

# Management of blunt pancreatic trauma: what's new?

D. A. Potoka<sup>1</sup> · B. A. Gaines<sup>1</sup> · A. Leppäniemi<sup>3</sup> · A. B. Peitzman<sup>1,2</sup>

Received: 29 October 2014 / Accepted: 3 March 2015 / Published online: 17 March 2015  
© Springer-Verlag Berlin Heidelberg 2015

**Abstract** Pancreatic injuries are relatively uncommon but present a major challenge to the surgeon in terms of both diagnosis and management. Pancreatic injuries are associated with significant mortality, primarily due to associated injuries, and pancreas-specific morbidity, especially in cases of delayed diagnosis. Early diagnosis of pancreatic trauma is a key for optimal management, but remains a challenge even with more advanced imaging modalities. For both penetrating and blunt pancreatic injuries, the presence of main pancreatic ductal injury is the major determinant of morbidity and the major factor guiding management decisions. For main pancreatic ductal injury, surgery remains the preferred approach with distal pancreatectomy for most injuries and more conservative surgical management for proximal ductal injuries involving the head of the pancreas. More recently, nonoperative management has been utilized, especially in the pediatric population, with the potential for increased rates of pseudocyst and pancreatic fistulae and the potential for the need for further intervention and increased hospital stay. This review presents recent data focusing on the diagnosis, management, and outcomes of blunt pancreatic injury.

**Keywords** Pancreatic injury · Blunt abdominal trauma · ERCP · Nonoperative management

## Introduction

Pancreatic trauma presents a major challenge to the surgeon in terms of both diagnosis and management. Despite improvements in prehospital care, intensive care, and operative techniques, the mortality and morbidity associated with pancreatic injury remain high. Recent literature has favored simplified management protocols utilizing external drainage and distal pancreatectomy rather than more complex procedures. In this review, we discuss recent data, focusing on diagnosis and management of pancreatic injury.

## Incidence and mechanism

Pancreatic injury is relatively uncommon, occurring in 0.2–2 % of all trauma patients and 3–12 % of patients with abdominal injury [1–7]. Pancreatic injury occurs predominantly in males (68–90 % male) and in the young (mean age 27–35 years) [4–11]. In the United States, penetrating trauma is responsible for the majority of pancreatic injuries in adults, 48–81 % [4, 5, 10, 12, 13]. Outside of the United States, blunt trauma is the cause of the majority of pancreatic injuries [14–17]. In blunt injury, the pancreas is most commonly injured by crushing force applied to the upper abdomen resulting in compression of the pancreas against the lumbar vertebral column with crush injury or transection in the region of the pancreatic neck. Blunt force to the right side of the abdomen may cause combined duodenal and pancreatic injuries as these structures are compressed against the vertebral column. Pancreatic injury can range in severity from traumatic pancreatitis, contusions, and minor lacerations to complete gland transection and main pancreatic ductal injury.

✉ A. B. Peitzman  
peitzmanab@upmc.edu

<sup>1</sup> University of Pittsburgh School of Medicine, Pittsburgh, PA 15213, USA

<sup>2</sup> F-1281, UPMC-Presbyterian, Pittsburgh, PA 15213, USA

<sup>3</sup> University of Helsinki, Helsinki, Finland

Due to the retroperitoneal location of the pancreas and its proximity to important viscera and major vascular structures, pancreatic injury is commonly associated with injury to multiple abdominal organs and vascular structures. Associated injuries occur in 50–100 % of patients with pancreatic injury, with an average of 1.6–4.5 associated injuries [5, 9, 12, 13, 15–18]. The liver, major vascular structures, colon or small bowel, duodenum, stomach, spleen, and kidney are the most commonly associated intra-abdominal injuries [5, 15]. Penetrating injuries result in concomitant vascular injuries in 50 % of patients [12]. Associated injuries to the chest, central nervous system, and skeletal system are also common [5]. Isolated pancreatic injuries occur in 22 % of patients with blunt abdominal trauma [4, 12].

Trauma is the most common cause of acute pancreatitis and pancreatic pseudocyst in the pediatric population [19–22]. Blunt trauma accounts for 75–100 % of pancreatic injuries in the pediatric population, mainly due to motor vehicle collisions, pedestrian injuries, bicycle accidents, child abuse, and falls [19–25].

Several pancreatic injury grading schemes have been devised (Table 1). The AAST grading scheme and the scheme devised by Lucas are most commonly used in the literature [26–28]. In both grading schemes, injury grade is primarily determined by the presence or absence of main pancreatic ductal injury and the anatomic location of the injury within the gland. These grading schemes are clinically useful since management of pancreatic injury is dependent upon the grade of injury; i.e. the presence or absence of main pancreatic ductal injury and the anatomic location of injury within the gland.

## Diagnosis

Early diagnosis of pancreatic injury, especially in patients with blunt abdominal trauma without an indication for urgent laparotomy, remains a challenge. Beyond diagnosis of the injury itself, the presence or absence of injury to the main pancreatic duct is the most important diagnostic question, since injury to the main pancreatic duct is associated with higher pancreas-specific mortality and morbidity [29, 30]. Furthermore, delays in diagnosis of pancreatic injury are associated with higher rates of complications [4, 31, 32].

Diagnosis of pancreatic injury following penetrating abdominal trauma is usually made at the time of laparotomy. The diagnosis of pancreatic injury and need for laparotomy in patients with blunt abdominal trauma remain difficult. In patients undergoing laparotomy for either mechanism, the increased morbidity associated with missed pancreatic injury mandates a careful assessment of the pancreas. The pancreas should be exposed via the lesser sac with thorough exploration of the pancreas performed in patients with signs of pancreatic injury such as peripancreatic hematoma or fluid. This involves visual inspection and bimanual palpation of the entire pancreas. Mobilization of the pancreas may be necessary to accomplish this. In patients undergoing damage control laparotomy, after control of hemorrhage and gastrointestinal contamination, peripancreatic drains should be placed when pancreatic injury is suspected to control pancreatic secretions with more thorough examination of the pancreas and definitive management deferred until re-exploration after the patient stabilizes.

**Table 1** Grading of pancreatic injury [26]

### AAST grading for pancreatic injury

Grade I	Hematoma: mild contusion without duct injury Laceration: superficial laceration without duct injury
Grade II	Hematoma: major contusion without duct injury or tissue loss Laceration: major laceration without duct injury or tissue loss
Grade III	Distal transection or parenchymal injury with duct injury
Grade IV	Proximal transection or parenchymal injury
Grade V	Massive disruption of pancreatic head

### Lucas classification of pancreatic injury [27]

Grade I	Simple superficial contusion with minimal parenchymal damage affecting any part of the pancreas but without ductal injury
Grade II	Deep laceration, perforation, or transection of the body/tail of the pancreas with the possibility of pancreatic duct injury
Grade III	Severe crushing, perforation, or transection of the head of the pancreas with or without duct injury, but with an intact duodenum
Grade IV	Combined pancreaticoduodenal injuries (a) With mild pancreatic injury (b) With severe pancreatic injury and duct disruption

In patients without an indication for urgent laparotomy at presentation, the diagnosis of pancreatic injury can be a challenge. Because of the retroperitoneal location of the pancreas, clinical signs of pancreatic injury may be subtle and become apparent only later in the post-injury course. The clinical signs and radiographic findings of pancreatic injury can evolve with time as pancreatic secretions become activated and pancreatic and peripancreatic inflammation increases. Both laboratory evaluation and radiographic studies have significant limitations in this situation, and a strong suspicion of pancreatic injury based on mechanism and clinical evaluation is required. The difficulty in diagnosis of blunt pancreatic injury is illustrated by the series reported by Wisner et al. [4], in which the time from injury to operation was 6–24 h in 23 % and greater than 24 h in 19 % of patients suffering blunt pancreatic injuries. Isolated blunt pancreatic injuries may especially provide a diagnostic challenge. In a series by Leppäniemi et al. [14], the mean time from injury to operation in patients with isolated blunt pancreatic injuries was 9.4 days. Patton et al. [10] showed that in patients with delayed diagnoses of pancreatic injuries, the mean delay in diagnosis was 3.8 days with an unfavorable outcome in 60 % of these patients. The importance of early diagnosis and management of pancreatic injury are further demonstrated by the series reported by Lin et al. [31]. In this report, Grade III injuries treated within 12 h had a complication rate of 58 % compared to 80 % for those operated on more than 24 h after injury. In addition, all five deaths in this group occurred in the group operated on more than 24 h after injury. Blunt trauma, intoxication on admission, low Injury Severity Score, low Abdominal Trauma Index, absence of associated abdominal organ injury, and initial nonoperative management have been identified as significant risk factors for the delayed diagnosis of major pancreatic injury [33].

Serum amylase determination is the most widely used laboratory test to aid in the diagnosis of pancreatic injury. However, serum amylase is neither sensitive nor specific. Serum amylase may be elevated in only 14–80 % of patients with blunt pancreatic injury [4, 12, 13, 16, 34–36]. Even in the presence of complete pancreatic transection, initial serum amylase may be normal in 30–35 % of patients [12, 37]. Furthermore, elevated amylase or lipase levels occur in patients without pancreatic injury [13, 38–42]. Several studies have demonstrated that severe head injury, in the absence of pancreatic injury, may result in hyperamylasemia in the early post-injury period [4, 12, 16, 34, 35, 39–42]. However, following the trend in serial amylase levels may be useful in the diagnosis of pancreatic injury. In the series reported by Takishima et al. including 73 patients with proven blunt pancreatic injury, 83.6 % of patients had an elevated serum amylase level on admission. However, in the patients presenting at least 3 h after injury,

100 % demonstrated elevation of serum amylase [36]. Serum lipase determination may also aid in the diagnosis of pancreatic injury [23, 29].

Because of the retroperitoneal location of the pancreas, initial physical examination, diagnostic peritoneal lavage (DPL), and ultrasonography are relatively insensitive in detection of pancreatic injury. Physical signs and symptoms such as abdominal pain, abdominal tenderness, and abdominal wall ecchymosis may suggest the presence of pancreatic injury; however, these findings are nonspecific. Early physical signs may be absent because the pancreatic injury often evolves over time, with activation of pancreatic enzymes and initiation of the inflammatory response. However, the absence of such clinical findings does not exclude pancreatic injury in a patient with a typical mechanism of injury. In the report by Smego, 34 % of patients with documented pancreatic injury had negative or unreliable abdominal exams at the time of presentation, while Coggill et al. reported a false-negative rate of 31 % for DPL [5, 13]. Even measurement of amylase in the dialysate fluid has been of low yield in diagnosis of pancreatic injuries [4, 40, 43, 44].

Computed tomography (CT) is the primary imaging modality used in the diagnosis of blunt pancreatic injury. As with serum amylase, CT is an imperfect test for the early diagnosis of pancreatic injury. CT findings of acute pancreatic injury include specific features such as fracture or transection of the pancreas, pancreatic enlargement or hematoma, fluid separating the splenic vein and pancreas, and increased attenuation of fat around the pancreas, as well as non-specific factors such as thickening of the anterior renal fascia, fluid in the lesser sac, extraperitoneal or intraperitoneal fluid, and associated left upper quadrant injuries [46]. The reported sensitivity of CT in detection of pancreatic injury varies widely in the literature, ranging from 28 to 85 % [6, 11, 15, 29, 39–45]. CT is especially insensitive in detection of pancreatic ductal injury. The reported sensitivity of CT for detection of pancreatic ductal injury ranges from 42.9 to 70 % when correlated with operative or ERCP findings [15, 29, 44]. In a study examining the efficacy of spiral CT in the diagnosis of pancreatic injury, Ilahi et al. [11] showed a sensitivity of 68 % and an accuracy of 69 %, but a positive predictive value of 100 %. In an AAST-sponsored multicenter retrospective study, the sensitivity and specificity of 16- and 64-multidetector CT (MDCT) for the diagnosis of pancreatic injury and pancreatic ductal injury were determined [45]. For pancreatic injury, the sensitivity was 60.1 and 47.2 % for 16- and 64-MDCT, respectively. For pancreatic ductal injury, the sensitivity was only 54 and 52.4 % for 16- and 64-MDCT, respectively. These data demonstrate the relatively poor sensitivity for pancreatic injury and pancreatic ductal injury even with advanced CT technology. The

sensitivity of CT scan may improve with time after injury, as tissue damage from activated pancreatic secretions and peripancreatic inflammation evolves over time. Therefore, repeat CT during a course of observation may be warranted for patients with persistent symptoms of abdominal pain, tenderness, fever, nausea, or vomiting or with persistent hyperamylasemia.

Endoscopic retrograde cholangiopancreatography (ERCP) is the most sensitive technique, short of operative exploration, for diagnosis of pancreatic ductal injury. However, the logistics of obtaining an ERCP in an acutely injured patient at all hours make the use of ERCP in the initial assessment phase of injury limited. Kim et al. [43] performed ERCP within 15 h to 28 days (mean 72 h) following injury in 23 patients with blunt abdominal trauma, and demonstrated pancreatic duct injury in 14 of these patients. In this study, patients undergoing ERCP within 72 h had a lower complication rate (22.2 %; pseudocyst and fistula) compared to patients undergoing ERCP at later times (100 %) emphasizing the morbidity associated with missed pancreatic ductal injuries and the potential utility for ERCP in early diagnosis of ductal injury [43]. Duchesne et al. [42] have also reported use of ERCP in identification and nonoperative management of low-grade pancreatic injury. In addition to the diagnosis of pancreatic ductal injury in patients with blunt abdominal trauma, treatment of such injuries with endoscopically placed stents has been described as case reports [15, 46–49]. Rogers et al. [48] reported the use of ERCP in 26 patients with blunt pancreatic trauma at a mean of 19 days following the injury. Of these patients, 69 % had demonstrated ductal abnormalities and half of these patients underwent endoscopic intervention alone. ERCP may especially prove useful in patients initially managed nonoperatively, in which demonstration of a ductal injury would alter management by prompting laparotomy [29, 42]. ERCP is also useful to diagnose pancreatic ductal disruption and to define ductal anatomy in patients presenting late after injury with complications such as persistent pancreatic fistula or pseudocyst [49]. In these often complex cases, knowledge of ductal anatomy may aid in operative planning. A recent paper from Cape Town reported ERCP being used diagnostically and therapeutically in 48 patients. ERCP allowed 25 % of the patients to be treated conservatively and 50 % of the patients had ERCP applied successfully therapeutically. Thus, operation was avoided in 75 % of their patients [15]. A useful management algorithm from the Western Trauma Association for the management of pancreatic injury was recently published [32] (Fig. 1).

Recently, magnetic resonance cholangiopancreatography (MRCP) has been used increasingly in the diagnosis of pancreaticobiliary disease [50, 51]. These reports have shown that MRCP can be performed in a subset of trauma

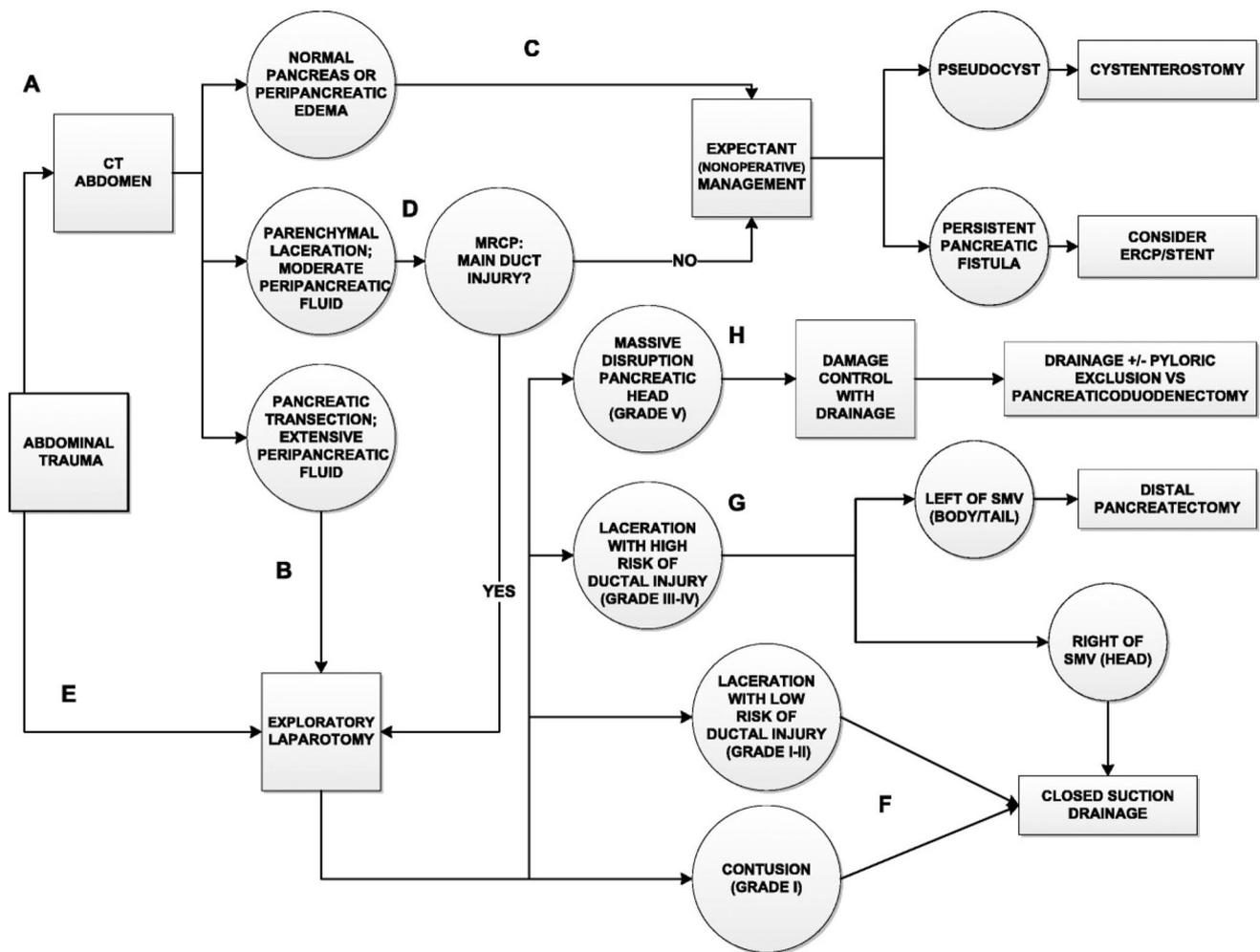
patients and can provide useful information concerning pancreatic ductal anatomy which may alter therapy. Although the noninvasive nature of this test is advantageous, its use in the trauma setting may be limited by a variety of factors including patient status, presence of associated injuries, and availability of MRI. In addition, MRCP does not offer the opportunity for therapeutic intervention which is available with ERCP.

## Management

The status of the pancreatic duct, the location of injury (proximal versus distal), and the overall status of the patient are the major determinants of the appropriate management for pancreatic injury. Recent reports suggest that more conservative management protocols utilizing external drainage and distal pancreatectomy result in lower mortality and morbidity rates compared to more radical procedures utilizing complex resections and pancreaticoenteric anastomoses [8, 10, 13, 15, 24, 25, 28, 42, 52, 53].

Adequate exposure of the pancreas is required to accurately diagnose and manage pancreatic injury. Initial exposure of the anterior surface of the pancreas can be obtained by entering the lesser sac via division of the gastrocolic omentum. More thorough examination of the pancreas requires an extended Kocher maneuver, division of the peritoneum along the inferior border of the pancreas, and left medial visceral rotation. After performing these maneuvers, the posterior aspects of the head and tail of the pancreas can be examined and the gland can be inspected further by bimanual palpation. The entire body and tail of the pancreas can be elevated into the field with these maneuvers to allow thorough inspection and palpation of the gland.

A variety of techniques have been advocated to assess the status of the main pancreatic duct at laparotomy. Some have advocated the use of intraoperative pancreatography to assess the status of the pancreatic duct. However, we strongly discourage these maneuvers as unnecessary due to avoidable added risk; i.e., a duodenotomy in the setting of pancreatic injury and enzymes. Intraoperative ERCP has also been proposed as a method for assessment of the status of the pancreatic duct, although multiple logistic factors may limit the usefulness of this technique. In most cases, a careful inspection and bimanual palpation of the pancreas is adequate to determine the presence of ductal injury. Heitsch et al. [3] described several intraoperative criteria to determine the presence of ductal injury including direct visualization of ductal injury, complete pancreatic transection, laceration through more than one half of the diameter of the pancreas, central perforation of the pancreas, and severe maceration of the pancreas. In the series reported by Patton et al. [10], a management protocol that was dependent upon



**Fig. 1** Western Trauma Association management algorithm for pancreatic injuries (from Biffl et al. [32]: Fig. 1, page 942)

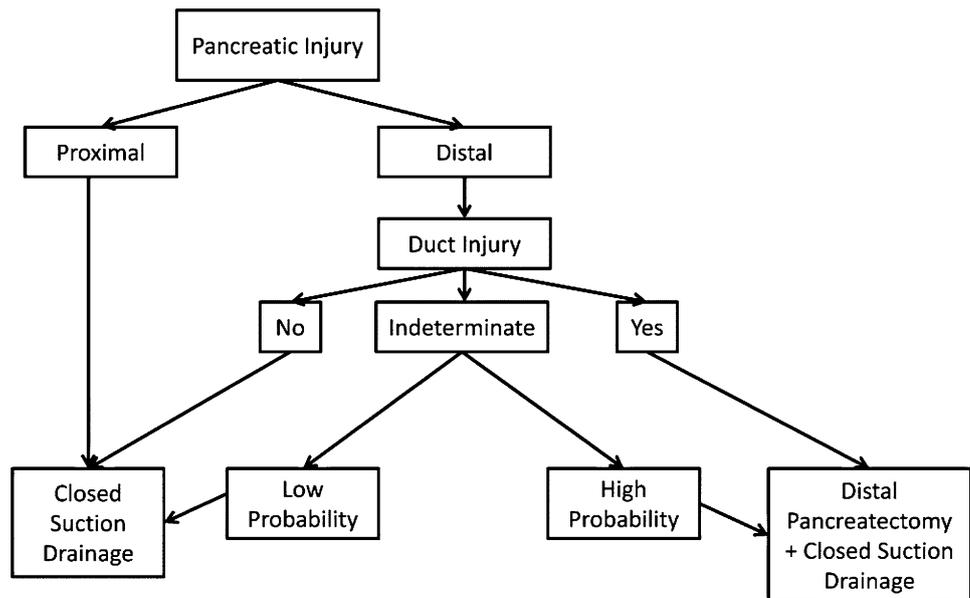
Careful intraoperative inspection without intraoperative pancreatography to assess the status of the pancreatic duct resulted in favorable outcomes with a pancreas-specific mortality rate of 1.6 % and a complication rate of 30 %. Validation of this approach by the Memphis group was recently published [28]. The algorithm for intraoperative decision-making is shown in Fig. 2. The authors used clinical criteria as described by Heitsch to define the patients as having an obvious ductal injury, an obvious intact duct, or an indeterminate duct injury and treated indeterminate patients with high suspicion of ductal injury as if they had a ductal injury. These results suggest that careful intraoperative assessment of the pancreatic duct while maintaining a high suspicion for ductal injury is adequate in most cases.

Pancreatic injury without evidence of ductal injury (Grades I and II) can be managed with debridement of devitalized tissue and external drainage alone. Management of low-grade injuries with drainage alone results in low morbidity rates (0–17 %) and minimal pancreatic-related

mortality [13, 28]. Suturing the injured capsule or parenchyma in these minor injuries is unnecessary and may result in greater complication rates, such as pseudocyst formation, compared to drainage alone.

The decision whether to drain limited pancreatic injuries and what type of drainage to use has been controversial. Although most authors recommend drainage of all pancreatic injuries, some series have reported higher complication rates in nonductal injuries treated with drainage compared to those left undrained [4, 6]. Series comparing the use of passive drains with active drains (sump or closed suction drains) demonstrate reduced complications with active drains [16, 17]. Furthermore, a randomized trial comparing closed suction drainage to sump drainage demonstrated that closed suction drainage resulted fewer intra-abdominal abscesses (2.6 %) compared to sump drainage (20.8 %) [52]. Most authors recommend drainage of all pancreatic injuries, with closed suction drains as the method of choice [10].

**Fig. 2** Defined management algorithm for traumatic pancreatic injuries. Proximal injuries are classified as those that occur to the right of the superior mesenteric vessels. Distal injuries are classified as those that occur to the left of the superior mesenteric vessels (from Sharpe et al. [28]: Fig. 1, page 101)



Distal pancreatectomy and drainage should be the primary mode of management for most distal pancreatic injuries with ductal injury (Grade III) [3–5, 8, 13]. Heitsch et al. [3] first showed a significant reduction in both mortality and morbidity for ductal injuries treated with distal pancreatectomy compared to drainage alone. In the report by Smego and colleagues [13], a protocolized approach to pancreatic injury involving precise classification of injury, an aggressive intraoperative search for pancreatic duct disruption, and the use of distal pancreatectomy for ductal injuries resulted in a low pancreas-related mortality of 3 % and an overall complication rate of 19 %. The transected margin of the pancreas can be controlled with a stapling device or permanent sutures with no difference in outcome [5, 54, 55]. Fibrin glue and other sealants have been applied in an attempt to decrease the incidence of pancreatic leak/fistula in elective cases without clear benefit [56, 57]. The main pancreatic duct should be ligated if easily identified, but not doing so does not appear to increase complications [5]. Spleen preservation should be attempted in otherwise stable patients with uninjured spleens [58]. Preservation of the spleen is especially desirable in children who have higher rates of post-splenectomy infection and present more commonly with isolated pancreatic injuries [5, 10, 57, 59]. Although the performance of a Roux-en-Y pancreaticojejunostomy to the area of ductal injury or to the distal pancreatic segment following transection has been described, we discourage it and recent studies suggest that a simplified approach utilizing distal pancreatectomy and avoiding pancreaticoenteric anastomosis results in relatively low mortality and morbidity [10, 13, 28]. In fact, pancreaticojejunostomy should be avoided whenever possible to limit the potential morbidity associated with pancreaticoenteric anastomoses.

Proximal pancreatic ductal injuries and disruption of the pancreatic head (Grades IV and V injuries) are difficult to manage with little data in the literature to suggest the optimal approach. Recommendations have ranged from simple drainage alone to complex procedures such as pancreaticoduodenectomy or onlay pancreaticojejunostomy. Recent trends favor more conservative management. In the reports by Patton et al. and Sharpe et al. [10, 28], all proximal ductal injuries were treated with closed suction drainage alone. In the earlier series, 37 patients with proximal injury were treated with drainage alone, with only four complications (11 %) and only one potential pancreas-related death (2.7 %) [10]. In follow-up of this protocol, the same group recently reported 245 patients with significant reduction in pancreas-related morbidity and no deaths attributable to the pancreatic injury [28]. The rationale for this approach is that the controlled pancreatic fistula that may result from a well-drained pancreatic injury is easier to deal with and potentially less morbid than the complications that may arise following more aggressive approaches such as pancreaticojejunostomy and pancreaticoduodenectomy.

Combined duodenal and pancreatic injuries are especially challenging. In a series of 129 patients with combined pancreaticoduodenal injuries reported by Feliciano et al. 24 % were managed by simple repair of the duodenum and drainage of the pancreatic injury, 61 % required more complex pancreatic repairs or resection most often done with pyloric exclusion, and 10 % required pancreaticoduodenectomy. In total, 59 % of patients had pyloric exclusion combined with some type of repair [60]. Mortality rates for simpler repair or resection with or without pyloric exclusion were 22.8–25.8 % compared with 46.2 % mortality for patients undergoing pancreaticoduodenectomy. In the

series reported by Mansour et al. [59], of 62 consecutive pancreaticoduodenal injuries, 46 % were treated with drainage with or without simple repair, 41 % underwent pyloric exclusion, and 7 % required pancreaticoduodenectomy with a 19 % overall mortality rate and a 35 % complication rate. Indications for pancreaticoduodenectomy include massive disruption of the pancreatic head with uncontrollable hemorrhage, massive hemorrhage from adjacent vascular structures, and severe combined duodenal, pancreatic, and biliary injuries. A recent review of grade 4 and 5 pancreatic injury using the NTDB compared cohorts undergoing pancreaticoduodenectomy to patients who underwent a less aggressive procedure [61]. Despite a lesser physiologic burden, outcomes were not improved by pancreaticoduodenectomy. In short, pancreaticoduodenectomy should rarely be required, only in the setting of both severe injury of the head of the pancreas and the duodenum; in essence completing the resection that the injury started. Generally, this should be done in a damage control fashion, with reconstruction at a second operation.

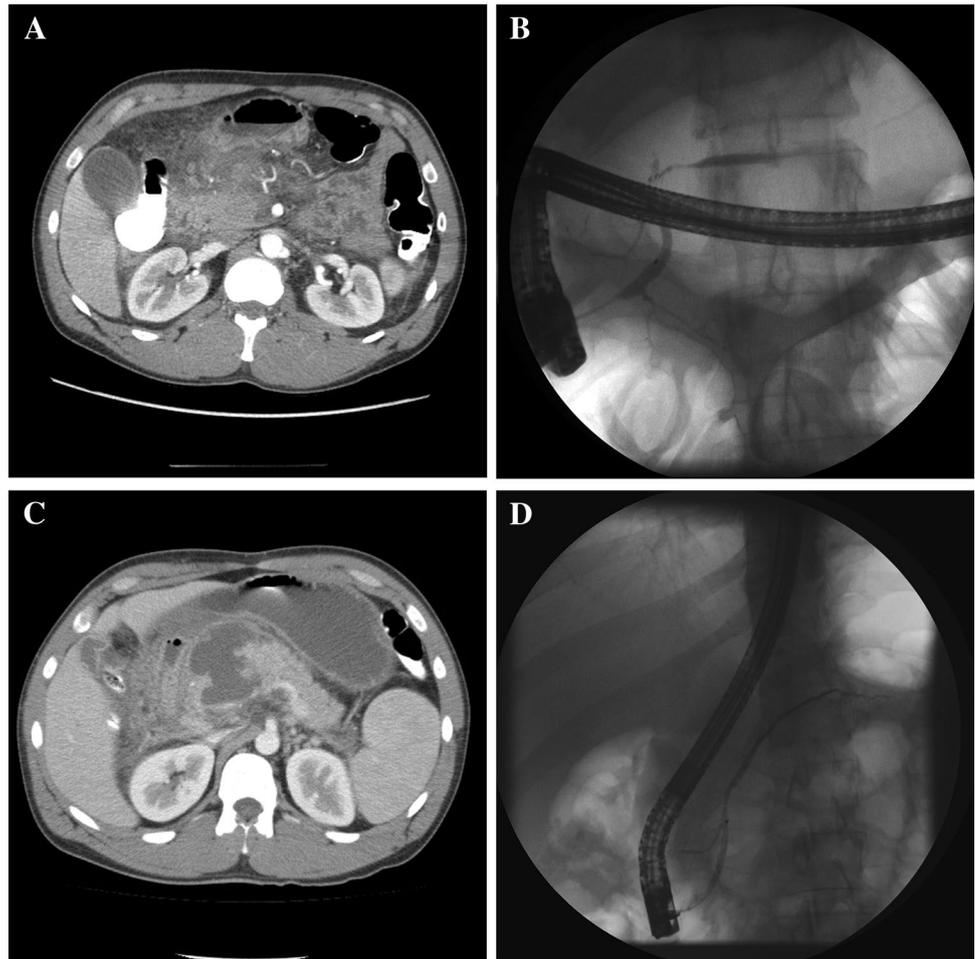
As with other blunt solid organ injuries, nonoperative management has been proposed in the management of select blunt Grade I or II pancreatic injuries, primarily in children. Nonoperative management consists of bowel rest with NG drainage and total parenteral nutrition, serial abdominal examination, and serial serum amylase determination with laparotomy or further imaging studies performed for worsening abdominal examination or persistent hyperamylasemia. Nonoperative management in the setting of main pancreatic ductal injury leads to a high incidence of complications, in particular pseudocysts and pancreatic fistula [49]. In the series reported by Bradley et al. [29], 47.7 % of patients initially managed nonoperatively failed and required operation. The presence of ductal injury was an important factor in failure of nonoperative management with 60 % of patients requiring operation shown to have ductal injury compared to 5 % of patients managed successfully without operation [29]. Furthermore, there was a trend toward increased pancreas-specific mortality and morbidity in patients who failed nonoperative management [29]. Traditionally, the diagnosis and management of pancreatic injury in children has followed the same principles as in adults. However, management of pancreatic trauma in children is influenced by the greater likelihood of blunt injury and isolated pancreatic injury in children as well as the overall trend toward nonoperative management of blunt abdominal solid organ injury in children. Multiple series have demonstrated that minor pancreatic injuries without ductal disruption can be managed nonoperatively with low morbidity [24, 62, 63]. However, nonoperative management for all blunt pancreatic injuries, including complete transection of the gland as proposed by some authors, remains controversial. In a retrospective review of 154 pediatric

patients (age 0–18 years) suffering pancreatic injury, 79 % of children with Grades I or II injuries and 48 % with Grade III, IV, and V injuries were managed without operative procedures specific for the pancreas [63]. However, long-term outcomes and complications in these patients are not clear from these retrospective data. In a series reported by Shilyansky et al. [64], 35 consecutive children with pancreatic injury, including 11 with main ductal transection, were managed nonoperatively with pseudocyst occurring in 29 %. In the series reported by Nadler et al. 47 % of children with pancreatic injury were initially managed nonoperatively, with 21 % of these patients requiring delayed operations for complications compared to 9 % undergoing early operation. Patients with transection of the pancreas who underwent early laparotomy (within 48 h of injury) had a significantly lower length of stay than patients undergoing delayed laparotomy [23]. A recent multi-institutional trial of 167 children reported that distal pancreatectomy was superior to nonoperative management with more rapid resumption of diet, fewer repeat interventions, and less time to complete resolution [9]. If the main pancreatic duct was involved, operative resection resulted in lower morbidity and shorter length of stay. These data demonstrate that in highly selective cases, even more severe pancreatic injuries with ductal injuries can be managed nonoperatively, but at the expense of high rates of pseudocyst formation and additional procedures with potential morbidity and longer hospital stay. This is demonstrated well by the case presented in Fig. 3 [9, 24, 25].

Octreotide, an eight-amino-acid synthetic analog of somatostatin, has been shown to reduce morbidity and enhance closure of pancreatic fistulae after elective pancreatic resections for cancer and chronic pancreatitis [65–67]. The role for octreotide following pancreatic injury remains undefined. In a small nonrandomized review of 28 pancreatic injuries, patients treated with prophylactic octreotide had no complications and no reported negative sequelae from the therapy in contrast to patients not treated with octreotide in which a 29 % complication rate was reported [68]. In a subsequent larger retrospective review including 80 patients with pancreatic injury, there was no difference in complication rates when stratified for pancreatic injury severity in patients treated or not treated with octreotide [69]. The routine use of prophylactic octreotide following pancreatic injury cannot currently be supported based on the existing data.

In 2009, the Eastern Association for the Surgery of Trauma (EAST) published evidence-based guidelines for pancreatic trauma diagnosis and management [8]. At that time, there were insufficient data in the literature to support Level I or Level II recommendations. Six Level III recommendations were made, including (1) delay in the recognition of main pancreatic duct injury causes increased

**Fig. 3** **a** 32-year-old male in an ATV crash. Hemodynamically normal. High suspicion for proximal pancreatic duct injury based on this CT image. Transferred to us 3 days post-injury, not ill, with mild upper abdominal tenderness. **b** ERCP confirmed an injury to the main pancreatic duct. A stent was placed. **c** Follow-up CT 3½ weeks post-injury because of more abdominal pain confirmed this retrogastric pseudocyst. This was drained percutaneously, going through both walls of the stomach. **d** Follow-up ERCP at 8 weeks shows complete healing of the pancreatic duct



morbidity; (2) CT scan is suggestive but not diagnostic of pancreatic injury; (3) amylase/lipase levels are suggestive but not diagnostic of pancreatic injury; (4) Grades I and II injuries can be managed by drainage alone; (5) Grade III injuries should be managed with resection, and drainage; and (6) closed suction is preferred to sump suction.

## Outcomes

### Mortality

Mortality for pancreatic injury ranges from 9 to 34 % [4, 5, 7–10, 12, 13]. Early mortality following pancreatic trauma is due primarily to associated injuries, predominantly vascular injuries. Combined pancreaticoduodenal injuries are associated with increased mortality compared to isolated pancreatic injuries. Feliciano et al. and Jones [12, 60], reported mortality rates of 29.4 and 32 % for combined pancreaticoduodenal injuries. Sepsis, multisystem organ failure, and respiratory failure account for the majority of late deaths [61]. Late deaths are generally associated with

complications of the pancreatic or hollow viscus injury. Outcomes in recent series of pancreatic trauma are shown in Table 2.

### Complications

Overall complication rates following pancreatic injury range from 8 to 45 % [4, 5, 7–10, 12, 13, 31]. Unlike mortality, which is dependent predominantly on the presence of associated injuries, the pancreatic injury itself becomes a major factor in late morbidity. Specifically, the presence of main pancreatic ductal injury is associated with increased morbidity, and the incidence of pancreas-related complications correlates with the grade of injury [10, 13, 29]. Smego et al. [13] demonstrated correlation between grade of injury and morbidity with complication rates of 9, 17, 36, and 50 % for Grade I, II, III, and IV injuries, respectively. Morbidity is increased in patients with delayed diagnoses of pancreatic injury, especially those with a missed ductal injury.

Pancreatic fistula occurs in 2–15 % of patients with pancreatic injury [4, 5, 7–10, 12, 13, 31]. Multivariate analyses

**Table 2** Pancreatic trauma outcomes

References	Mechanism	Mortality	Morbidity
Smego et al. [13]	56 % Penetrating 44 % Blunt	29 % Overall 3 % PRM <sup>a</sup>	19 % Abscess and fistula
Wisner et al. [4]	48 % Penetrating 52 % Blunt	9 % Overall	25 % Overall 16 % Fistula 10 % Abscess
Cogbill et al. [5]	54 % Penetrating 46 % blunt	12 % Overall 3 % PRM	45 % Overall 32 % Intra-abdominal abscess 14 % Pancreatic fistula 8 % Pancreatitis 3 % Pseudocyst 3 % Pancreatic hemorrhage 1 % Endocrine insufficiency
Patton et al. [10]	81 % Penetrating 19 % blunt	13 % Overall 1.6 % PRM	31 % Overall 15 % Fistula 14 % Pancreatic abscess 8 % Pancreatitis 3 % Drain tract infection 1.6 % Pseudocyst
Vasquez et al. [7]	100 % Penetrating	27.4 % Overall	29.7 % Overall 15 % Intra-abdominal abscess 11 % Fistula 9 % Postoperative hemorrhage 4 % Pseudocyst 4 % Pancreatitis
Lin et al. [31]	100 % Blunt All major ductal injuries	15.5 % Overall	46.7 % Overall 24.4 % Intra-abdominal abscess 13.3 % Sepsis/MSOF 8.9 % Pancreatic duct stricture 4.4 % Pseudocyst 4.4 % Pancreatitis 2.2 % Fistula
Krige et al. [16]	100 % Blunt	16.4 % Overall	74.5 % Overall 14.5 % Fistula 13.6 % Pseudocyst 2.7 % Pancreatic ascites 50.9 % Nonpancreatic abdominal complications
Heuer et al. [17]	95.5 % Blunt	19 % Overall	50 % Organ failure 32.9 % Multiple organ failure 14.6 % Sepsis
Sharpe et al. [28]	69 % Penetrating	16 % Overall No PRM	8.5 % Pancreatic fistula 8.1 % Pancreatic abscess

<sup>a</sup> Pancreas-related mortality

have demonstrated that the presence of main pancreatic ductal injury is an independent predictor of fistula formation [10]. Fistulae are especially common after proximal pancreatic injuries and combined pancreaticoduodenal injuries, reflecting the difficulty in managing these injuries and the often complex procedures utilized for combined injuries [12, 13]. Most fistulae can be managed conservatively with adequate drainage, maintenance of adequate fluid and electrolyte balance, and nutritional support [10, 28]. In multiple series, operation for management of persistent fistulae is required in only 0–7 % of patients [12, 13, 16, 17, 28, 60]. Patients with fistulae that do not resolve

should undergo evaluation with ERCP to define ductal anatomy and locate the area of ductal injury followed by operative resection by distal pancreatectomy or drainage via pancreaticoenteric anastomosis depending on the location of the injury.

Pancreatic abscess occurs in 10–25 % of patients following pancreatic injury [4, 5, 7–10, 12, 13, 31]. Pancreatic ductal injury and colon injury are independent predictors of abscess formation following pancreatic trauma [10]. Cultures from pancreatic abscesses grow primarily enteric organisms [5, 7]. In the trauma setting, pancreatic abscesses usually arise due to infection of undrained peripancreatic

fluid collections and can initially be managed with percutaneous drainage and antibiotic therapy [10]. Pancreatic abscess can be a lethal complication with mortality rates as high as 20–27 % [60, 64].

Pancreatic pseudocysts occur in 1.6–4 % of patients following pancreatic injury [4, 5, 7–10, 12, 13, 31]. Pseudocysts are most likely to form after missed or inadequately treated ductal injury. Furthermore, the rate of pseudocyst formation following nonoperative management of pancreatic injuries may be as high as 30–44 % [29, 64, 70]. Pseudocysts may be initially managed with percutaneous drainage to convert the pseudocyst into a controlled fistula [10]. However, chronic pseudocysts or those with ductal injuries documented by ERCP will likely require operative intervention (internal drainage versus pancreatic resection) for resolution.

Post-traumatic pancreatitis should be suspected in patients with persistent abdominal pain, nausea, vomiting, and hyperamylasemia. Pancreatitis complicates 3–8 % of pancreatic injuries [4, 5, 7–10, 12, 13, 31]. Post-traumatic pancreatitis can be managed with supportive care including bowel rest and nutritional support. The mortality from post-traumatic pancreatitis depends on the severity of the pancreatitis [10, 12, 16].

Postoperative hemorrhage occurs in 3–10 % of patients following pancreatic trauma [5, 18]. Major hemorrhage occurs secondary to erosion of adjacent retroperitoneal vessels due to the presence of activated pancreatic secretions or the presence of pancreatic abscess. This complication requires reoperation in most patients [18].

Exocrine or endocrine insufficiency following pancreatic trauma is uncommon [4, 12]. Animals and humans have demonstrated that more than 80–90 % of an otherwise normal pancreas must be removed to produce diabetes or malabsorption. Thus, a distal pancreatectomy to the level of the mesenteric vessels is well tolerated in most patients.

## Conclusion

Pancreatic injury is relatively uncommon but can be associated with significant mortality and morbidity, especially in cases where diagnosis and management are delayed. Early diagnosis of pancreatic injury, especially following blunt abdominal trauma, remains a challenge despite the introduction of more advanced imaging modalities. Therefore, a high index of suspicion must be maintained for pancreatic injury in patients with an appropriate mechanism of injury. The presence of main pancreatic ductal injury is the major determinant of morbidity and the major factor guiding treatment decisions. More recently, nonoperative management has been utilized, commonly supplemented by intervention via ERCP, especially in the pediatric population. This nonoperative management has the potential for

increased rates of pseudocyst and pancreatic fistulae with the resultant need for further intervention and increased total hospital stay. For main pancreatic ductal injuries, surgery remains the preferred approach with the type of procedure based on the site of ductal injury.

**Conflict of interest** Douglas A. Potoka, Barbara A. Gaines, Ari Leppäniemi, and Andrew B. Peitzman declare that they have no conflict of interest.

**Ethical standard** This work is in compliance with ethical requirements. Douglas A. Potoka, Barbara A. Gaines, Ari Leppäniemi, and Andrew B. Peitzman declare that this is a review article that includes no studies on humans or animals.

## References

1. Davis JJ, Con I, Nance FC. Diagnosis and management of blunt abdominal trauma. *Ann Surg.* 1976;183:672–8.
2. Jurkovich GJ, Carrico CJ. Pancreatic trauma. *Surg Clin North Am.* 1990;70:575–93.
3. Heitsch RC, Knutson CO, Fulton RL, Jones CE. Delineation of critical factors in the treatment of pancreatic trauma. *Surgery.* 1976;80:523–9.
4. Wisner DH, Wold RL, Frey CF. Diagnosis and treatment of pancreatic injuries. An analysis of management principles. *Arch Surg.* 1990;125:1109–13.
5. Cogbill TH, Moore EE, Morris JA, Hoyt DB, Jurkovich GJ, Mucha P, Ross SE, Feliciano DV, Shackford SR. Distal pancreatectomy for trauma: a multicenter experience. *J Trauma Acute Care Surg.* 1991;31:1600–6.
6. Akhrass R, Yaffe MB, Brandt CP, Reigle M, Fallon WF, Malanconi MA. Pancreatic trauma: a ten-year multi-institutional experience. *Am Surg.* 1997;63:598–604.
7. Vasquez JC, Coimbra R, Hoyt DB, Fortlage D. Management of penetrating pancreatic trauma: an 11-year experience of a level-1 trauma center. *Injury.* 2001;32:753–9.
8. Bokhari F, Phelen H, Holevar M, Brautigam R, Collier B, Como JJ, Clancy K, Cumming JK, Cullinane D, Smith L. East guidelines for the diagnosis and management of pancreatic trauma. The Eastern Association for the Surgery of Trauma. 2009. <http://www.east.org/tpg/pancreas.pdf>. Accessed 15 Aug 2014.
9. Iqbal CW, St Peter SD, Tsao K, et al. Operative vs nonoperative management for blunt pancreatic transection in children: multi-institutional trial. *J Amer Coll Surg.* 2014;218:157–62.
10. Patton JH, Lyden SP, Croce MA, Pritchard FE, Minard G, Kudsk KA, Fabian TC. Pancreatic trauma: a simplified management guideline. *J Trauma Acute Care Surg.* 1997;43:234–9.
11. Ilahi O, Bochicchio GV, Scalea TM. Efficacy of computed tomography in the diagnosis of pancreatic injury in adult blunt trauma patients: a single-institutional study. *Am Surg.* 2002;68:704–7.
12. Jones RC. Management of pancreatic trauma. *Am J Surg.* 1985;150:698–704.
13. Smego DR, Richardson JD, Flint LM. Determinants of outcome in pancreatic trauma. *J Trauma Acute Care Surg.* 1985;25:771–6.
14. Leppaniemi AK, Haapiainen R, Kiviluoto T, Lempinen M. Pancreatic trauma: acute and late manifestations. *Br J Surg.* 1988;75:165–7.
15. Thomason DA, Krige JEJ, Thomson SR, et al. The role of endoscopic retrograde pancreatography in pancreatic trauma: a critical appraisal of 48 patients treated at a tertiary institution. *J Trauma Acute Care Surg.* 2014;76:1362–6.

16. Krige JEJ, Kotze UK, Hameed M, et al. Pancreatic injuries after blunt abdominal trauma: an analysis of 110 patients treated at a level I trauma centre. *South Afr J Surg.* 2011;49:58–67.
17. Heuer M, Hussman B, Lefering R, et al. Pancreatic injury in 285 patients with severe trauma: outcome, course and treatment algorithm. *Lagenbecks Arch Surg.* 2011;396:1067–76.
18. Kong Y, Zhang H, He X, et al. Endoscopic management for pancreatic injuries due to blunt abdominal trauma decreases failure of nonoperative management and incidence of pancreatic associated complications. *Injury.* 2014;45:134–40.
19. Vane DW, Grosfeld JL, West KW, Rescorla FJ. Pancreatic disorders in infancy and childhood: experience with 92 cases. *J Pediatr Surg.* 1989;24:771–6.
20. Lerner A, Branski D, Leberthal E. Pancreatic diseases in children. *Pediatr Clin North Am.* 1996;43:125–56.
21. Takishima T, Sugimoto K, Asari Y, Kikuno T, Hirata M, Kakita A, Ohwada T, Maekawa K. Characteristics of pancreatic injury in children: a comparison with such injury in adults. *J Pediatr Surg.* 1996;31:896–900.
22. Arkovitz MS, Johnson N, Garcia VF. Pancreatic trauma in children: mechanisms of injury. *J Trauma Acute Care Surg.* 1997;42:49–53.
23. Nadler EP, Gardner M, Schall LC, Lynch JM, Ford HR. Management of blunt pancreatic injury in children. *J Trauma Acute Care Surg.* 1999;47:1098–103.
24. Beres AL, Wales PW, Christison-Lagay ER, et al. Non-operative management of high grade pancreatic trauma: is it worth the wait? *J Ped Surg.* 2013;48:1060–4.
25. Mattix KD, Tataria M, Holmes J, et al. Pediatric pancreatic trauma: predictors of nonoperative management failure and associated outcomes. *J Ped Surg.* 2007;42:340–4.
26. Moore EE, Cogbill TH, Malangoni MA, Jurkovich GJ, Champion HR, Gennarelli TA, McAninch JW, Pachter HL, Shackford SR, Trafton PG. Organ injury scaling, II: pancreas, duodenum, small bowel, colon, and rectum. *J Trauma Acute Care Surg.* 1990;30:1427–9.
27. Lucas CE. Diagnosis and treatment of pancreatic and duodenal injury. *Surg Clin North Am.* 1977;57:49–65.
28. Sharpe JP, Magnotti LJ, Weinberg JA, et al. Impact of a defined management algorithm on outcome after traumatic pancreatic injury. *J Trauma ACS.* 2012;72:100–5.
29. Bradley EL, Young PR, Chang MC, Allen JE, Baker CC, Meredith W, Reed L, Thomason M. Diagnosis and initial management of blunt pancreatic trauma: guidelines from a multiinstitutional review. *Ann Surg.* 1998;227:861–9.
30. Tyburski JG, Dente CJ, Wilson RF, Shanti C, Steffes CP, Carlin A. Infectious complications following duodenal and/or pancreatic trauma. *Am Surg.* 2001;67:227–30.
31. Lin BC, Chen RJ, Fang JF, Hsu YP, Kao YC, Kao JL. Management of blunt major pancreatic injury. *J Trauma Acute Care Surg.* 2004;56:774–8.
32. Biffi WL, Moore EE, Croce M, et al. Western Trauma Association critical decisions in trauma: management of pancreatic injury. *J Trauma Acute Care Surg.* 2013;75:941–6.
33. Leppaniemi AK, Haapiainen RK. Risk factors of delayed diagnosis of pancreatic trauma. *Eur J Surg.* 1999;165:1134–7.
34. Moretz JA, Campbell DP, Parker DE, Williams GR. Significance of serum amylase level in evaluating pancreatic trauma. *Am J Surg.* 1975;130:739–41.
35. Takishima T, Sugimoto K, Hirata M, Asari Y, Ohwada T, Kakita A. Serum amylase level on admission in the diagnosis of blunt injury to the pancreas: its significance and limitations. *Ann Surg.* 1997;226:70–6.
36. Adamson WT, Hebra A, Thomas PB, Wagstaff P, Tagge EP, Othersen HB. Serum amylase and lipase alone are not cost-effective screening methods for pediatric pancreatic trauma. *J Pediatr Surg.* 2003;38:354–7.
37. Vitale GC, Larson GM, Davidson PR, Bouwman DL, Weaver DW. Analysis of hyperamylasemia in patients with severe head injury. *J Surg Res.* 1987;43:226–33.
38. Buechter KJ, Arnold M, Steele B, Martin L, Byers P, Gomez G, Zeppa R, Augenstein J. The use of serum amylase and lipase in evaluating and managing blunt abdominal trauma. *Am Surg.* 1990;56:204–8.
39. Pate SV, Spencer JA, el-Hasani S, et al. Imaging of pancreatic trauma. *Br J Radiol.* 1998;71:985–90.
40. Akhrass R, Kim K, Brandt C. Computed tomography: an unreliable indicator of pancreatic trauma. *Am Surg.* 1996;62:647–51.
41. Bigattini D, Boverie JH, Dondelinger RF. CT of blunt trauma of the pancreas in adults. *Eur Radiol.* 1999;9:244–9.
42. Duchesne JC, Schmiege R, Islam S, et al. Selective nonoperative management of low-grade blunt pancreatic injury: are we there yet? *J Trauma.* 2008;65:49–53.
43. Kim HS, Lee DK, Kim IW, Baik SK, Kwon SO, Park JW, Cho NC, Rhoe BS. The role of endoscopic retrograde pancreatography in the treatment of traumatic pancreatic duct injury. *Gastrointest Endosc.* 2001;54:49–55.
44. Smith DR, Stanley RJ, Rue LW. Delayed diagnosis of pancreatic transection after blunt abdominal trauma. *J Trauma Acute Care Surg.* 1996;40:1009–13.
45. Phelan HA, Velmahos GC, Jurkovich GJ, et al. An evaluation of multidetector computed tomography in detecting pancreatic injury: results of a multicenter AAST study. *J Trauma Acute Care Surg.* 2009;66:641–6.
46. Canty TG, Weinman D. Treatment of pancreatic duct disruption in children by an endoscopically placed stent. *J Pediatr Surg.* 2001;36:345–8.
47. Huckfeldt R, Agee C, Nichols WK, Barthel J. Nonoperative treatment of traumatic pancreatic duct disruption using an endoscopically placed stent. *J Trauma Acute Care Surg.* 1996;41:143–4.
48. Rogers SJ, Cello JP, Schechter WP. Endoscopic retrograde cholangiopancreatography in patients with pancreatic trauma. *J Trauma Acute Care Surg.* 2010;68:538–44.
49. Wind P, Turet E, Cunningham C, Frileux P, Cugnenc PH, Parc R. Contribution of endoscopic retrograde pancreatography in management of complications following distal pancreatic trauma. *Am Surg.* 1999;65:777–83.
50. Nirula R, Velmahos GC, Demetriades D. Magnetic resonance cholangiopancreatography in pancreatic trauma: a new diagnostic modality? *J Trauma Acute Care Surg.* 1999;47:585–7.
51. Fulcher AS, Turner MA, Yelon JA, McClain LC, Broderick T, Ivatury RR, Sugerman HJ. Magnetic resonance cholangiopancreatography (MRCP) in the assessment of pancreatic duct trauma and its sequelae: preliminary findings. *J Trauma Acute Care Surg.* 2000;48:1001–7.
52. Fabian TC, Kudsk KA, Croce MA, Payne LW, Mangiante EC, Voeller GR, Britt LG. Superiority of closed suction drainage for pancreatic trauma. A randomized, prospective study. *Ann Surg.* 1990;211:724–8.
53. Farrell RJ, Krige JE, Bornman PC, Knottenbelt JD, Terblanche J. Operative strategies in pancreatic trauma. *Br J Surg.* 1996;83:934–7.
54. Zhou W, Lv R, Wang X, et al. Stapler vs suture closure of pancreatic remnant after distal pancreatectomy: a meta-analysis. *Am J Surg.* 2010;200:529–36.
55. Diener MK, Seiler CM, Rossian I, et al. Efficacy of stapler versus handsewn closure after distal pancreatectomy (DISPACT): a randomized, controlled multicentre trial. *Lancet.* 2011;377:1514–22.

56. Orci LA, Oldani G, Berney T, et al. Systematic review and meta-analysis of fibrin sealants for patients undergoing pancreatic resection. *HPB*. 2014. doi:10.1111/hpb.12064.
57. Hanna EM, Martinie JB, Swan RZ, et al. Fibrin sealants and topical agents in hepatobiliary and pancreatic surgery: a critical appraisal. *Langenbecks Arch Surg*. 2014. doi:10.1007/s00423-014-1215-5.
58. Pachter HL, Hofstetter SR, Liang HG, Hoballah J. Traumatic injuries to the pancreas: the role of distal pancreatectomy with splenic preservation. *J Trauma Acute Care Surg*. 1989;29:1352–5.
59. Mansour MA, Moore JB, Moore EE, et al. Conservative management of combined pancreatoduodenal injuries. *Am J Surg*. 1989;158:531–5.
60. Feliciano DV, Martin TD, Cruse PA, et al. Management of combined pancreatoduodenal injuries. *Ann Surg*. 1987;205:673–80.
61. van der Wilden GM, Yeh DD, Hwabejire JO, et al. Trauma Whipple: do or don't after severe pancreaticoduodenal injuries? An analysis of the National Trauma Data Bank. *World J Surg*. 2014;38:335–40.
62. Holland AJ, Davey RB, Sparnon AL, Chapman M, LeQuesne GW. Traumatic pancreatitis: long-term review of initial non-operative management in children. *J Paediatr Child Health*. 1999;35:78–81.
63. Keller MS, Stafford PW, Vane DW. Conservative management of pancreatic trauma in children. *J Trauma Acute Care Surg*. 1997;42:1097–100.
64. Shilyansky J, Sena LM, Kreller M, Chait P, Babyn PS, Filler RM, Pearl RH. Nonoperative management of pancreatic injuries in children. *J Pediatr Surg*. 1998;33:343–9.
65. Friess H, Bordihn K, Ebert M, Malfertheiner P, Kemmer T, Dennler HJ, Bucher MW. Inhibition of pancreatic secretion under long-term octreotide treatment in humans. *Digestion*. 1994;55(Suppl 1):10–5.
66. Bassi C, Falconi M, Lombardi D, Briani G, Vesentini S, Camboni MG, Pederzoli P. Prophylaxis of complications after pancreatic surgery: results of a multicenter trial in Italy. Italian Study Group. *Digestion*. 1994;55(Suppl 1):41–7.
67. Buchler M, Friess H, Klempa I, Hermanek P, Sulkowski U, Becker H, Schafmayer A, Baca I, Lorenz D, Meister R. Role of octreotide in the prevention of postoperative complications following pancreatic resection. *Am J Surg*. 1992;163:125–30.
68. Amirata E, Livingston DH, Elcavage J. Octreotide acetate decreases pancreatic complications after pancreatic trauma. *Am J Surg*. 1994;168:345–7.
69. Nwariaku FE, Terracina A, Mileski WJ, Minei JP, Carrico CJ. Is octreotide beneficial following pancreatic injury? *Am J Surg*. 1995;170:582–5.
70. Wales PW, Shuckett B, Kim PC. Long-term outcome after non-operative management of complete traumatic pancreatic transection in children. *J Pediatr Surg*. 2001;36:823–7.