive transfusion protocol activation should be considered to allow hemostatic resuscitation. Additionally, a highly trained anesthesia team, cell saver, and rapid transfuser are needed for a successful operation. The primary goal is control of hemorrhage. As such, the first maneuver should again be manual compression and perihepatic packing. This will stop bleeding and allow resuscitation of the patient. The proper technique requires approximation of hepatic parenchyma and restoration of anatomy, with inward and posterior pressure. Perihepatic packing will control most sources of bleeding. When this is the case, additional maneuvers to mobilize the liver are not warranted and may, in fact, increase hemorrhage. At this point, a damage control laparotomy can be completed and temporary abdominal closure applied. Postoperative hepatic angiography may be useful after damage control surgery for liver injury, and as many as half of these patients will have angiographic findings consistent with bleeding and require therapeutic embolization [45]. When bleeding is not controlled by compression, the next step is a Pringle maneuver, which is both therapeutic and diagnostic. Control of hemorrhage with the Pringle maneuver indicates injury to branches of the portal vein and/or hepatic artery and at the same time will temporarily arrest hemorrhage. This maneuver requires occlusion of the portal vein and hepatic artery in the hepatoduodenal ligament and can be accomplished with digital compression, an atraumatic clamp, or with a doubled large vessel loop [6]. At this point, the injured vessels within the liver parenchyma must be identified and sutured. This should be done with the simplest maneuvers. When the injury is readily identified through the injured parenchyma, it should be sutured. If the source of bleeding cannot be readily exposed, it may be necessary to quickly enlarge the defect in the liver for adequate access. As the operation proceeds proximally into the parenchyma of the liver, the vessels are progressively larger. Often stapled hepatotomy or hepatectomy is the simplest means to expose and surgically control the bleeding, and resectional debridement in an anatomic or nonanatomic fashion may be useful, especially when significantly devitalized liver is present. While the need for hepatic resection is uncommon at the time of initial operation, it has been shown to be safe in the management of complex liver injuries [7, 62]. It is critically important to recognize early in the operation that simple maneuvers will not control the bleeding and a bigger operation is required, generally resectional debridement, hepatorrhaphy, or nonanatomic resection. If this decision and the procedure are not completed expeditiously, the patient has greater risk of hemorrhage and transfusion requirement with resultant coagulopathy, hypothermia, and acidosis. When the Pringle maneuver does not subdue the bleeding, a juxtahepatic venous injury should be suspected. Again, a thorough understanding of anatomy and technique is required [6, 25, 61]. Control of the infrahepatic and suprahepatic IVC may be required to prevent exsanguination as the injury exposed and rapid mobilization of the liver may be necessary. Venovenous bypass may also be useful to maintain venous return, limit the volume given for resuscitation, allow rapid transfusion, and prevent the development of bowel edema [63]. Rarely, selective ligation of the hepatic artery may be required. Postoperative angioembolization is generally a better option. Definitive management of the liver injury involves not only control of bleeding, but also removal of devitalized or necrotic liver, control of bile leak, and drainage. This may best be accomplished with resection. For the definitive operation, drains are not necessary for low-grade liver injury, but closed suction drains should be placed when bile is encountered at laparotomy or when operating for high-grade injury given the risk of bile leak [52, 64–67]. However, when damage control laparotomy is employed, definitive management beyond arrest of hemorrhage should be delayed until subsequent operation. Identification and treatment of bile duct leak/injury should be a routine component of the definitive re-exploration of the patient with major liver injury. This involves a cholangiogram and seeking, and oversewing bile leaks within the liver. This can be performed as a cholecystectomy and transcystic cholangiography. Alternatively, saline is injected through the catheter with simultaneous gentle manual compression of the distal common bile duct. Injured bile ducts (what will be a postoperative bile leak) are localized by leakage of saline from the liver parenchymal, oversew this. The ideal time to identify a bile duct leak is in the operating room at the final operation for the liver injury, not by contrast study postoperatively. The complications of nonoperative management—necrosis, abscess, and bile leak—also occur after surgical management of liver injury. Necrosis has been shown to be associated with the use of angioembolization after damage control laparotomy and, as is the case in nonoperative management, often requires surgical management [45, 68]. Similarly, bile leak after operative or nonoperative management of liver injury can often be managed effectively with ERCP and percutaneous drainage, when surgical drains were not previously placed [69].

Summary

Nonoperative management has become the surgical treatment of choice in the hemodynamically stable patient with

blunt hepatic trauma. This treatment is now utilized in the vast majority of liver injuries and has resulted in reduced mortality. The increased use and success of nonoperative management have been facilitated by the development of increasingly higher resolution CT imaging, improved management of physiology and resuscitation (damage control), and routine availability of interventional procedures such as angiography and embolization, image-guided percutaneous drainage, and endoscopy. On the other hand, recognition of the patient who should proceed to immediate laparotomy is of utmost importance. A systematic and logical approach to the control of hemorrhage is required in the operating room. Furthermore, surgeons should have a thorough knowledge of the anatomy and surgical techniques, such as perihepatic packing, effective Pringle maneuver, hepatic mobilization, infrahepatic and suprahepatic control of the IVC, and stapled hepatectomy.

Conflict of interest Jeremy Ward, Louis Alarcon, and Andrew B. Peitzman declare that they have no conflict of interest.

Compliance with ethical requirements This work is in compliance with ethical requirements. Jeremy Ward, Louis Alarcon, and Andrew B. Peitzman declare that this is a review article that includes no studies on humans or animals.

References

1. Matthes G, Stengel D, Seifert J, Rademacher G, Mutze S, Ekkernkamp A. Blunt liver injuries in polytrauma: results from a cohort study with the regular use of whole-body helical computed tomography. World J Surg. 2003;27(10):1124–30. doi:10.1007/s00268-003-6981-0.
2. Lee SK, Carrillo EH. Advances and changes in the management of liver injuries. Am Surg. 2007;73(3):201–6.
3. Malhotra AK, Fabian TC, Croce MA, Gavin TJ, Kudsk KA, Minard G, et al. Blunt hepatic injury: a paradigm shift from operative to nonoperative management in the 1990s. Ann Surg. 2000;231(6):804–13.
4. Velmahos GC, Toutouzas KG, Radin R, Chan L, Demetriades D. Nonoperative treatment of blunt injury to solid abdominal organs: a prospective study. Arch Surg. 2003;138(8):844–51. doi:10.1001/archsurg.138.8.844.
5. Kozar RA, Moore FA, Cothren CC, Moore EE, Sena M, Bulger EM, et al. Risk factors for hepatic morbidity following nonoperative management: multicenter study. Arch Surg. 2006;141(5):451–8. doi:10.1001/archsurg.141.5.451 (discussion 8–9).
6. Peitzman AB, Marsh JW. Advanced operative techniques in the management of complex liver injury. J Trauma Acute Care Surg. 2012;73(3):765–70. doi:10.1097/TA.0b013e318265cef5.
7. Polanco P, Leon S, Pineda J, Puyana JC, Ochoa JB, Alarcon L, et al. Hepatic resection in the management of complex injury to the liver. J Trauma. 2008;65(6):1264–9. doi:10.1097/TA.0b013e3181904749 (discussion 9–70).
8. Becker CD, Mentha G, Terrier F. Blunt abdominal trauma in adults: role of CT in the diagnosis and management of visceral injuries. Part 1: liver and spleen. Eur Radiol. 1998;8(4):553–62.
9. Ochsner MG. Factors of failure for nonoperative management of blunt liver and splenic injuries. World J Surg. 2001;25(11):1393–6.
10. Scalea TM, Rodriguez A, Chiu WC, Brenneman FD, Fallon WF Jr, Kato K, et al. Focused assessment with sonography for trauma (FAST): results from an international consensus conference. J Trauma. 1999;46(3):466–72.
11. Sirlin CB, Brown MA, Andrade-Barreto OA, Deutsch R, Fortlage DA, Hoyt DB, et al. Blunt abdominal trauma: clinical value of negative screening US scans. Radiology. 2004;230(3):661–8. doi:10.1148/radiol.2303021707.
12. Wallis A, Kelly MD, Jones L. Angiography and embolisation for solid abdominal organ injury in adults—a current perspective. World J Emerg Surg. 2010;5:18. doi:10.1186/1749-7922-5-18.
13. Kozar RA, Moore FA, Moore EE, West M, Cocanour CS, Davis J, et al. Western Trauma Association critical decisions in trauma: nonoperative management of adult blunt hepatic trauma. J Trauma. 2009;67(6):1144–8. doi:10.1097/TA.0b013e3181ba361f (discussion 8–9).
14. Moore EE, Cogbill TH, Jurkovich GJ, Shackford SR, Malangoni MA, Champion HR. Organ injury scaling: spleen and liver (1994 revision). J Trauma. 1995;38(3):323–4.
15. Poletti PA, Mirvis SE, Shanmuganathan K, Killeen KL, Coldwell D. CT criteria for management of blunt liver trauma: correlation with angiographic and surgical findings. Radiology. 2000;216(2):418–27. doi:10.1148/radiology.216.2.r00au44418.
16. Yoon W, Jeong YY, Kim JK, Seo JJ, Lim HS, Shin SS, et al. CT in blunt liver trauma. Radiographics. 2005;25(1):87–104. doi:10.1148/rg.251045079.
17. Taourel P, Vernhet H, Suau A, Granier C, Lopez FM, Aufort S. Vascular emergencies in liver trauma. Eur J Radiol. 2007;64(1):73–82. doi:10.1016/j.ejrad.2007.07.013.
18. Nance ML, Peden GW, Shapiro MB, Kauder DR, Rotondo MF, Schwab CW. Solid viscus injury predicts major hollow viscus injury in blunt abdominal trauma. J Trauma. 1997;43(4):618–22 (discussion 22–23).
19. Bilgic I, Gelecek S, Akgun AE, Ozmen MM. Predictive value of liver transaminases levels in abdominal trauma. Am J Emerg Med. 2014;32(7):705–8. doi:10.1016/j.ajem.2014.03.052.
20. Tan KK, Bang SL, Vijayan A, Chiu MT. Hepatic enzymes have a role in the diagnosis of hepatic injury after blunt abdominal trauma. Injury. 2009;40(9):978–83. doi:10.1016/j.injury.2009.02.023.
21. Tian Z, Liu H, Su X, Fang Z, Dong Z, Yu C, et al. Role of elevated liver transaminase levels in the diagnosis of liver injury after blunt abdominal trauma. Exp Ther Med. 2012;4(2):255–60. doi:10.3892/etm.2012.575.
22. Christmas AB, Wilson AK, Manning B, Franklin GA, Miller FB, Richardson JD, et al. Selective management of blunt hepatic injuries including nonoperative management is a safe and effective strategy. Surgery. 2005;138(4):606–10. doi:10.1016/j.surg.2005.07.018 (discussion 10–11).
23. Feliciano DV. Continuing evolution in the approach to severe liver trauma. Ann Surg. 1992;216(5):521–3.
24. Pachter HL, Knudson MM, Esrig B, Ross S, Hoyt D, Cogbill T, et al. Status of nonoperative management of blunt hepatic injuries in 1995: a multicenter experience with 404 patients. J Trauma. 1996;40(1):31–8.
25. Badger SA, Barclay R, Campbell P, Mole DJ, Diamond T. Management of liver trauma. World J Surg. 2009;33(12):2522–37. doi:10.1007/s00268-009-0215-z.
26. David Richardson J, Franklin GA, Lukan JK, Carrillo EH, Spain DA, Miller FB, et al. Evolution in the management of hepatic trauma: a 25-year perspective. Ann Surg. 2000;232(3):324–30.
27. Pachter HL. Prometheus bound: evolution in the management of hepatic trauma–from myth to reality. J Trauma Acute Care Surg. 2012;72(2):321–9. doi:10.1097/TA.0b013e31824b15a7.
28. Parks NA, Davis JW, Forman D, Lemaster D. Observation for nonoperative management of blunt liver injuries: how long

is long enough? J Trauma. 2011;70(3):626–9. doi:10.1097/TA.0b013e31820d1c69.
29. Piper GL, Peitzman AB. Current management of hepatic trauma. Surg Clin North Am. 2010;90(4):775–85. doi:10.1016/j.suc.2010.04.009.
30. Raza M, Abbas Y, Devi V, Prasad KV, Rizk KN, Nair PP. Non operative management of abdominal trauma—a 10 years review. World J Emerg Surg. 2013;8:14. doi:10.1186/1749-7922-8-14.
31. Stassen NA, Bhullar I, Cheng JD, Crandall M, Friese R, Guillamondegui O, et al. Nonoperative management of blunt hepatic injury: an Eastern Association for the Surgery of Trauma practice management guideline. J Trauma Acute Care Surg. 2012;73(5 Suppl 4):S288–93. doi:10.1097/TA.0b013e318270160d.
32. Leppaniemi AK, Mentula PJ, Streng MH, Koivikko MP, Handolin LE. Severe hepatic trauma: nonoperative management, definitive repair, or damage control surgery? World J Surg. 2011;35(12):2643–9. doi:10.1007/s00268-011-1309-y.
33. van der Wilden GM, Velmahos GC, Emhoff T, Brancato S, Adams C, Georgakis G, et al. Successful nonoperative management of the most severe blunt liver injuries: a multicenter study of the research consortium of new England centers for trauma. Arch Surg. 2012;147(5):423–8. doi:10.1001/archsurg.2012.147.
34. Velmahos GC, Toutouzas K, Radin R, Chan L, Rhee P, Tillou A, et al. High success with nonoperative management of blunt hepatic trauma: the liver is a sturdy organ. Arch Surg. 2003;138(5):475–80. doi:10.1001/archsurg.138.5.475 (discussion 80–81).
35. Tinkoff G, Esposito TJ, Reed J, Kilgo P, Fildes J, Pasquale M, et al. American Association for the Surgery of Trauma Organ Injury Scale I: spleen, liver, and kidney, validation based on the National Trauma Data Bank. J Am Coll Surg. 2008;207(5):646–55. doi:10.1016/j.jamcollsurg.2008.06.342.
36. Polanco PM, Brown JB, Puyana JC, Billiar TR, Peitzman AB, Sperry JL. The swinging pendulum: a national perspective of nonoperative management in severe blunt liver injury. J Trauma Acute Care Surg. 2013;75(4):590–5. doi:10.1097/TA.0b013e3182a53a3e.
37. Carrillo EH, Spain DA, Wohltmann CD, Schmieg RE, Boaz PW, Miller FB, et al. Interventional techniques are useful adjuncts in nonoperative management of hepatic injuries. J Trauma. 1999;46(4):619–22 (discussion 22–24).
38. Goldman R, Zilkoski M, Mullins R, Mayberry J, Deveney C, Trunkey D. Delayed celiotomy for the treatment of bile leak, compartment syndrome, and other hazards of nonoperative management of blunt liver injury. Am J Surg. 2003;185(5):492–7.
39. Fang JF, Chen RJ, Wong YC, Lin BC, Hsu YB, Kao JL, et al. Pooling of contrast material on computed tomography mandates aggressive management of blunt hepatic injury. Am J Surg. 1998;176(4):315–9.
40. Fang JF, Wong YC, Lin BC, Hsu YP, Chen MF. The CT risk factors for the need of operative treatment in initially hemodynamically stable patients after blunt hepatic trauma. J Trauma. 2006;61(3):547–53. doi:10.1097/01.ta.0000196571.12389.ee (discussion 53–54).
41. Croce MA, Fabian TC, Menke PG, Waddle-Smith L, Minard G, Kudsk KA, et al. Nonoperative management of blunt hepatic trauma is the treatment of choice for hemodynamically stable patients. Results of a prospective trial. Ann Surg. 1995;221(6):744–53 (discussion 53–55).
42. Ciraulo DL, Luk S, Palter M, Cowell V, Welch J, Cortes V, et al. Selective hepatic arterial embolization of grade IV and V blunt hepatic injuries: an extension of resuscitation in the nonoperative management of traumatic hepatic injuries. J Trauma. 1998;45(2):353–8 (discussion 8–9).
43. Huang YC, Wu SC, Fu CY, Chen YF, Chen RJ, Hsieh CH, et al. Tomographic findings are not always predictive of failed nonoperative management in blunt hepatic injury. Am J Surg. 2012;203(4):448–53. doi:10.1016/j.amjsurg.2011.01.031.
44. Letoublon C, Morra I, Chen Y, Monnin V, Voirin D, Arvieux C. Hepatic arterial embolization in the management of blunt hepatic trauma: indications and complications. J Trauma. 2011;70(5):1032–6. doi:10.1097/TA.0b013e31820e7ca1 (discussion 6–7).
45. Misselbeck TS, Teicher EJ, Cipolle MD, Pasquale MD, Shah KT, Dangleben DA, et al. Hepatic angioembolization in trauma patients: indications and complications. J Trauma. 2009;67(4):769–73. doi:10.1097/TA.0b013e3181b5ce7f.
46. Mohr AM, Lavery RF, Barone A, Bahramipour P, Magnotti LJ, Osband AJ, et al. Angiographic embolization for liver injuries: low mortality, high morbidity. J Trauma. 2003;55(6):1077–81. doi:10.1097/01.TA.0000100219.02085.AB (discussion 81–82).
47. Abdelrahman H, Ajaj A, Atique S, El-Menyar A, Al-Thani H. Conservative management of major liver necrosis after angioembolization in a patient with blunt trauma. Case Rep Surg. 2013;2013:954050. doi:10.1155/2013/954050.
48. Dabbs DN, Stein DM, Philosophe B, Scalea TM. Treatment of major hepatic necrosis: lobectomy versus serial debridement. J Trauma. 2010;69(3):562–7. doi:10.1097/TA.0b013e3181ebf591.
49. Kanakis MA, Thomas T, Martinakis VG, Brountzos E, Varsamidakis N. Successful management of severe blunt hepatic trauma by angiographic embolization. Updates Surg. 2012;64(4):303–6. doi:10.1007/s13304-011-0122-3.
50. Wahl WL, Brandt MM, Hemmila MR, Arbabi S. Diagnosis and management of bile leaks after blunt liver injury. Surgery. 2005;138(4):742–7. doi:10.1016/j.surg.2005.07.021 (discussion 7–8).
51. Yuan KC, Wong YC, Fu CY, Chang CJ, Kang SC, Hsu YP. Screening and management of major bile leak after blunt liver trauma: a retrospective single center study. Scand J Trauma Resusc Emerg Med. 2014;22(1):26. doi:10.1186/1757-7241-22-26.
52. Bala M, Gazalla SA, Faroja M, Bloom AI, Zamir G, Rivkind AI, et al. Complications of high grade liver injuries: management and outcomewith focus on bile leaks. Scand J Trauma Resusc Emerg Med. 2012;20:20. doi:10.1186/1757-7241-20-20.
53. Singh V, Narasimhan KL, Verma GR, Singh G. Endoscopic management of traumatic hepatobiliary injuries. J Gastroenterol Hepatol. 2007;22(8):1205–9. doi:10.1111/j.1440-1746.2006.04780.x.
54. Carrillo EH, Reed DN Jr, Gordon L, Spain DA, Richardson JD. Delayed laparoscopy facilitates the management of biliary peritonitis in patients with complex liver injuries. Surg Endosc. 2001;15(3):319–22. doi:10.1007/s004640000300.
55. Letoublon C, Chen Y, Arvieux C, Voirin D, Morra I, Broux C, et al. Delayed celiotomy or laparoscopy as part of the nonoperative management of blunt hepatic trauma. World J Surg. 2008;32(6):1189–93. doi:10.1007/s00268-007-9439-y.
56. Pilgrim CH, Usatoff V. Role of laparoscopy in blunt liver trauma. ANZ J Surg. 2006;76(5):403–6. doi:10.1111/j.1445-2197.2006.03731.x.
57. Pearl LB, Trunkey DD. Compartment syndrome of the liver. J Trauma. 1999;47(4):796–8.
58. Devakumar Murugesan S, Sathyanesan J, Lakshmanan A, Ramaswami S, Perumal S, Ulagendra Perumal S, et al. Massive hemobilia: a diagnostic and therapeutic challenge. World J Surg. 2014;. doi:10.1007/s00268-013-2435-5.
59. Green MH, Duell RM, Johnson CD, Jamieson NV. Haemobilia. Br J Surg. 2001;88(6):773–86. doi:10.1046/j.1365-2168.2001.01756.x.
60. Srivastava DN, Sharma S, Pal S, Thulkar S, Seith A, Bandhu S, et al. Transcatheter arterial embolization in the management of hemobilia. Abdom Imaging. 2006;31(4):439–48. doi:10.1007/s00261-005-0392-7.

61. Buckman RF Jr, Miraliakbari R, Badellino MM. Juxtahepatic venous injuries: a critical review of reported management strategies. J Trauma. 2000;48(5):978–84.
62. Li Petri S, Gruttadauria S, Pagano D, Echeverri GJ, Di Francesco F, Cintorino D, et al. Surgical management of complex liver trauma: a single liver transplant center experience. Am Surg. 2012;78(1):20–5.
63. Baumgartner F, Scudamore C, Nair C, Karusseit O, Hemming A. Venovenous bypass for major hepatic and caval trauma. J Trauma. 1995;39(4):671–3.
64. Gillmore D, McSwain NE Jr, Browder IW. Hepatic trauma: to drain or not to drain? J Trauma. 1987;27(8):898–902.
65. Mohseni S, Talving P, Kobayashi L, Kim D, Inaba K, Lam L, et al. Closed-suction drain placement at laparotomy in isolated solid organ injury is not associated with decreased risk of deep surgical site infection. Am Surg. 2012;78(10):1187–91.
66. Mullins RJ, Stone HH, Dunlop WE, Strom PR. Hepatic trauma: evaluation of routine drainage. South Med J. 1985;78(3):259–61.
67. Noyes LD, Doyle DJ, McSwain NE Jr. Septic complications associated with the use of peritoneal drains in liver trauma. J Trauma. 1988;28(3):337–46.
68. Dabbs DN, Stein DM, Scalea TM. Major hepatic necrosis: a common complication after angioembolization for treatment of high-grade liver injuries. J Trauma. 2009;66(3):621–7. doi:10.1097/TA.0b013e31819919f2 (discussion 7–9).
69. Anand RJ, Ferrada PA, Darwin PE, Bochicchio GV, Scalea TM. Endoscopic retrograde cholangiopancreatography is an effective treatment for bile leak after severe liver trauma. J Trauma. 2011;71(2):480–5. doi:10.1097/TA.0b013e3181efc270.